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DIAGNOSTICS OF INTERNAL MEDICINE

A CLINICAL TREATISE UPON THE RECOGNISED PRINCIPLES OF MEDICAL DIAGNOSIS, PREPARED FOR THE USE OF STUDENTS AND PRACTITIONERS OF MEDICINE

BY

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WITH FIVE COLOURED PLATES AND TWO HUNDRED AND SEVENTY-TWO ILLUSTRATIONS AND CHARTS IN THE TEXT

THIRD REVISED EDITION

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PREFACE TO THIRD EDITION

THE third edition of this work—the preparation of which has naturally been an agreeable task—is now offered by the publishers. It again becomes a pleasure, as well as a duty, to acknowledge a number of suggestions from widely separated localities, some of which have been incorporated in this revision. There is, perhaps, no more interesting and reliable measure by which one can estimate the progress of our art, than the omissions, modifications, and additions which are required in successive editions of a medical book. There are few of prime importance, but many which afford much encouragement for the present and for the future.

The section on Kryoscopy has been omitted, as occupying space which can be used to better advantage. The section on X-Ray Diagnosis has been curtailed for a similar reason.

I am again indebted to Dr. Henry G. Webster for essential aid. Dr. Archibald Murray has made some useful changes in the section on Examination of the Blood. The section on Examination of the Stomach Contents and the Fæces has been much improved by Dr. Dudley D. Roberts. The section on the Nervous System has been carefully gone over by Dr. Smith Ely Jelliffe and Dr. Frederick Tilney. Dr. Thomas C. Craig, Surgeon, U.S.A. (retired), has prepared a new section on Life Insurance Examination. A new section on Diseases of the Tropics has been compiled by Dr. Hartwig Kandt. The recent tuberculin tests have been described and valued by Dr. Edward R. Baldwin.

As in previous editions, the matter of illustration has been in the hands of Mr. Howard J. Shannon.

G. R. B.

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CITY OF NEW YORK.

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PREFACE

THIS book has been written from the point of view of practical clinical work. The physician meets primarily symptoms and signs—the evidences of disease; subsequently it is decided that the symptoms found indicate the presence of a specific ailment. This volume, therefore, naturally divides itself into two parts: first, a study of *symptoms and their indications;* and, second, a study of *diseases and their characteristics.*

Part I—The Evidences of Disease—comprises: (1) A brief consideration of the clinical anatomy and physiology of certain organs and systems; practical points of everyday utility. (2) A description of the approved methods of examination. It has been well said by a capable reviewer that "the basis of the art of diagnosis is a thorough knowledge of clinical methods." (3) A careful consideration of the many signs and symptoms encountered in the practice of internal medicine. (4) A statement of the diagnostic significance of each sign and symptom—i. e., the disease or diseases, the presence of which is more or less strongly suggested by the finding of a given sign or symptom. While a prominent symptom seldom leads directly to the discovery of a disease, yet it is of importance to know the diagnostic value of individual symptoms.

Part II—Diagnosis, Direct and Differential—contains: (1) Succinct descriptions of recognised diseases and their symptoms, with (2) special reference to the diagnosis, direct and differential, of each disease. The qualifying terms applied to diagnosis are scientifically indefensible, but clinically useful.

The two parts are, indeed, complementary. For example, if in Part I it is stated that the finding of a persistently rapid pulse may be explained by the presence of exophthalmic goitre; or of a dry tongue and an inordinate thirst, by diabetes, one can turn to Part II and compare his case with the symptom-group of the disease in question. Conversely, when in Part II a high-tension pulse is mentioned as a symptom of angina pectoris, or Kernig's sign of meningitis, a reference to Part I will discover the method of estimating high tension or of eliciting Kernig's sign.

It is hoped that, owing to its choice of material and method of arrangement, the book contains between two covers practically all that is essential for the making of a diagnosis, and that no helpful clew in obscure cases has been overlooked. The value of modern laboratory methods has been fully appreciated; so also has the importance of symptoms, subjective and objective.

No one can write upon the subject of this book without lying under obligations for the major portion of his material to the Masters of Internal Medicine, but, as space forbids detailed references, this brief acknowledgment must stand as a very inadequate voucher for a heavy debt. Everything, indeed, has been subordinated to the main purpose of the book, which is to facilitate in a practical way the making of a thorough examination and a correct diagnosis. It is believed that the Synopsis of Examinations, which immediately precedes the body of the book, will be found useful.

Special care has been taken to secure clearness of arrangement by the liberal use of italics and bold-face type to catch the eye; and to promote ease of reference by varying the odd-page headings, as well as by the provision of an ample, but not too bulky, index.

Plates III and IV are composed of selections reproduced (with the kind permission of the authors and publishers) from the excellent illustrations in Cabot's Examination of the Blood and Simon's Clinical Diagnosis, mainly from the former; Plate V of similar selections from Thayer's fine drawings of the malarial parasite. A large proportion of the illustrations are either original or redrawn, without, it is believed, sacrificing utility for originality. Mr. Howard J. Shannon has put my rough sketches into workmanlike and, so far as compatible with the subject, artistic form. For his aid I am indebted to the liberality of the publishers, whose imprint is a guarantee of good work and good material.

The bulk of the volume (pages 1 to 908) is from my own pen. Of the remainder, Dr. Frank W. Shaw, my associate at the Methodist Episcopal Hospital, has prepared the sections on Parasites and the Intoxications; Dr. Henry G. Webster, my associate at the Brooklyn Hospital, those on Diseases of the Kidney and Constitutional Diseases; Dr. Henry P. De Forest, of Brooklyn, that on Diseases of the Blood and Ductless Glands; and Dr. Smith Ely Jelliffe and Dr. A. B. Bonar, of Manhattan, that upon Diseases of the Nervous System—assistance kindly given and gratefully received. Dr. J. P. Warbasse has made valuable criticisms.

G. R. B.

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"I do not know . . . I will investigate."—PASTEUR.

"First tell me what I am to look for."—FARADAY.

SYNOPSIS (OR SCHEDULE) OF EXAMINATIONS

CONSTITUTING AN ORDER OF PROCEDURE, AND A SYMPTOM-GUIDE; WITH REFERENCES TO PART I OF THIS BOOK

To insure completeness in the examination of patients and for purposes of record, it is desirable to have a definite and comprehensive order of procedure. The subjoined schedule, which may be modified according to personal requirements, is based partly on scientific necessities, partly on clinical convenience. Incidentally, the symptoms indicating disease of a particular viscus or system are grouped, in order to direct attention to the organ at fault. Furthermore, for convenience, references are given to the pages upon which special symptoms, signs, or methods of examination are described in detail.

The three main divisions of the schedule comprise:

I. The History or Anamnesis.
II. The General Examination } Present Condition or
III. The Special Examinations } Status Præsens.

I. THE HISTORY OR ANAMNESIS

Ascertain the name, age, sex, civil condition (single, married, widow, widower), nationality, occupation, and residence. Note the date of examination.

Family History.—Inquire concerning the diseases which have prevailed and the causes of deaths (if such have occurred) among father, mother, brothers, sisters, or children; also as to the diseases, if any, which prevail among the living. Consider whether or not the stated ailments are of an hereditary character (pages 13 to 15).

Previous Personal History.—Bear in mind the diseases which predominate:

(1) At the age period of the patient (pages 15 to 18).

(2) In the sex (pages 18, 19); and if the patient is a woman, inquire regarding the menstrual life, pregnancies, and miscarriages.

(3) In the race or nationality (page 19).

(4) What is the character of the occupation, and does it predispose toward special diseases (pages 19 to 21)?

(5) Consider the residence, bearing in mind the geographical distribution of disease (pages 21 to 22).

(6) Inquire concerning the habits: of men, with reference to the daily amount and kind of alcoholic beverages taken, whether before or after meals; tobacco, kind, amount, and manner of using; sexual indulgence, frequency; of both men and women, with reference to the amount and strength of tea and coffee taken (page 22).

(7) Inquire with reference to previous injuries and diseases (pages 22 to 25), ascertaining their date, duration, character, and whether or not recovery was considered to have been complete. Are the previous diseases of such a nature that a second attack is probable; or is it unlikely; or are sequelæ to be expected? Search especially for previous gonorrhœa, syphilis, nephritis, rheumatism, or malaria.

History of Present Illness.—Inquire regarding the possible cause of the illness; the date and manner of its onset, never failing to fix in mind the nature of the earliest symptoms, and, if possible, the organ or system to which they belong—e. g., stomach, circulatory apparatus—the subsequent symptoms and their order of appearance to the present time; the symptoms now present; and the previous treatment, if it can be ascertained (pages 25 to 27).

II. THE GENERAL EXAMINATION

1. Observe the dress and general behaviour (pages 27, 28).

2. Estimate (or measure) the height and weight, and note the amount and character of the adipose and muscular tissue (pages 28 to 30).

3. Study the shape and general configuration of the body (pages 30, 31).

4. Note the complexion, and colour of hair and eyes (pages 31, 181, 199).

5. So far as possible, determine the diathesis (usually done at the end of the examination), and note the presence of any cachexia (pages 31 to 33).

6. Observe the posture and manner of moving (pages 33 to 35).

7. If practicable, test the station or power of standing, and observe the gait or manner of walking (pages 35 to 38).

8. **Pain.**—If pain is a subject of complaint, make due allowance for susceptibility (page 39) and manner of statement (pages 39, 40). Can any diagnostic inference be drawn from the character (pages 40, 41) or the seat (pages 42 to 60) of the pain?

9. **Tenderness.**—Is there tenderness (pain on pressure), and is its location significant (pages 60 to 62)?

10. **Paræsthesias.** — Are there abnormalities of sensation not amounting to pain (paræsthesias)? If so, consider the site, variety, and possible significance (pages 62 to 66).

11. **Vertigo.**—Inquire for the existence of vertigo, having in mind both its common and less frequent causes (pages 66 to 68).

12. **Temperament.**—What is the temperament (mainly psychical) of the patient (pages 68 to 70)?

13. **Psychical State.**—What is the present psychical state of the patient as shown by the facial expression (pages 70, 71); the emotional state (page 71); and the condition of intellection (pages 71 to 73); the abnormalities of the latter, embracing mental dulness, loss of memory, delusions, or delirium? Inquire as to sleep (page 73).

14. **Consciousness.**—Is the patient fully conscious? If not, what is the degree of disturbance (page 74); what may it signify in general (pages 74 to 76); and to what is it due in this particular case (pages 76 to 79)?

15. **Convulsions.**—If general convulsions have occurred or are present, to what may they be attributed (pages 79 to 82)?

16. **Cutaneous Surface.**—Observe and examine the cutaneous surface with reference to colour (pages 83 to 89), heat (pages 89, 90), moisture (pages 90, 91), rash or eruption (pages 91 to 94), scars (pages 94, 95), dropsy (pages 95 to 98), condition of the veins (page 99), and emphysema (pages 100, 101).

17. **Pulse.**—Take the pulse (pages 387 to 392). Observe its frequency, rhythm, tension, and other qualities. Note the condition of the arteries. If variations from the normal are found, consider their significance (pages 392 to 407).

18. **Respiration.**—Take the respiration. Observe its frequency, type, rhythm, and other characteristics (pages 416 to 425).

19. **Temperature.**—Take the temperature of the body (pages 104 to 109). If fever is present, consider its height, type, manner of invasion, course, and termination (pages 109 to 114). What diagnostic inferences may be drawn from these observations (pages 114 to 120)? Has the fever been preceded or accompanied by chills (pages 120, 121)? If the temperature is subnormal what may it indicate (page 120)?

20. Inquire concerning the **appetite** and **thirst** (pages 122, 123); **vomiting** (pages 123 to 131) and the gross characters of the **vomitus** (pages 132 to 135); **defecation** (pages 135 to 142) and the gross characters of the **stools** (pages 142 to 155); the character and frequency of **urination** (pages 155 to 160); and certain **genital symptoms** in men and women (pages 160 to 165).

III. SPECIAL EXAMINATIONS

By means of a more or less discursive examination, as just outlined, the observer obtains a conception of the general condition of the patient; and also, in the majority of cases, an indication for a special examination of a particular part, organ, or system. The special examinations embrace the signs and symptoms which occur in connection with various parts of the body—e. g., head and face, tongue; or which belong to an organ—e. g., spleen; or a system—e. g., respiratory.

1. **Head and Face.**—Observe the size and contour of the head and face; in infants, the condition of the fontanels and sutures; and the consistence and surface of the cranial bones (pages 170 to 176). Study the expression of the face and consider whether it is indicative of certain diseases (pages 176 to 179). Note the colour of the face and the state of the skin of the face (page 180). What is the colour of the hair, and is it abundant, or scanty (page 181)? Is there general or circumscribed swelling of the face (page 182)? Are there abnormal movements of the head, or does it lack normal mobility (pages 182, 183)? Are the facial muscles in a state of clonic or tonic spasm (pages 183, 184), or are they paralyzed (pages 185 to 190)?

2. **Ear.**—Has the patient complained of pain in the ear (page 191)? What is the colour and shape (pages 191, 192) of the ear? Is there a discharge from the external meatus (page 192)? Does the patient complain of tinnitus (pages 192, 193)? Does the patient hear well; is he deaf, and, if so, is the deafness due to nerve lesions or aural lesions (pages 193 to 195)? Is the hearing hyperacute (page 196)?

3. **Eye.**—Are the eyelids swollen or ulcerated; in a state of spasm; too widely opened; or abnormally drooping (pages 196 to 199)? What is the colour of the sclerotic, the state of dryness or moisture of the eye, and the condition of the cornea (pages 199 to 201)? Are the pupils large or small, equal or unequal; do they respond to light and to accommodation (pages 201 to 206)? Are the eyeballs painful; do they protrude, or are they more sunken than normal; what is their position (pages 206 to 214)? Are the eyeballs normally mobile, or are there symptoms of ocular paralysis; and if ocular paralysis is found, what is its cause (pages 214 to 219)? Does the patient complain of any abnormality of sight (page 219)? If alterations in the shape or size of the visual fields have been found, what may be their significance (pages 219 to 225)? If an ophthal-

moscopic examination of the eye grounds has been made, do the findings indicate extra-ocular disease (pages 225 to 228)?

4. **Nose.**—The following symptoms demand an examination of the nose: *Pain in or around the nose* (page 230), *frontal headache, or trigeminal neuralgia. Mouth-breathing and its typical facial expression due to nasal stenosis* (page 178). *Snoring and restless sleep. Nasal voice. Nasal discharges* (page 232), *epistaxis* (pages 232, 233), *or bad odour of the expired air* (pages 236, 237). *Deafness. Cough or bronchial asthma.*

To Examine the Nose.—Having noted the shape and colour of the nose, together with such other points as may be observed by ordinary inspection, examine the nasal chambers (using the probe) by anterior and posterior rhinoscopy (pages 228 to 230). Test the sense of smell (pages 233 to 235).

5. **Mouth.**—Examine the lips, buccal cavity, gums, and teeth (pages 235 to 242). Note the condition of the tongue with reference to colour, size, spasm, tumour, paralysis, scars, fissure, ulcers, etc. (pages 242 to 249). Does the tongue present an appearance which is of general diagnostic value (pages 247 to 249)? Test the sense of taste (pages 249, 250).

6. Examine the **palate, tonsils,** and **pharynx** (pages 250 to 254). What is the shape of the palate; is it paralyzed, anæsthetic, or otherwise abnormal? Are the tonsils acutely swollen, chronically enlarged, ulcerated, or covered with exudate? What is the colour of the pharynx; is there exudate or ulceration; is there bulging posteriorly; is it paralyzed or anæsthetic?

7. Does the patient complain of **dysphagia,** and, if so, to what may it be due (pages 254, 255)?

8. **Larynx.**—The following symptoms demand an examination of the larynx: *Pain, burning, or soreness over and around the larynx. Alterations in the character of the voice sounds, viz., aphonia or hoarseness* (*dysphonia*). *Inspiratory dyspnœa, especially if accompanied by stridulous* (*wheezing or squeaking*) *respiration. Cough, particularly of the laryngeal type* (*tight or croupy*). *Dysphagia, difficulty or pain in swallowing.*

To Examine the Larynx.—(See pages 255 to 260.) Do not omit an inspection of the lingual tonsil.

9. **Cough.**—Has the patient a cough? If so, observe its character and consider its causes (pages 273 to 276). Examine the sputum (if any) with reference to its character (pages 276 to 278 and 650 to 657). Has he had hæmoptysis (pages 278 to 280)?

10. **Speech.**—Note alterations in the voice or the manner of speaking (pages 260 to 263). Is there aphasia (pages 263 to 272)?

11. **Neck.**—Observe the shape of the neck; is it rigid? Are the sterno-mastoids or clavicles prominent; is the thyroid gland enlarged or atrophied (page 281)? Does the trachea descend with inspiration, or can tracheal tugging be felt (page 282)? What is the condition of the cervical glands (pages 282 to 284); of the arteries of the neck (page 284); of the veins of the neck (pages 284 to 288).

12. **Extremities.**—Examine the nails (pages 288, 289); the hand and fingers (pages 289 to 295); the arm (page 295); the foot and leg (pages 296 to 300); and the joints (pages 101 to 104).

13. **Back.**—Examine the back for alterations of shape, prominence of the scapulæ, stiffness, and swellings or bulgings (pages 301 to 304).

14. **Chest.**—Examine—perhaps measuring and outlining—the chest with reference to bilateral or unilateral deformities, flexibility of ribs, and the presence of enlarged veins (pages 314 to 321).

15. **Heart and Blood-vessels.**—The following symptoms demand an examination of the heart and blood-vessels: *Dyspnœa (perhaps orthopnœa), especially if made worse by physical exertion or accompanied by cyanosis. Œdema, especially of the feet and ankles. Palpitation, præcordial pain, anxiety, or distress, particularly if increased by exertion. Sudden vertigo. Restless sleep, dreaming, starting during sleep. Cough, especially if chronic; or an unusually persistent attack of bronchitis. Chronic digestive disturbances. Hemorrhoids. Great obesity.*

If such symptoms are present inquire further (with reference to causation) concerning: *Prolonged and severe muscular exertion. Many years of constant mental excitement or anxiety. Excessive eating and drinking, especially of rich food and alcoholic beverages; these, and the foregoing, partly with reference to arteriosclerosis. Excessive use of tobacco, tea, and coffee (in relation to cardiac neuroses). Previous attacks of chorea, gout, rheumatic fever, or other, usually acute, infectious diseases, especially scarlatina, diphtheria, typhoid fever, tonsilitis, syphilis. The family history: does it reveal rheumatism, gout, angina pectoris, apoplexy, or organic cardiac disease?*

To Examine the Heart.—Inspect and palpate the thorax, noting, if present, distended veins, pulsating jugulars, epigastric pulsation, and pulsating liver. Note, as of prime importance, the position, character, and extent of the apex-beat (pages 341 to 350). Percuss the heart (pages 350 to 359). Auscultate the heart with reference to the intensity and character of the sounds (pages 359 to 366), and the presence of adventitious sounds, either endocardial (pages 367 to

380), or exocardial (pages 380 to 383). Examine the pulse (pages 387 to 407). Use the sphygmograph (pages 407 to 411).

To Examine the Blood-vessels.—Inspect, palpate, and auscultate the accessible arteries and veins (pages 383 to 387). Note any abnormal capillary pulsation (page 288).

16. **Lungs and Pleuræ.**—The following symptoms demand an examination of the lungs and pleuræ: *Cough, with or without expectoration. Hæmoptysis or spitting of blood. Pain in the side of the chest. Dyspnœa. Night sweats. Loss of flesh and strength.*

Additional evidence should be sought for, viz., *a family history of consumption, asthma, bronchitis, or scrofulous (tuberculous) diseases; and a personal history of enlarged cervical glands, or tuberculous disease of bone, or association with a consumptive, or an occupation predisposing toward pulmonary disease.*

To Examine the Lungs.—Inspect and palpate the chest with reference to its shape (pages 314 to 321). Measure it. Count the respiration (pages 416, 417); determine its type, degree of expansion and retraction, and its rhythm and other characters (pages 416 to 425). Is fremitus obtained (pages 425 to 427)? Is dyspnœa present? If so, what is its character? Percuss the lungs—front, sides, and back (pages 427 to 441). Auscultate the lungs—front, sides, and back, determining the character of the breath sounds (pages 441 to 450) and the presence and variety of adventitious sounds (pages 450 to 454).

17. **Abdomen.**—If complaint is made of *abdominal pain* or *discomfort,* inspect the abdomen (pages 459 to 462). Palpate and percuss the abdomen (pages 462 to 478). Auscultate the abdomen (pages 478, 479).

18. **Stomach.**—The following symptoms require an examination of the stomach: *Fulness, sinking feelings, pain or discomfort in epigastrium, lower sternum, between the scapulæ. Increased or lessened appetite or increased thirst. Nausea or vomiting (of stomach contents, or blood). Pyrosis, eructations, or flatulence. Mental depression. Rapid emaciation.*

To Examine the Stomach.—Incidentally inspect the lips, mouth, gums, teeth, and tongue. If the food is arrested in the throat, or before it enters the stomach, and is regurgitated, palpate, auscultate, and instrumentally examine the esophagus (pages 479 to 481). Inspect and palpate the stomach (pages 483, 484). Percuss the stomach by ordinary and auscultatory percussion (pages 484 to 488). Inflate the stomach (page 488). Obtain the contents of the stomach (pages 490, 491) after a test meal, and examine by chemical and microscopical methods (pages 658 to 673).

19. **Intestines.**—*Constipation, diarrhœa,* and *abdominal pain* are the symptoms which require an examination of the intestines.

To Examine the Intestines.—Inspect the stools (pages 142 to 155). Inspect, palpate, percuss, and auscultate the abdomen in general (pages 459 to 478), and the intestines (including a digital examination of the rectum) in particular (pages 493 to 497).

20. **Liver and Gall Bladder.**—The following symptoms demand an examination of the liver and gall bladder: *Pain, of the hepatic type, over the right hypochondrium. Jaundice, dark urine, clay-coloured stools. Irregular chills and fever. Cutaneous pruritus. Hæmatemesis. Digestive disturbances.*

If such symptoms are present, inquire further (with reference to causation) concerning: *Previous attacks of jaundice with or without hepatic colic. Previous catarrh of the stomach, or acute indigestion (catarrh of bile ducts). Strong emotions (anger or fright). Chronic alcoholism (hepatic cirrhosis). Syphilis, tuberculosis, or long-continued suppuration (amyloid disease). Possibility of phosphorus poisoning.*

To Examine the Liver and Gall Bladder.—Rarely inspection and auscultation are of use, ordinarily palpation and percussion (pages 499 to 506) are to be relied upon.

21. **Spleen.**—The size, shape, and position of the spleen should be determined, mainly by palpation and percussion (pages 507 to 511), in the following conditions and diseases: *Emphysema, left pleural effusion, and left pneumothorax. Ascites, tympanites, and large abdominal tumours. In all acute infectious diseases (e. g., typhoid fever, malarial fever). Leucœmia. Cirrhosis or amyloid disease of the liver.*

22. **Kidneys.**—The following symptoms demand a physical examination of the kidneys, and a chemical and microscopical examination of the urine: *Pain in the posterior lumbar region, especially if of the renal type. Œdema or puffiness of the face, especially about the eyelids in the morning. General œdema (anasarca). Painful or frequent urination. Smoky or turbid urine; notable increase or diminution in its amount. Headache, drowsiness, nausea, and vomiting. Dyspnœa or asthma, without other discoverable cause. Dimness of vision. Convulsions or paralyses. Irregular chills and fever (pyelitis).*

If such symptoms are present, inquire further (with reference to causation) concerning: *A family history of nephritis, apoplexy, or gout. A personal history of alcoholism, gout, lead-poisoning, renal colic, chilling of the surface of the body, acute infectious diseases (such as scarlet fever, malaria, tonsilitis, diphtheria), and long-*

continued suppuration, tuberculosis, tertiary syphilis, and malaria (amyloid disease).

To Examine the Kidneys.—Inspect and palpate anteriorly; inspect, palpate, and percuss posteriorly (pages 511 to 516). Examine also the heart and blood-vessels for cardiac hypertrophy and general arteriosclerosis. Examine the urine physically, chemically, and microscopically (pages 681 to 712). If necessary, examine the bladder and ureters.

23. **Nervous System.**—The following symptoms require an examination of the nervous system: *Frequent or continuous headache. Frequent vomiting. General convulsions, or localized spasm. Paralyses (ocular or skeletal). Vertigo. Speech disturbances. Difficulty in standing or walking if not due to weakness, injury, or disease of joints. Mental disturbances. Dysphagia (sometimes).*

If such symptoms are present, inquire further (with reference to etiology) concerning: *A family history of psychoses (insanity), hysteria, chorea, epilepsy, neurasthenia, paralysis, convulsions, or hereditary syphilis. A personal history of alcoholism, syphilis, injury; discharge from the ear; any of the acute infectious diseases; poisoning from lead, mercury, arsenic, tobacco, or naphtha; and exposure to cold.*

To Examine the Nervous System.—Note the presence of the stigmata of degeneration (pages 541 to 543). Examine the muscles with reference to their nutrition, tone, and motor power (pages 543 to 548). Are there motor disturbances? If so, is there increased motility (spasm, pages 548 to 553), or decreased motility (paralysis, pages 554 to 562)? Are there disturbances of sensation (pages 562 to 570)? What is the condition of the superficial reflexes (pages 571 to 573); of the deep reflexes (pages 573 to 578)? What is the electrical reaction of the muscles and nerves (pages 578 to 585)? Are there vasomotor and trophic disturbances (pages 585, 586)? What is the condition of the cranial nerve functions (pages 586 to 589)? What are the findings from an examination of the eye grounds (pages 225 to 228)?

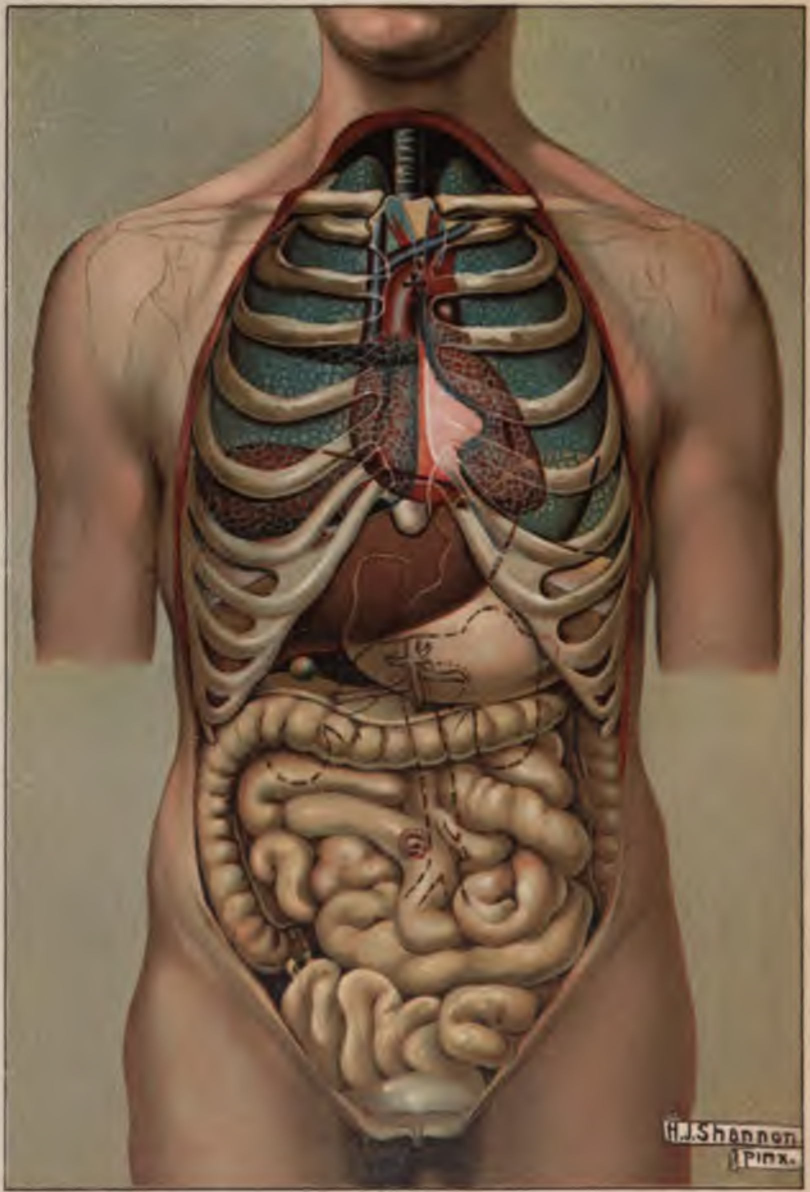
24. **Blood.**—The following symptoms require an examination of the blood: *Dyspnœa and palpitation upon exertion. Pallor of the skin and mucous membranes. Headache and vertigo. Debility. Disturbances of digestion and gastric pain. Œdema of the feet.*

If such symptoms are present, inquire further (with reference to causation) concerning: *Hereditary or personal hæmophilia. Loss of blood (injury, menorrhagia, bleeding piles, hæmoptysis, hæmatemesis, etc.). Malaria, rheumatic fever, lead-poisoning. Chronic gastric or intestinal catarrh; or a long-continued diarrhœa. Worry and mental excitement. Wasting diseases.*

To Examine the Blood.—Count the red and white cells (pages 595 to 604). Estimate the hæmoglobin (pages 605 to 613). Stain a dried specimen of the blood and make a differential count of the leucocytes (pages 613 to 631). Examine a fresh specimen of the blood (especially for the malarial organism, pages 631 to 638).

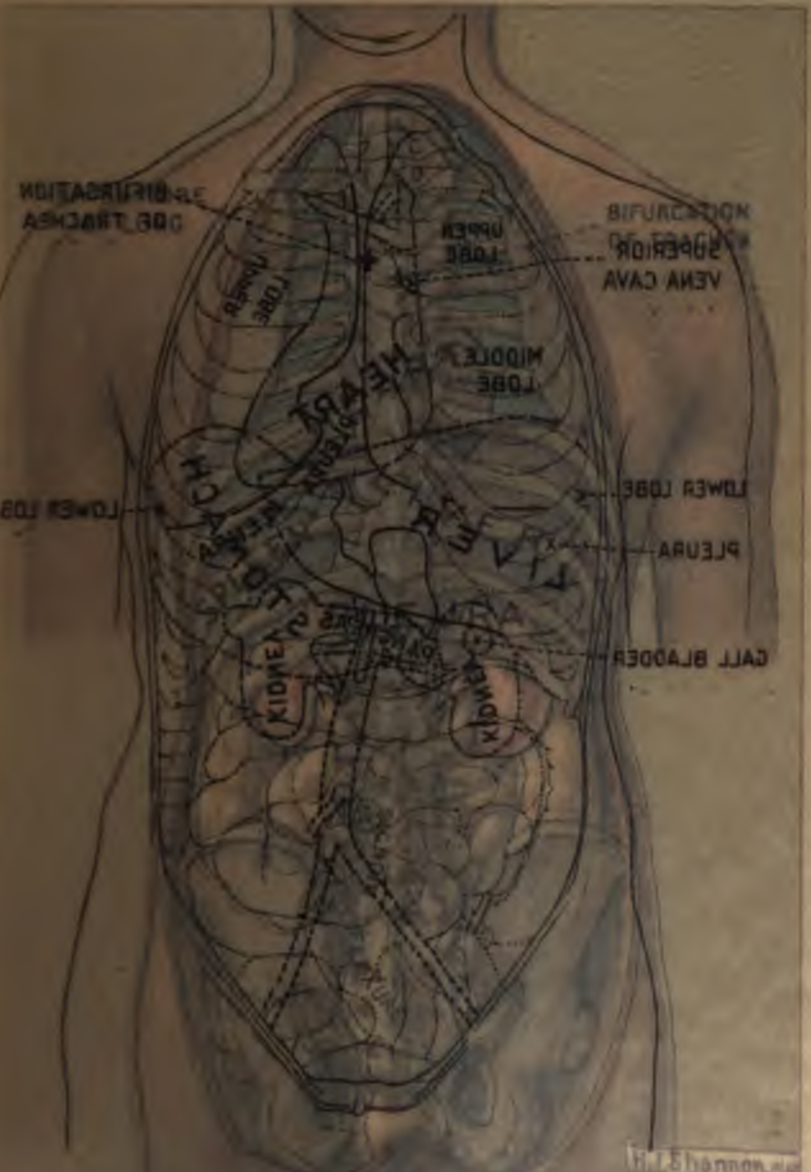
25. **Diagnostic Puncture.**—If desirable, obtain fluid by puncture (pages 712, 713) from cavities or cysts; examine the fluid, and from its character endeavour to determine its source (pages 713 to 718).

PLATE I.



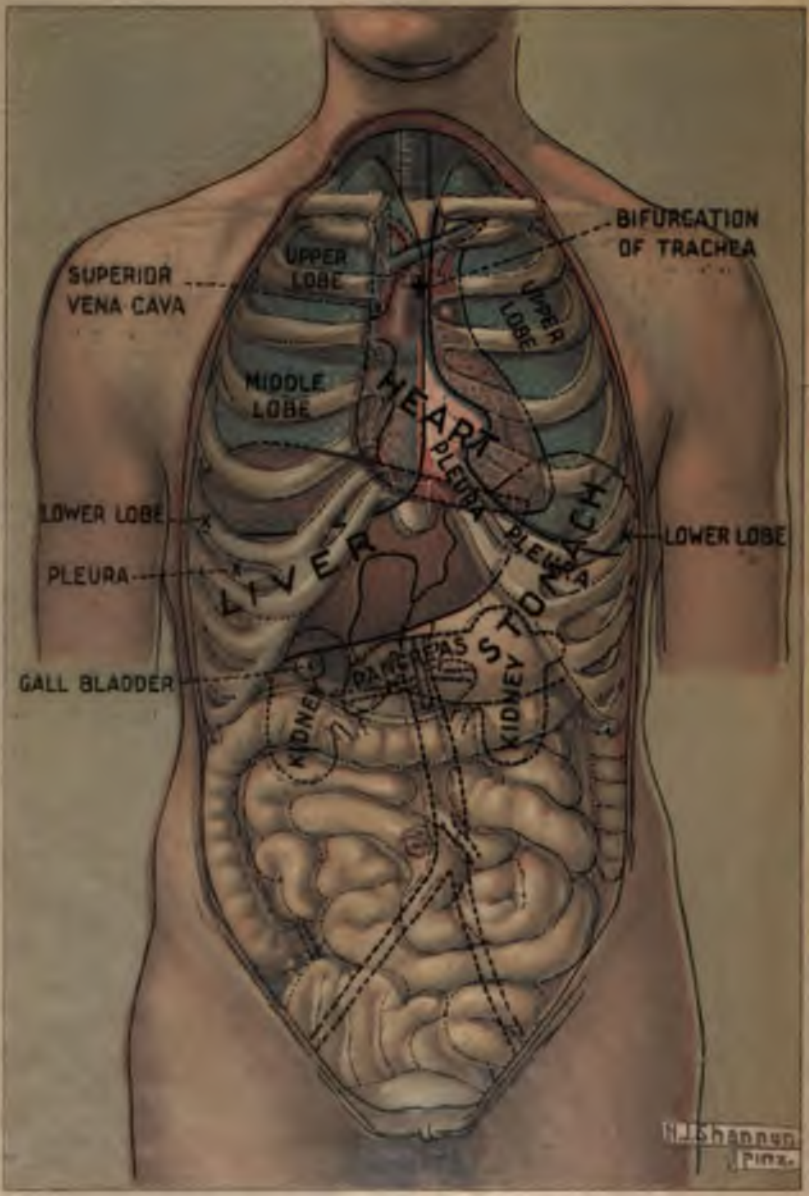
THE SHAPE AND RELATIONS OF THE THORACIC AND ABDOMINAL VISCERA,
ANTERIOR ASPECT (SEMI-DIAGRAMMATIC)

PLATE II.



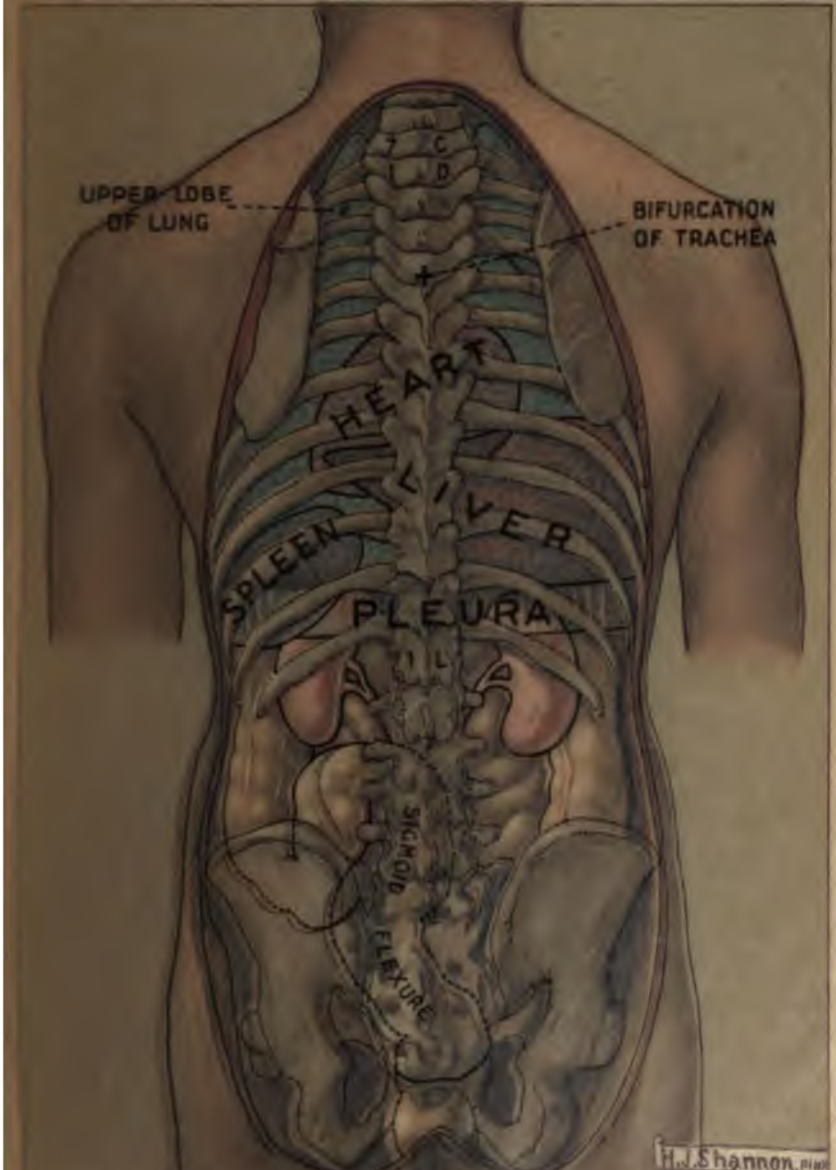
THE SHAPE AND RELATIONS OF THE THORACIC AND ABDOMINAL VISCERA.
POSTERIOR ASPECT (SEMI-DIAGRAMMATIC)

PLATE I.



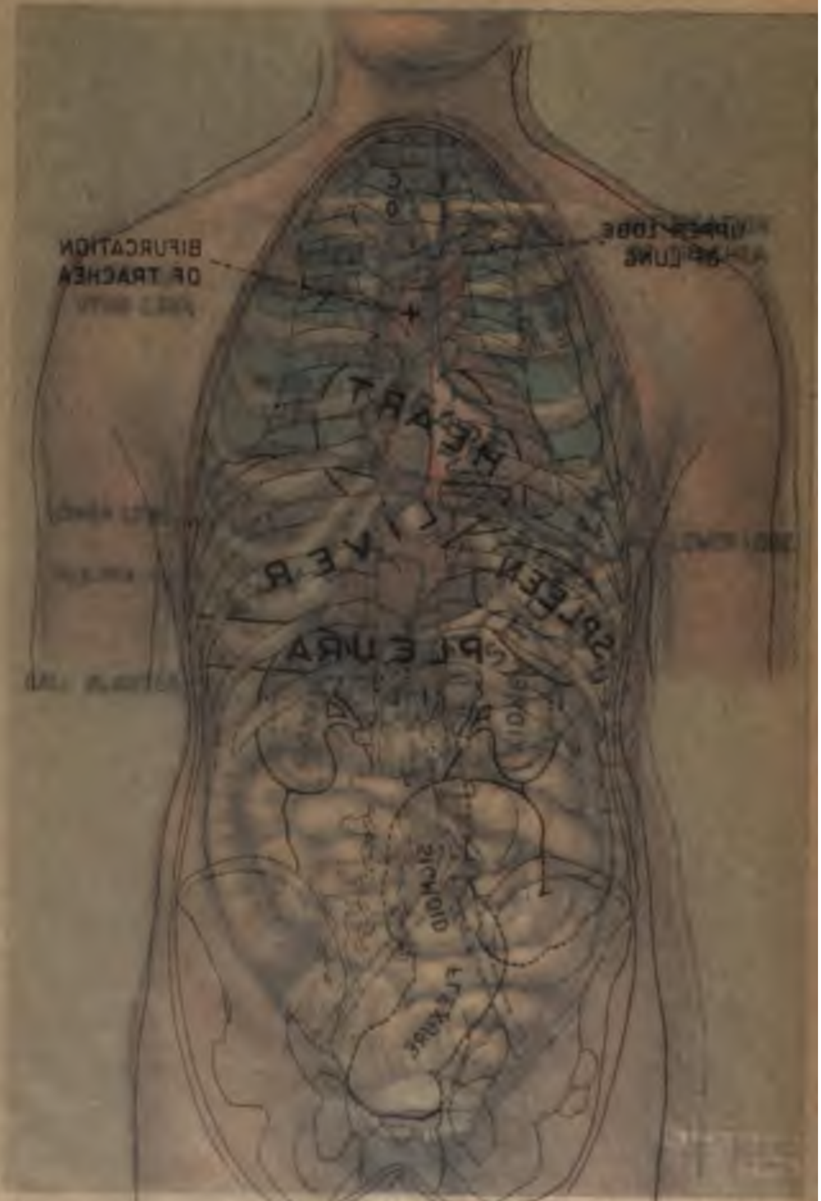
The Shape and Relations of the Thoracic and
Abdominal Viscera,
Anterior Aspect (Semi-diagrammatic)

PLATE II.



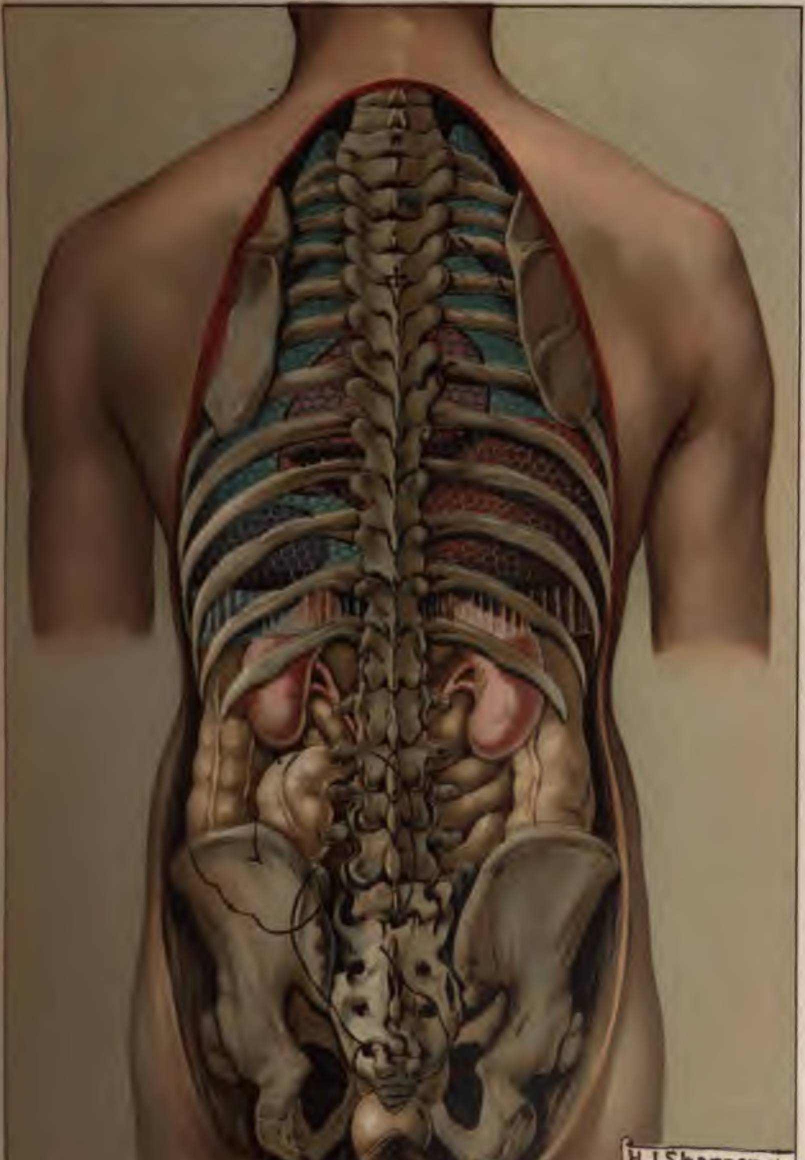
THE SHAPE AND RELATIONS OF THE THORACIC AND ABDOMINAL VISCERA.
POSTERIOR ASPECT (SEMI-DIAGRAMMATIC)

PLATE I.



THE SHAPE AND RELATIONS OF THE THORACIC AND ABDOMINAL VISCERA.
ANTERIOR ASPECT (SEMI-DIAGRAMMATIC)

PLATE II.



THE SHAPE AND RELATIONS OF THE THORACIC AND ABDOMINAL VISCERA, POSTERIOR ASPECT (SEMI-DIAGRAMMATIC)

THE DIAGNOSTICS OF INTERNAL MEDICINE

PRELIMINARY CONSIDERATIONS

Diagnosis.—This, in its narrowest sense, consists in bestowing a name upon a certain assemblage of pathological phenomena. It should include also a knowledge of the causal factors of the disease; a determination of its character with reference to type and severity; an estimate of the amount and kind of damage, both general and local, which has been sustained by the organism; a forecast of the probable course and duration of the morbid process; and a cognizance of the personal characteristics of the patient, whether psychic or physical, inherited or acquired. Its final object is to be able to treat disease intelligently, and the application of scientific methods to the completest discrimination and recognition of disease constitutes the art of diagnosis.

A diagnosis is made by means of symptoms, which constitute the evidence upon which is based a judgment as to the nature of the case. Symptoms, the phenomena caused by morbid processes, are divided into *subjective*, those which can be appreciated only by the patient, and *objective*, comprising those which are detected by the personal examination made by the physician. The term *physical signs* is by common consent applied to the objective symptoms revealed by special methods of examination, used mainly in determining the condition of the organs contained in the chest and abdomen.

In addition to a knowledge of the symptoms, subjective or objective, which may exist at the time of examination, it is necessary to ascertain the presence or absence of hereditary taints or tendencies, to know something about the habits and occupation of the patient, to learn of past illnesses or injuries, and to obtain a clear idea of the manner of onset and subsequent evolution of the present disease. Finally, it is well to study the temperament and personal characteristics, mainly psychical, of the individual patient so far as practicable.

The family and personal history, the history of the present illness, and the results of the examination constitute the evidence upon which the final judgment as to the nature of the case is to be based. A necessarily heterogeneous collection of facts must be classified with reference to their relative value and significance and compared with the previous knowledge and experience of the diagnostician, after which a judgment may be rendered as nearly as possible in accordance with the facts. This constitutes the second and final step in the making of a diagnosis. The process is thus seen to consist of two elements—*observation*, in its broadest sense, and *reasoning*, applied to the results of the observation.

Certain terms of some practical value and convenience are used to qualify a diagnosis, as follows (the definitions are largely from Foster):

LIST OF DESCRIPTIVE TERMS EMPLOYED WITH REFERENCE TO DIAGNOSES, SYMPTOMS, AND SIGNS

Diagnoses:

1. *Anatomical.*—Based on a knowledge not only of symptoms or phenomena, but also of definite anatomical alterations on which the phenomena depend; or a post-mortem diagnosis.
2. *Clinical.*—Based upon the symptoms manifested during life.
3. *By Exclusion.*—Reached by a deductive process, all the affections which present salient points of similarity with the one to be diagnosticated being reviewed in turn, and each successively discarded as one or more of its essential features are missed in a given case, until but one possibility remains, which is accepted as the true one.
4. *Differential.*—The process of distinguishing between different diseases which resemble one another more or less closely.
5. *Direct.*—The symptoms are of such a nature that they point to the presence of one special disease, and are not capable of misinterpretation.
6. *Pathological.*—Of the nature of a lesion, without regard to its situation.
7. *Physical.*—By means of physical (objective) signs, irrespective of subjective symptoms, as by palpation, auscultation, etc.
8. *Presumptive.*—Not regarded as certain.
9. *Retrospective.*—Of some antecedent disease or injury, the nature of which can be deduced only from the history given and from the persistent effects.
10. *Symptomatic.*—Consisting simply in the determination of the most striking symptoms.
11. *Topographical.*—Of the seat of a lesion.

Symptoms:

1. *Constitutional.*—Those that may result from unbalancing of the organism as a whole, and are common to affections of many kinds.
2. *Direct.*—Those that depend directly upon the disease.
3. *General.*—Constitutional. (See above.)
4. *Indirect.*—Which are the indirect consequences of the disease.
5. *Local.*—Which result from localized disease, and are usually confined to the site of the diseased organ or tissue.
6. *Negatively Pathognomonic.*—Which seldom or never occur in a certain disease, and consequently, if present, show that the case is not one of that disease.
7. *Pathognomonic.*—Which undeniably indicate the existence of a certain disease.
8. *Reflex.*—Which are caused by local disease, but manifest themselves by means of the nervous system in an otherwise unrelated organ or part of the body.
9. *Sympathetic.*—Which appear with the essential ones, but for the presence of which no cause can be assigned except that of sympathy.

Signs:

1. *Physical.*—Already defined.
2. *Rational.*—Signs and symptoms, subjective or objective, corresponding to the alterations in structure and mechanical conditions discovered by physical examination.
3. *Stethoscopic.*—Those discovered by auscultation.

Difficulties in Diagnosis.—For various reasons it may be *difficult or impossible to make a diagnosis.* The most important of these reasons are as follows:

(1) The subjective symptoms may be puzzling or incongruous.

(2) The objective symptoms and signs may be ill-defined, obscure, or, if present, as discovered later, may be impossible of detection by the most searching examination—e. g., a beginning, small perinephritic abscess in an unusually obese patient, which can not be palpated until it reaches a certain size.

(3) Certain symptoms essential to a diagnosis may not appear until the disease has advanced to a certain stage—e. g., the splenic enlargement and rose rash of typhoid fever.

(4) Several diseases, each of which in other cases may constitute the sole morbid process, may co-exist, one as the primary or main disease, the others attending as complications or sequelæ. One or more of the secondary lesions may be recognised, and the underlying or primary disease or condition be overlooked—e. g., pleurisy with

effusion occurring as a result of previously unrecognised pulmonary tuberculosis, and masking the primary lesion in the lungs.

(5) The rarity of a disease may lead to its non-recognition because of unfamiliarity with its history and symptomatology, and perhaps the consequent failure to elicit all the diagnostic data.

(6) The lack of a full and accurate history is at times a serious hindrance in making a diagnosis. The patient may be deaf and dumb or speak an unfamiliar tongue, or he may be unconscious, delirious, mentally unsound, or so ignorant and stupid that no reliable information can be obtained from him, and intelligent friends or relatives may not be available. On the other hand, in consequence of a variety of motives, essential facts may be concealed by the patient or the friends.

(7) Drug symptoms, unless known and due allowance made for them, may so disguise, add to, or simulate certain diseases that the diagnosis may be shrouded in uncertainty.

(8) The diagnostician must be a good observer, and at the same time be able to reason correctly. As Huxley well says: "Scientific reasoning differs from ordinary reasoning in just the same way as scientific observation and experiment differ from ordinary observation and experiment—that is to say, it strives to be accurate; and it is just as hard to reason accurately as it is to observe accurately. In scientific reasoning general rules are collected from the observation of many particular cases; and, when these general rules are established, conclusions are deduced from them, just as in everyday life. If a boy says that 'marbles are hard,' he has drawn a conclusion as to marbles in general from the marbles he happens to have seen and felt, and has reasoned in that mode which is technically termed induction. If he declines to try to break a marble with his teeth, it is because he consciously, or unconsciously, performs the converse operation of deduction from the general rule 'marbles are too hard to break with one's teeth.' . . . The man of science, in fact, simply uses with scrupulous exactness the methods which we all, habitually and at every moment, use carelessly."

While medicine is to a certain extent a science, and requires scientific modes of reasoning, medical art is, in a large proportion of cases, obliged to reason from probabilities as premises, and its final results can not be expressed in the exact formulæ of the mathematician. In obedience to some law which we do not yet know, 2 and 2 do not always make 4 in the reactions of the human body, otherwise patients would not be encountered who present serious symptoms after a medicinal dose of morphine, or blaze out with urticaria after eating strawberries.

To study and to compare; to approach a case with a mind open to impressions, and without preconceived or fixed ideas as to its nature, based upon previous hearsay; to review and balance the evidence from time to time in the course of the disease; to question one's self "Is there any other disease or condition which may better explain these symptoms than that which I have already assigned?"—these and other habits of thought make the difference between the man who sees without learning and he who learns by seeing.

Obtaining Evidence.—The diagnostician acquires the facts upon which he is to form an opinion: (1) By *Interrogation*—inquiry of the patient or his friends; (2) by *Observation*—an examination, mainly objective, of the patient. Information obtained by interrogation is called the *History* or *Anamnesis* (remembrance); that derived from observation, the *Present Condition* or *Status Præsens.*

From a purely scientific and diagnostic point of view, the first questions addressed to the patient will be with reference to his ancestry; next, in regard to his personal history antecedent to the present disease; then as to the existing disease, followed by a careful and systematic examination, first of the general condition, then of special organs, one by one, together with such chemical, microscopical, bacteriological, and other investigations as appear to be demanded. But for obvious reasons this order of pursuing the investigation is for the most part impracticable, and consumes an unnecessary amount of time.

In the vast majority of cases the facts are acquired by the physician in the reverse order. The first question asked is, "In what way do you feel ill?" "Of what are you complaining?" Or, the patient will volunteer a statement as to his subjective sensations. The question or the statement will direct attention at once to the probable or possible seat or nature of the disease. Further inquiries are put as to the duration and character of the morbid sensations. During these interrogations the physician attentively scrutinizes the general aspect of the patient in search of obvious objective symptoms. The pulse, respiration, and temperature are taken. The organ or part which appears to be most at fault is first examined, after which due attention is paid to other portions of the body. Finally, the family and social history may be ascertained.

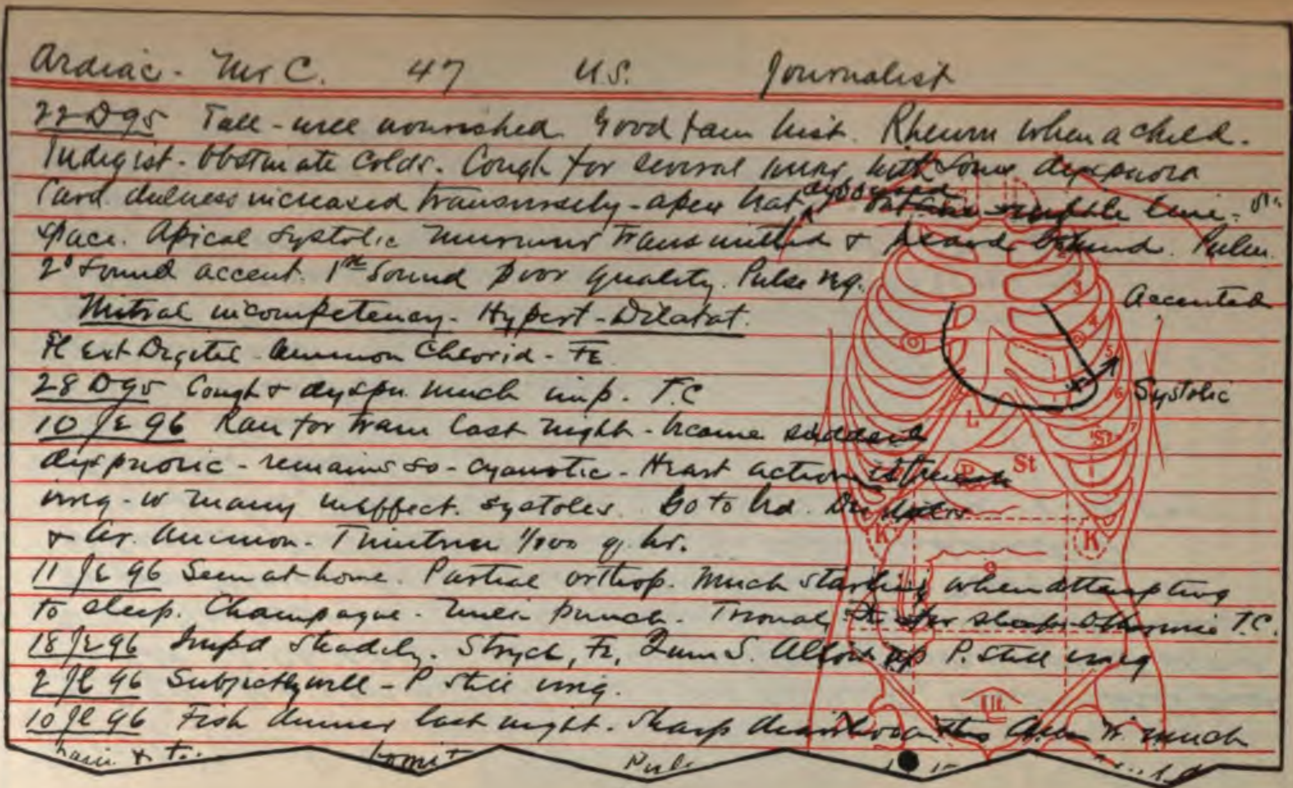
This is the logical order of investigation, *as conditioned by actual circumstances.* It is a matter of indifference as to the sequence in which the symptoms are learned, provided that the examination is sufficiently intelligent and systematic to be sure of eliciting all the facts, and that the facts when obtained are so arranged in the mind of the physician that they form a clear and coherent picture, and are capable of being recorded in an orderly manner. It is to be remembered in this connection that in many cases it is just as necessary to note negative facts—i. e., the absence of certain symptoms or signs—as it is to ascertain the presence of others.

Keeping Case Histories.—This habit promotes accuracy of observation, completeness in examination, and affords trustworthy material. The physician who keeps adequate records acquires facility in describing symptoms, signs, and morbid conditions. The drawbacks are the time consumed and the amount of work involved, but by late methods the time and labour required are reduced to a minimum. To accomplish this requires certain materials and accessories. The essential elements (DICKINSON) of the outfit are of two kinds: (1) Cards made and handled according to the card catalogue system, and (2) rubber stamps made to suit individual requirements.

(1) *Card Outfit.*—Records are made (Figs. 1 and 2) upon cards (6 by 6⅝ inches), one or more being used for each case, standing on edge in a box or drawer and ranged under an alphabetical index, each letter printed on a buff Bristol card (guide or index cards) which stands higher than the history cards.

The size of the history card is such that, by folding once (the line of the fold must be vertical), it will fit into the pocket-book or visiting list. The cards are kept in a box with a sliding top, or with a lid which is the upper third of the box (Fig. 3). When Mr. A. or Mrs. S. enters the consulting room, the cards under A or S are picked up and shuffled through until the desired one is found. The necessary record or entry is made and the card returned to its proper place. For patients ill at home, cards are picked out and placed in the pocket-book before starting on morning rounds. Cards of convalescents are dropped. If two or more cards are found to be necessary for a long case, they may be fastened together with a brass clip.

Cards differing in colour are useful for special purposes, i. e., buff-coloured cards for consultation cases, salmon colour for surgical cases, and the like. Another method of special classification is to have two or more alphabetical indexes, the cards which belong to a special class of cases being ranged under the corresponding separate index, in the same box or drawer. Loose notes, letters, or sketches may be pasted to the case card or kept in envelopes of the same size as the cards and filed.

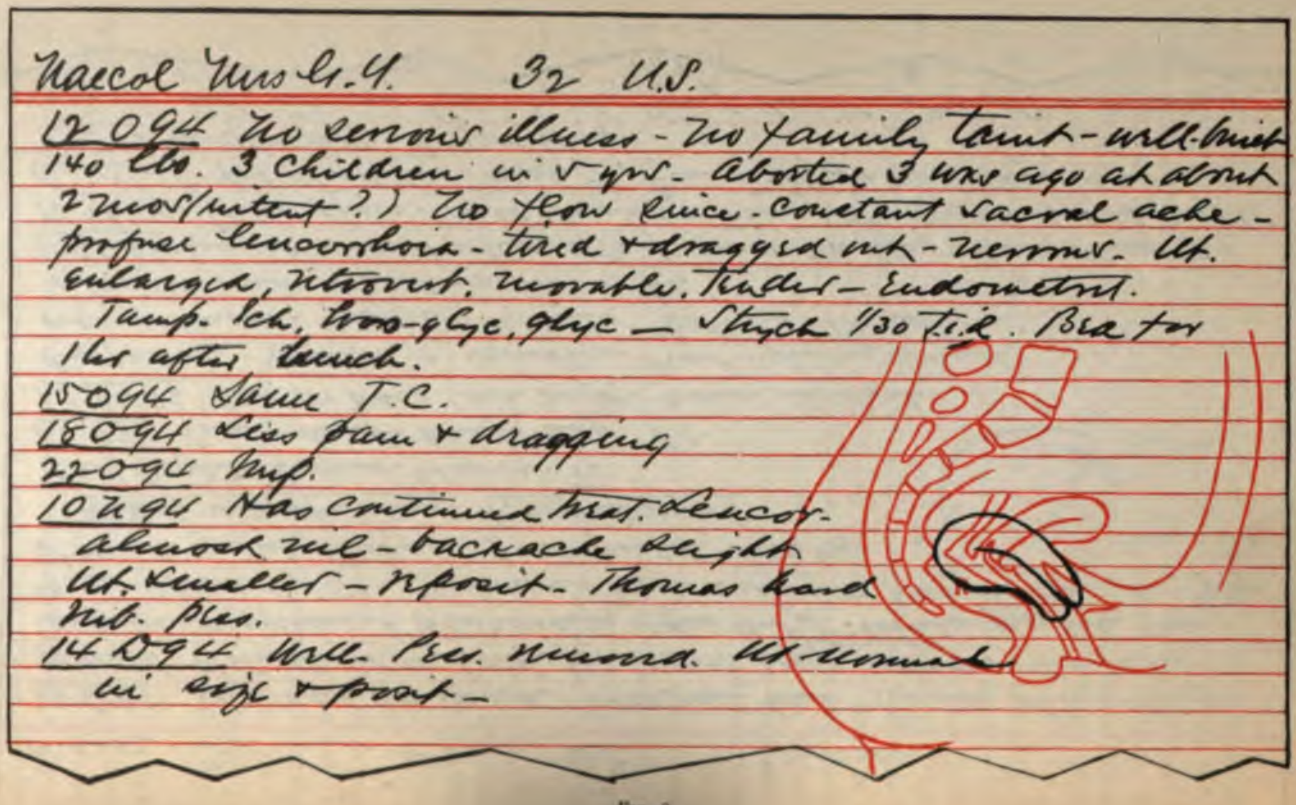


Cardiac - Mr C. 47 U.S. Journalist

22 D 95 Tall - well nourished. Good fam hist. Rheum when a child.
Indigest. - obstinate colds. Cough for several years with some dyspnoea
Card. dulness increased transversely - apex beat [illegible] outside nipple line. 6th
space. Apical systolic murmur transmitted & heard behind. Pulm.
2d sound accent. 1st sound poor quality. Pulse irreg.
Mitral incompetency. Hypert.-Dilatat.
℞ Ext Digitalis - Ammon Chlorid - Fe
28 D 95 Cough & dyspn. much imp. T.C
10 Ja 96 Ran for train last night - became suddenly
dyspnoeic - remains so - cyanotic. Heart action extremely
irreg. - w many ineffect. systoles. Go to bed. [illegible]
& Liq. Ammon. - Trinitrin 1/100 q. hr.
11 Ja 96 Seen at home. Partial orthop. Much starting when attempting
to sleep. Champagne - Milk punch. Trional [illegible] for sleep - otherwise T.C.
18 Ja 96 Impd steadily. Strych, Fe, Quin S. Allow up. P. still irreg
2 Fe 96 Subjectively well - P still irreg.
10 Fe 96 Fish dinner last night. Sharp diarrhoea this a.m. & much

Hair & F. Vomit Pul

Fig. 1.



Maccol Mrs G. Y.   32   U.S.

12 O 94 No serious illness - No family taint - well-built
140 lbs. 3 children in 5 yrs. Aborted 3 wks ago at about
2 mos (intent ?) No flow since. Constant sacral ache -
profuse leucorrhoea - tired & dragged out - nervous. Ut.
enlarged, retrovert. movable. tender - Endometrit.
Tamp. Ich, boro-glyc, glyc — Strych 1/30 t.i.d. Bed for
1 hr after lunch.
15 O 94 Same T.C.
18 O 94 Less pain & dragging
22 O 94 Imp.
10 N 94 Has continued treat. Leucor-
almost nil - backache slight
Ut. smaller - r'posit. Thomas hard
rub. pes.
14 D 94 Well. Pess. removed. Ut. normal
in size & posit.

FIG. 2.

Temperature cards (Chart I), urinalysis cards (Fig. 4), and cards for blood examinations (Chart IX) have been devised by De Forest.

(2) *Rubber Stamps.*—These are of two kinds—outline stamps and stamp forms for recording special data.

Outline Stamps.—These, as the name indicates, are rubber stamps representing in more or less detail various regions or organs of the body, and employed when it is desired to represent by the graphic method any changes of structure, the exact location and character of physical signs, the outline of tumours, etc. An ink pad is required, preferably red or blue. The case card is placed upon a level surface, the stamp inked, and adjusted on the desired place, and a quick, light blow given with the palm of the hand. On the outline thus printed may be sketched or written with pen or pencil whatever abnormalities it is desired to represent. Further changes which occur may be entered on the same outline, the date being appended, or a new print may be made. The use of the outline stamp has proved invaluable, because of the increased accuracy of observation to which it leads, even leaving out of consideration its value as a record. (See Figs. 1 and 2, red outlines.)

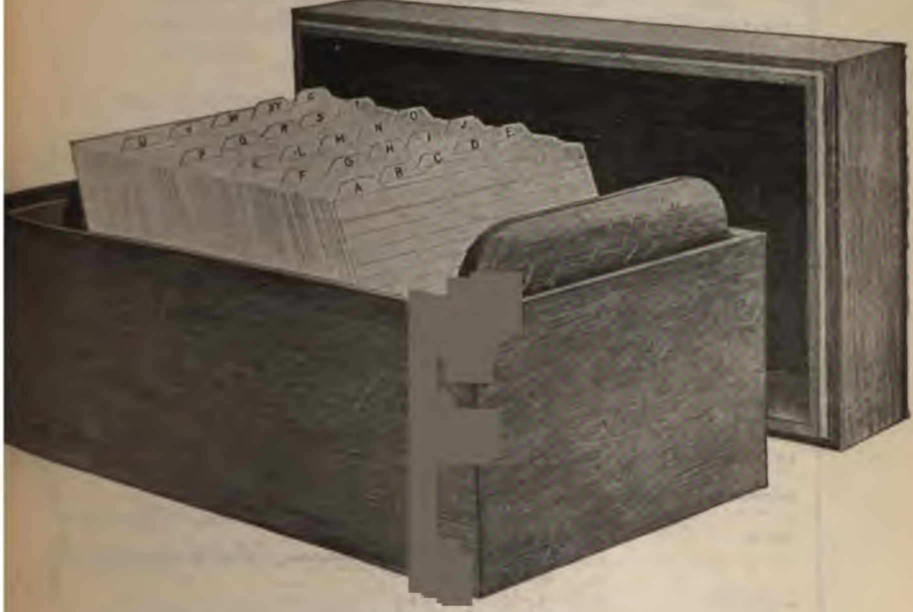
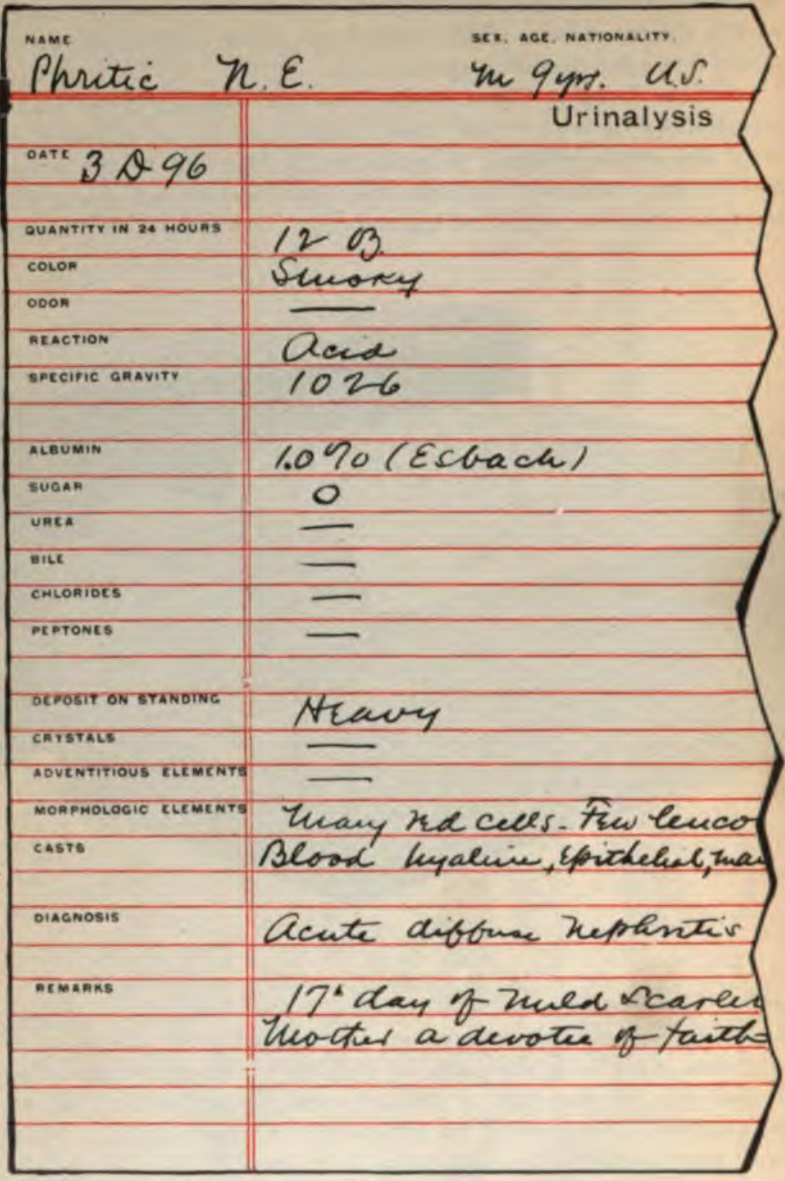


Fig. 3.



NAME	Phritic N. E.
SEX, AGE, NATIONALITY	M 9 yrs. U.S.
	Urinalysis
DATE	3 D 96
QUANTITY IN 24 HOURS	12 oz.
COLOR	Smoky
ODOR	—
REACTION	Acid
SPECIFIC GRAVITY	1026
ALBUMIN	1.0 % (Esbach)
SUGAR	0
UREA	—
BILE	—
CHLORIDES	—
PEPTONES	—
DEPOSIT ON STANDING	Heavy
CRYSTALS	—
ADVENTITIOUS ELEMENTS	—
MORPHOLOGIC ELEMENTS	Many red cells. Few leuco
CASTS	Blood hyaline, Epithelial, ma
DIAGNOSIS	Acute diffuse Nephritis
REMARKS	17th day of Mild Scarle Mother a devotee of faith-

FIG. 4.

Type Stamps.—These are used in noting any set of answers which require frequent asking, or which may be involved in special investigations, like the following example:

SHORT MED TALL FT IN : STOUT THIN WEIGHT LBS:
BLON BRU INDETER: EYES BLU BR BLK : SLIGHTLY VERY NEUROTIC:
MENTALITY HIGH MED LOW: SLEEPS WELL POORLY: APPT GOOD
POOR: VOMITG ERUCTS FULNESS DISCOMF PAIN IMMED HRS
AFTER BEFORE EATING: BOWELS HABITUAL OCCAS CONSTIP LOOSE
REG FLATULENCE: URINATION NOT FREQ PAINFUL: URINE AMT
INCREAS DIMIN: MENSES PAIN SLIGHT SEVERE NOT REG FREE
SCANTY VAG DISCH LABOURS SEVERE NO FEVER: TOBACCO HABIT
OCCAS MOD EXCESS: COFFEE TEA BOTH HABIT OCCAS MOD EXCESS:
PULSE RATE NOT REG TENSION HIGH LOW: ARTERIES HARD SOFT:
RESP RATE COSTAL ABDOM EXPANS GOOD POOR: TEMP

A large amount of writing is saved by this device. The imprint is made in the same manner as with the outline stamps. The words required are underlined, special emphasis indicated by double underlining, and doubtful points followed by a question mark. Any desired set of words can be made to suit individual requirements. The set should not cover a space greater than 1½″ × 3″, or 2″ × 4″, as a larger size will not print evenly. The stamp may be a self-inker or a hand stamp. The hand stamp is less expensive and, with care in printing, answers perfectly.

PART I

THE EVIDENCES OF DISEASE

SECTION I

CONSIDERATIONS, NEITHER SYMPTOMS NOR SIGNS, WHICH MAY QUALIFY OR SUGGEST A DIAGNOSIS

THE considerations which may qualify or suggest the diagnosis in a given case relate to the family history or hereditary tendencies, and the personal history up to the date of the present illness. The personal history includes age, sex, nationality, occupation, residence, habits, and previous diseases or injuries. Such considerations embrace also the chronological occurrence, seasonal or diurnal, of certain diseases, and the comparative infrequency of others.

I. **Family History.**—The family history of the patient is of much importance, because of the light which may be cast by it, not only upon the present illness, but also upon the constitution and tendencies of the patient.

Unfortunately, it is not always possible to obtain a complete and accurate family history. It is usually necessary to cross-examine the patient with some particularity, inquiring into the symptoms and duration of illnesses attributed to ancestors, and bearing in mind the approximate meanings of various popular terms, such as "gastric fever," "dropsy," "blood-poisoning," "teething," "cold," "nervous prostration," which latter may cover insanity or hysteria. "Old age" is frequently assigned as a cause of death, which has, of course, little meaning. "Childbirth," when assigned as a lethal cause, not infrequently proves to be a rapid phthisis pulmonalis.

Inquiries regarding certain diseases should be made very cautiously, because of the possibility of arousing feelings of shame or fear in the patient. It is better to ask if there is "lung trouble" in the family than to use the word "consumption." So, too, it is strongly advisable to ask after the symptoms without mentioning the names of suspected syphilis, tuberculosis, or cancer. There is a certain reproach to family or personal pride in the acknowledgment of the existence of some ailments, which may lead to the concealment of important information.

A full statement of the family history includes the nature of the illnesses (with the age of the living) and the causes of deaths (with the age at death) which may have occurred in the patient's parents, paternal and maternal grandparents, brothers and sisters. It is requisite at times to ascertain similar facts with reference to aunts, uncles, and cousins. It should be borne in mind that transmissible tendencies may pass over one generation.

It is important during this inquiry to bear in mind that certain diseases are either frequently associated or are manifestations of a common cause. In some cases there is a curious alternation between two diseases, one replacing the other—e. g., migraine and epilepsy. This may occur in the individual, or in alternating generations.

Heredity may be direct, the offspring showing the lesions of the disease at birth, as in syphilis. In the majority of cases, a certain stamp or type of tissue and organization is transmitted which renders the individual vulnerable to special micro-organisms, as in tuberculous subjects, or liable to perversions of the nervous system, or prone to degenerations and disturbances of metabolism.

CONGENITAL AFFECTIONS

The following diseases and conditions may be congenital:

Malformations.
Dislocated hip-joint.
Angeioma.
Nævi.
Ichthyosis.
Pemphigus.
Sclerema.
Dermatitis neonatorum.
Icterus neonatorum.
Syphilis.
Acute fatty degeneration (Buhl's disease).
Achondroplasia.
Osteomata.
Hæmophilia.
Hæmoglobinuria.
Hypertrophy of pylorus.
Atelectasis.
Idiocy.
Hydrocephalus.
Tetanus neonatorum.
Myotonia congenita (Thomsen's disease).
Infantile hemiplegia.
Progressive muscular atrophy.

A list of diseases and conditions which are considered to be transmissible in varying degrees follows. The bracketed groups are composed of those which have affiliations either of conjoint occurrence or common causes.

HEREDITARY DISEASES AND CONDITIONS

Rheumatism.
Cardiac diseases.
Chorea.
Nephritis.
Renal calculus.
Emphysema.
Bronchitis.
Psoriasis.

Insanity.
Hysteria.
Hypochondria.
Epilepsy.
Migraine.
Neuralgias.
Neurasthenia.
Many neuroses.

Tuberculosis.
Phthisis pulmonalis.
Tuberculous glands.
Tuberculous disease of bones.
Tuberculous peritonitis.
Tuberculous meningitis.
Hydrocephalus.

Premature senility.
Hæmophilia.
Diabetes.
Obesity.
Syphilis.
Alcoholism.
Acne.
Eczema.
Ichthyosis.
Psoriasis.
Leprosy.
Lichen.
Nævus.
Malformations.
New growths.
Arthritis deformans.

Gout.
Diseases of the liver.
Chronic nephritis, especially cirrhotic.
Renal calculus.
Angina pectoris.
Cardio-vascular disease.
Apoplexy.
Asthma.

Hernia.
Dupuytren's contraction.
Disseminated sclerosis.
Hereditary cerebellar ataxia.
Pseudo-hypertrophic paralysis.
Progressive muscular atrophy.
Hereditary peroneal atrophy.
Facio-scapulo-humeral atrophy.
Laryngismus stridulus.
Tetany.
Paramyoclonus multiplex.
Huntington's chorea.
Thomsen's disease.
Friedreich's ataxia.
Adiposis dolorosa.

II. **Age.**—Anatomical structure varies with age, and physiological processes have peculiarities which are characteristic of different periods of life. Moreover, the effects of environment, occupation, habits, the beginning and end of sexual life, and the wearing out of the organism by constant friction, are necessarily manifested at varying ages. Consequently there is a distinct preponderance in the frequency of certain diseases or classes of disease at special age periods. The diseases of youth are often the direct progenitors of those of old age, and the life of the individual may be a constant struggle with diseased conditions which began in antenatal life.

(1) *Infancy and Childhood.*—At this age there is a special liability to digestive disorders, because of the relatively large amount of work required to meet the pressing needs of a rapidly developing organism, and because of the peculiar susceptibility to infection of the gastro-intestinal tract in children. Inflammations of the respiratory apparatus are frequent, because of the tendency of the cells of the mucous membranes to undergo rapid proliferation under slight and unaccustomed irritations. Nervous diseases and reflex disturbances are common for the reason that the cerebro-spinal and sympathetic apparatus is developing, and has not yet settled into habits of regular

innervation. This is also the age, above all others, at which the organism is susceptible to certain infectious diseases—the exanthemata.

The following diseases are those which are most common at this age period. Some are congenital. Diseases mentioned under a given head may begin later than the period under which they are classed.

Eczema.
Intertrigo.
Impetigo contagiosa.
Pemphigus.
Seborrhœa.
Strophulus.
Ringworm.
Malformations.
Nævi.
Soft cataract.
Bronchitis.
Broncho-pneumonia.
Laryngismus stridulus.
Spasmodic laryngitis.
Laryngeal diphtheria.
Œdema of larynx.
Adenoids.
Post-pharyngeal abscess.
Noma.
Amyloid disease.
Chloroma.
Rachitis.
Hæmophilia.
Infantile scurvy.
Endocarditis.
Primary renal sarcoma.
Hypertrophic cirrhosis of liver.
Diarrhœal diseases.
Intussusception.
Lumbrici.
Congenital syphilis.
Diphtheria.
Mumps.
Measles.
Roseola.
Rötheln.
Scarlatina.
Cerebro-spinal meningitis.
Varicella.
Variola.
Tuberculosis of bones and lymph glands.
Cretinism.
Idiocy.
Convulsions.
Chorea.
Epilepsy.
Hydrocephalus.
Infantile paralysis.
Pseudo-hypertrophic paralysis.
Progressive muscular atrophy.
Tetany.
Nodding spasm.
Meningitis.
Friedreich's disease.
Thomsen's disease.
Little's disease.

(2) *Puberty and Adolescence:*

Acne.
Seborrhœa.
Anæmia.
Chlorosis.
Myoma.
Chondroma.
Myeloma.
Exostoses.
Sacro-iliac disease.
Acute tuberculosis.
Mollities ossium.
Addison's disease.
Diabetes insipidus.
Chloroma.
Myxœdema.
Exophthalmic goitre.
Simple goitre.
Disease of lymph glands.
Catalepsy.
Trance.

Pulmonary tuberculosis.
Typhoid fever.
Mumps.
Rheumatic fever.
Rötheln.
Diphtheria.
Pharyngomycosis.
Œdema of the larynx.
Gastric ulcer.
Appendicitis.
Myocarditis, acute.
Endocarditis.
Epilepsy.
Hysteria.
Acute dementia.
Meningitis, all varieties.
Cerebral embolism and thrombosis.
Syringomyelia, beginning.
Cerebellar ataxia.
Friedreich's disease.
Spasmodic spinal paralysis.
Periodic paralysis.
Spinal apoplexy.

(3) *Early Adult Age:*

Acute rheumatism.
Endocarditis.
Peliosis rheumatica.
Pharyngomycosis.
Acute tuberculosis.
Pulmonary phthisis.
Acute yellow atrophy.
Typhoid fever.
Actinomycosis.
Gastric ulcer.
Gastralgia.
Myoma.
Cerebral embolism.
Cerebral abscess.
Hæmorrhage into cord.
Disseminated sclerosis.
Syringomyelia.
Landry's paralysis.
Progressive muscular atrophy.
Periodic paralysis.

(4) *Middle Age:*

Exophthalmic goitre.
Myxœdema.
Diabetes.
Gout.
Mollities ossium.
Addison's disease.
Cirrhosis of liver.
Acute yellow atrophy.
Weil's disease.
Gallstones.
Gastric ulcer.
Stricture of rectum.
Colonic ulcer.
Chronic nephritis.
Cysts of kidney.
Myocardial diseases.
Endocarditis.
Aneurism.
Angina pectoris.
Dupuytren's contraction.
Leucæmia.
Pernicious anæmia.
Myoma.
Epithelioma.
Carcinoma.
Apoplexy.
Intracranial tumours.
Hypochondriasis.
Melancholia.
Psychosis polyneuritica.
Locomotor ataxia.
Myelitis.
Anterior poliomyelitis.
Intraspinal hæmorrhage.
Syringomyelia.
Landry's paralysis.
Ataxic paraplegia.
Progressive muscular atrophy.
Dementia paralytica.
Paralysis agitans.
Disseminated sclerosis.
Sciatica.
Paralysis of serratus magnus.

(5) *Old Age:*

Pruritus.
Ecthyma.
Pemphigus.
Epithelioma.
Carcinoma.
Gout.
Prostatic disease.
Cataract.
Arteriosclerosis.
Gangrene.
Myocardial diseases.
Aneurism.
Angina pectoris.
Broncho-pneumonia.
Bronchitis.
Cerebral apoplexy.
Pachymeningitis.
Paralysis agitans.
Melancholia.

III. **Sex.**—Putting aside the diseases due to differences in structure and function between male and female, there remain certain maladies which occur more frequently in one sex than in the other. These discrepancies are caused mainly by the manner of life. Men suffer especially from diseases induced by exposure, hard physical or mental work and worry, and by the acquirement of injurious habits. Women lead an indoor life, and many are harassed by household and domestic anxieties. If not occupied by domestic cares, or if without definite aims and interests, a habit of morbid self-examination is apt to be formed. Moreover, the nervous system in women is normally more unstable in its equilibrium. Because of all these factors, functional nervous diseases (neuroses) are much more common in women than in men.

The following list, which is by no means exhaustive, contains some of the more common diseases, classified according to sex frequency. The figures are only approximate.

(1) *Males:*

Alcoholism.
Gout.
Diabetes insipidus and mellitus, 2 to 1.
Addison's disease, 2 to 1.
Peliosis rheumatica.
Hæmophilia, 11 to 1.
Leucæmia, 2 to 1.
Pseudo-leucæmia, 3 to 1.
Chloroma, 19 to 7.
Hæmoglobinuria.
Diseases of larynx.
Pneumonia.
Emphysema.
Valvular diseases of heart.
Malignant endocarditis, 3 to 1.
Angina pectoris.
Cysts of kidney, 2 to 1.
Carcinoma of kidney.
Colour blindness.
Hæmatoma auris.
Exostoses.
Angeioneurotic œdema.
Dupuytren's contraction, 20 to 1.
Typhoid fever.
Cerebro-spinal meningitis.
Mumps.
Actinomycosis, 3 to 1.
Apoplexy.
Cerebral abscess.
Simple meningitis, 2 to 1.
Pachymeningitis.
Bulbar paralysis.

Myocarditis.
Fatty heart, 2 to 1.
Aneurism, of all varieties.
Aneurism of abdominal aorta, 8 to 1.
Chronic gastritis.
Carcinoma of stomach.
Gastric cirrhosis.
Appendicitis, 7 to 3.
Intussusception.
Carcinoma of rectum.
Cirrhotic liver, 3 to 1.
Weil's disease.
Pancreatitis.
Interstitial nephritis, 2 to 1.
Intraspinal hæmorrhage.
Spinal meningitis.
Landry's paralysis, 3 to 1.
Amyotrophic lateral sclerosis.
Progressive muscular atrophy.
Syringomyelia, 2 to 1.
Paralysis agitans.
Pseudo-hypertrophic paralysis.
Ataxic paraplegia.
Thomsen's disease.
Dementia paralytica.
Epilepsy.
Hypochondriasis.

(2) *Females:*

Anæmia.
Chlorosis.
Erythema nodosum.
Lupus erythematosus, 5 to 1.
Scleroderma.
Gastralgia.
Gastric ulcer.
Enteroptosis and floating kidney, 15 to 1.
Constipation.
Pulsating aorta.
Mucous colic.
Stricture of rectum.
Acute yellow atrophy.
Gallstones, 7 to 2.
Tuberculous peritonitis, 2 to 1.
Mitral stenosis.
Myxœdema.
Goitre, simple and exophthalmic.
Osteomalacia, 30 to 1.
Diphtheria.
Pharyngomycosis.
Acute dementia.
Catalepsy.
Trance.
Hysteria.
Neurasthenia.
Acroparæsthesia.
Chorea, 3 to 1.
Facial hemiatrophy.
Adiposis dolorosa.
Raynaud's disease.

In women the regularity, profuseness, and attendant pain of the menstruation, the number of pregnancies and miscarriages and their sequelæ should be ascertained, for the reason that deviations from the normal in these respects may be of much importance as possible causes of subsequent disease.

IV. **Nationality.**—The susceptibility or its opposite, immunity, possessed by certain races has been commented upon by some observers. Among these may be noted the liability of the Jewish race to diabetes, of the Scandinavian and African to phthisis pulmonalis, and the comparative immunity of the African to yellow fever.

V. **Occupation.**—With reference to the effects of occupation in causing disease, it will be found necessary to ascertain the details of the patient's employment, whether active or sedentary in character, and whether or not it requires the handling or breathing of toxic or irritating substances. Possible overuse of the eyes, playing wind instruments, and the care of domestic animals, are other details a knowledge of which may be useful. All previous occupations should be ascertained. It should be remembered that the state of health sometimes enforces the occupation.

(1) *Diseases Incident to Active Occupations:*

Aneurism.
Pneumonia.
Rheumatism.

(2) *Diseases Incident to Sedentary Occupations, including Mental Work:*

Anæmia.
Chlorosis.
Constipation.
Digestive disorders.
Gallstones.
Hæmorrhoids.
Hysteria.
Hypochondriasis.
Neuroses.
Obesity.
Gout.
Ulcer of the stomach.
Phthisis pulmonalis.

(3) *Diseases Incident to Special Occupations:*

Pulmonary phthisis.—Accountants, book-keepers, clerks, compositors, printers, pressmen, marble and stone cutters, miners. *Fibroid phthisis, or chronic laryngitis from dust.*—Grinders, file cutters, potters, glass polishers, wool and cotton spinners, millers. *Anthrax.*—Skin handlers. *Lichen.*—Grocers, bakers, and bricklayers. *Internal anthrax.*—Wool and rag sorters. *Glanders and tetanus.*—Hostlers. *Actinomycosis.*—Grain handlers. *Aspergillosis.*—Bird dealers and grain handlers. *Psittacosis.*—Bird dealers. *Anæmia, gastric ulcer, eczema, erythema nodosum.*—Domestic servants (female). *Varicose veins.*—Coachmen, shop girls, and others accustomed to long maintenance of the standing or part standing position. *Writer's cramp (scrivener's palsy).*—Clerks and writers. *Septic infection.*—Butchers and slaughterhouse employees. *Conjunctivitis.*—Electric-light workers. Probably caused by actinic rays. *Nystagmus.*—Miners. *Emphysema.*—Players upon wind instruments. *Insomnia, dyspepsia, disease of liver and kidneys, neurasthenia, irritable heart, apoplexy, and paralysis.*—Brain workers. *Typhoid fever, pneumonia, cardio-vascular and renal disease, morphine and cocaine habits.*—Physicians. *Lead poisoning, gout.*—Lead miners and smelters, potters, painters, glass-polishers, gilders, plumbers, dyers, makers of white and red lead, seamstresses (from silk thread loaded with acetate of lead), makers of artificial flowers. *Mercurial poisoning.*—Cinnabar miners, makers of cheap looking glasses or mirrors, makers of felt hats (from the bath of acid nitrate of mercury used to promote felting), furriers. *Arsenical poisoning.*—Wall-paper workers (formerly), workers on artificial flowers and fancy glazed-paper boxes, furriers. *Phosphorus poisoning.*—Matchmakers. *Chromium and zinc poisoning.*—"Founders' ague" in brass foundries.

Disease of hair follicles.—Operatives in oil refineries and paraffin works. *Deafness.*—Boiler-makers. *Caisson disease.*—Divers and workers in compressed air. *Laryngeal spasm.*—Speakers, cornet and flute players. *Clonic or tonic spasms of one or more extremities.*—Typewriters, pianists, telegraph operators, violinists, blacksmiths, tailors and seamstresses, shoemakers, professional dancers.

VI. **Residence.**—A knowledge of the place of residence may be of considerable importance, if not with regard to diagnosis, at least with regard to the prophylaxis of future attacks. In the diagnosis of suspected cases of cholera, yellow fever, and the pernicious or severe malarial fevers, the fact of the patient having visited or lived in countries or localities where they are prevalent may furnish a clew otherwise lacking. Goitre, rachitis, calculus, cretinism, dysentery, and lung diseases have at times special affinities with certain localities.

The following list comprises the more important geographical associations of disease, which may be of diagnostic value in connection with a patient fresh from residence in the countries named:

Africa.—Dengue, Guinea-worm disease.

Africa, South.—Bilharzia hæmatobia.

Africa, West Coast.—Yellow fever, frambœsia.

America, South.—Chigoe, ainhum (negro).

Arabia.—Bilharzia hæmatobia.

Canada, New Brunswick, Cape Breton.—Leprosy.

China.—Beri-beri, bilharzia hæmatobia.

Egypt.—Bilharzia hæmatobia, plague.

England, Certain Counties.—Renal calculus.

Europe, Large Cities.—Rachitis.

France, South of.—Pellagra.

India, East.—Beri-beri, Delhi boil, dengue, bilharzia hæmatobia, Asiatic cholera, frambœsia, Guinea-worm disease, ainhum (negro).

West Indies.—Chigoe, dengue, yellow fever, frambœsia.

Italy.—Goitre, cretinism, pellagra.

Japan.—Beri-beri, bubonic plague, bilharzia hæmatobia.

Malta.—Malta fever.

Morocco.—Plague.

Naples.—Malta fever.

Norway.—Leprosy.

Spain.—Pellagra, goitre (Pyrenees).

Switzerland, Alps.—Goitre, cretinism.

Syria.—Plague.

Tropical Regions in General.—Epidemic dysentery, pernicious intermittent and remittent fevers, acute hepatitis and hepatic abscess, leprosy, filaria sanguinis hominis.

United States, Southern Portion, especially the Gulf States.—Pernicious intermittent and remittent fevers, yellow fever (epidemic), dengue, leprosy, filaria sanguinis hominis, ainhum (negro).

Other items embraced under the head of residence concern the effects of climate, city or country, seashore or inland residence, and the sanitary condition of dwellings with reference to ventilation, drainage, heating, cleanliness, and water supply.

VII. **Habits.**—The habits formed by individuals are so closely interwoven with age, occupation, and residence that it is only necessary to include here the possible existence of alcohol, opium, cocaine, or other drug addiction. Diet; the use of tea, coffee, and tobacco; clothing, sleep, and exercise are largely governed by the social condition and environment. In men, the habits as to the frequency of sexual intercourse and a history of early self-abuse require investigation. Ascertain also the kind and amount of alcoholic beverages taken and the time of taking—i. e., before, during, or between meals (with reference to the causation of sclerotic changes in stomach, liver, kidneys, arteries, etc.). How much tobacco is used, of what kind, and in what manner (with reference to naso-pharyngeal catarrh, nervousness, cardiac neuroses).

Both men and women should be interrogated as to the amount and strength of tea and coffee taken daily. The "tea-and-bread" habit is mainly found in women.

VIII. **Previous Diseases or Injuries.**—A knowledge of prior illnesses and injuries is of value, provided that their date, nature, and severity can be ascertained, for three reasons:

(1) The history of a previous attack of certain diseases *renders subsequent attacks probable.* Among such diseases are:

Malarial fevers.
Influenza.
Erysipelas.
Diphtheria.
Pneumonia.
Acute rheumatism.
Tonsilitis (follicular and phlegmonous).
Gout.
Bronchitis.
Asthma.
Nephritis.
Renal colic.
Intermittent hæmoglobinuria.
Lead poisoning (colic).
Appendicitis.
Angina pectoris.
Neuralgias.
Migraine.
Delirium tremens.
Convulsions (infantile or epileptic).
Apoplexy.

(2) With other diseases, a *previous attack*, as a rule, *negatives* its subsequent occurrence. Among these are:

Measles (not uniformly).
Parotitis (epidemic).
Pertussis.
Rötheln. Scarlatina.
Typhus fever.
Variola.
Varioloid. Varicella.
Yellow fever. Typhoid fever.

(3) A history of the previous existence of certain diseases or in-

juries may throw light upon present conditions which stand in the relation of *sequelæ* to the primary ailments. Examples of these are:

Syphilis, followed by skin eruptions, alopecia, ulcers, periostitis, gummata, amyloid diseases, locomotor ataxia, dementia paralytica, and other affections of the nervous system, arterio-sclerosis, etc. *Gonorrhœa*, with reference to gonorrhœal rheumatism, orchitis, stricture, conjunctivitis, and pelvic tubal inflammations in the female. *Scarlet fever*, with subsequent middle ear inflammations, renal disease, and rheumatism. *Rheumatism*, initiating chronic processes which result in valvular cardiac lesions, and renal disease; also associated with chorea. *Septic* or *suppurating foci*, leading to subsequent embolic or general inflammations of heart, lungs, liver, pleura, or peritoneum. *Lead poisoning* causing gout; and *gout* or lead poisoning producing a chronic interstitial nephritis.

A history of a fall or other injury may be of some value in connection with suspected meningitis, disease of the spine, Jacksonian epilepsy, and arthritis. A previous surgical operation may point to the possibility of a recurrence of the condition which required operative interference.

(4) There are certain diseases of which a diagnosis should be made with caution because of the *infrequency of their occurrence*, or the difficulty of their recognition. A provisional diagnosis is justified in many cases, but a positive diagnosis of the diseases in the following list demands good evidence, not mere conjecture. This list applies to the general practitioner. The specialist in certain lines may and does have a different experience.

Leprosy.
Scleroderma.
Morphœa.
Acanthosis nigricans.
Keloid.
Lichen ruber.
Sclerema.
Trichiniasis.
Actinomycosis.
Hydatids (except of liver).
Psittacosis.
Paratyphoid fever.
Noma.
Hydrophobia.
Anthrax.
Pharyngomycosis.
Glanders.
Aspergillosis.
Hæmophilia.
Peliosis rheumatica.
Banti's disease.
Leucæmia.
Hodgkin's disease.
Chloroma.
Myxœdema.
Pancreatic disease, except carcinoma.
Inflammation of spleen.
Chyluria.
Infantile hæmoglobinuria.
Atrophy of brain and porencephalus.
Amyotrophic lateral sclerosis.
Spinal intermeningeal hæmorrhage.
Syringomyelia and Morvan's disease.
Acute myelitis.
Hæmatomyelia.
Pseudo-hypertrophic paralysis.

Polymyositis.
Spondylitis.
Osteomalacia.
Myositis ossificans.
Osteitis deformans.
Leontiasis ossea.
Acromegaly.
Achondroplasia (fœtal rickets).
Addison's disease.
Acute yellow atrophy.
Weil's disease.
Dextrocardia.
Tuberculosis of pericardium.
Pyo- or pyopneumopericardium.
Abscess, aneurism, or rupture of heart.
Coronary thrombosis.
Primary tricuspid valve lesions.
Acquired pulmonary valve lesions.
Aortic stenosis (relatively rare).
Aortitis.
Aneurism of pulmonary artery.
Periarteritis nodosum.
Stokes-Adams's syndrome.
Fat embolism.
Scapulo-humeral paralysis.
Facio-scapulo-humeral atrophy.
Hereditary peroneal atrophy.
Friedreich's disease.
Hereditary cerebellar ataxia.
Landry's paralysis.
Spastic cerebral paralysis.
Adiposis dolorosa.
Erythromelalgia.
Reynaud's disease.
Meralgia paræsthetica.
Acroparæsthesia.
Nodding spasm.
Huntington's chorea.
Dubini's chorea.
Athetosis.
Catalepsy.
Myasthenia gravis.
Hysteria (in men).
Myotonia congenita.
Tetany.
Periodic paralysis.
Ophthalmoplegia.

(5) There is little of diagnostic value to be gained from the varying statistics of the *seasonal prevalence of disease*, beyond the broad statement that diarrhœal diseases predominate during the summer months, while pulmonary disorders and rheumatic affections are most prevalent in the winter and early spring. Zymotic diseases occur in largest number during the cold season, but this is to be explained rather by the opening of the schools and the closing of house windows than by the effect of season *per se*. Typhoid fever has a notable seasonal incidence in the autumn months.

(6) It may be mentioned here that certain diseases either begin or show an *exacerbation of symptoms at special diurnal periods*. Bronchial asthma is apt to make its onset or to intensify in severity in the early morning hours; spasmodic croup, as well as diphtheritic stenosis of the larynx, between 10 and 12 at night. The suffering from painful diseases is usually worse at night, and in febrile disorders the temperature generally reaches its highest point between 7 and 8 P. M. The paroxysms of whooping-cough are more frequent and severe at night. The pain due to diseases of the bones and joints presents a nocturnal aggravation.

(7) It is well to remember that some ailments present a more or less regular *periodicity of recurrence*. Among these are neuralgias,

migraine, pseudo-angina, epilepsy, periodic paralysis, relapsing fever, malarial infections, ulcerative endocarditis, paroxysmal hæmoglobinuria, pyelo-nephritis, bronchial asthma, and menstrual disorders.

(8) A history of *alcoholism* is important as explanatory of the presence of the following diseases; in their prognosis; or as suggestive in regard to their possible occurrence in a given individual: Myocarditis, arteriosclerosis, aneurism, cirrhosis of liver, chronic pharyngitis, chronic gastritis, pneumonia, emphysema, tuberculosis, chronic nephritis, apoplexy, delirium tremens, neuritis, pachymeningitis, Korsakoff's disease, and gout.

(9) The following diseases are essentially chronic (figures from Leftwich):

Chorea (1 to 4 months).
Dementia paralytica (months to years).
Idiopathic muscular atrophy (years).
Locomotor ataxia (1 to many years).
Chronic myelitis (6 months to 10 years).
Disseminated sclerosis (5 to 10 years).
Syringomyelia (5 to 20 years).
Acromegaly (10 to 20 years).
Exophthalmic goitre (months to years).
Addison's disease (2 to 3 years).
Lymphadenoma (2 years).
Leucæmia (6 months to 7 years).
Interstitial nephritis (4 to 10 years).
Amyloid disease (years).

SECTION II

THE HISTORY OF THE PRESENT ILLNESS

It is desirable to obtain a full and accurate history of the present illness, as in all cases it is more or less necessary, in some absolutely essential. The greater part of this history is subjective, but there may have been some symptoms sufficiently objective to have been observed by the patient, such as œdema or hemorrhages.

It is in obtaining this history that the largest draughts are made upon the tact and experience of the physician. The patient may be one of the odd people from whom it is difficult to extract more than a monosyllabic answer, or may be so talkative that a question is slipped in only after patient waiting for a pause. Dense ignorance may be an obstacle, so also may false modesty or shame. Exaggeration of symptoms, a not uncommon failing, must be guarded against; less often its opposite, a stoic pride in making light of pain. These and other difficulties (e. g., malingering) require the exercise of some skill in the art of cross-examination.

Except in the case of suspected malingering, where the answer may flatly contradict the alleged condition, leading questions are to be avoided, especially with impressionable or ignorant patients. For instance, it is better to say, "Did you have any pain in the head?" than "You had pain in the head, did you not?" The first question is a simple interrogation, which may elicit the reply, "Yes" or "No." The second almost forces the answer "Yes." Care is to be taken lest the patient's story should be too narrowly limited, otherwise a knowledge of important symptoms may not be gained. It is better to expend additional time and patience, which may be utilized in a careful scanning of the general appearance, behaviour, and temperament of the patient, than to miss a possibly vital point in the history.

The physician's object in this portion of the examination is to gain a clear conception of the origin and course of the disease up to the present time. The inquiry should therefore be conducted with reference to the following points:

(1) **Possible Exciting Causes.**—The most important, with reference to early diagnosis, is a known exposure to some infectious disease. Other causes of consequence are fatigue of mind or body, dietetic imprudences, toxic agents, and chilling of the body or "taking cold." This last factor is frequently assigned by patients as a satisfactory etiological explanation of the most diverse ailments, and is a convenient substitute for other demonstrable causes.

(2) **Date and Manner of Onset.**—A definite statement of the time of onset will generally place the disease in one of two categories, acute or chronic. Nevertheless it must not be forgotten that an acute attack may be an expression, perhaps the first, of some underlying and causal disease or condition; for instance, uræmic convulsions in renal disease. As a rule, however, acute diseases begin suddenly, while with chronic maladies a long period may elapse before the symptoms force themselves upon the attention of the patient. Lobar pneumonia and cirrhosis of the liver may be cited as illustrations, respectively, of acute and chronic diseases.

The manner of onset should be accurately ascertained, strictly separating the symptoms which initiated the attack from those which appeared at a later period, for otherwise their relative importance may not be appreciated.

Closely connected with the date and manner of onset are the—

(3) **Subsequent Symptoms in the Order of Appearance up to the Present Time.**—The value of a strict chronological history of the symptoms which succeed the onset of any disease, can not be overestimated. In some, as typhoid fever, the diagnosis may depend largely upon the order of evolution of the present symptoms. Each symptom should be elicited and the time of its appearance borne in mind.

(4) **Symptoms now Present.**—The present sufferings and complaints of the patient should be attentively listened to, as in connection with previous symptoms they may determine the direction of the first step in the special objective examination. Thus, a colicky pain in the abdomen will prompt an immediate palpation of the appendical region. Moreover, the present condition may demand instant palliative or other treatment, as in pulmonary hemorrhage, before the physician is able to proceed with his special investigations.

One thing more is needed to complete the antecedent history—namely, information as to—

(5) **Previous Treatment.**—It is rarely possible to obtain reliable information in regard to previous treatment. The statements of patients with reference to the therapeutic agencies used by previous physicians are ordinarily quite untrustworthy. But, if such knowledge can be obtained from authoritative sources, it may be of considerable value. Thus, certain symptoms, otherwise unaccounted for, may be explained as due to the administration of certain drugs (acne from bromides, dilated pupils from atropine, etc.). The "therapeutic test" may have been applied, and the result weigh for or against a certain diagnosis (quinine in malaria, in the absence of a blood examination; mercury and iodides in suspected syphilis).

SECTION III

DIAGNOSTIC INDICATIONS FROM THE GENERAL APPEARANCE

THE general appearance is studied with reference to the dress, height, and weight; amount and character of adipose and muscular tissue; complexion, colour of hair and eyes; diathesis and cachexia.

I. **Dress and General Behaviour.**—An occasional hint may be derived from the clothing. Omission to use fastenings which may be needed for common decency, the coat or trousers buttoned with the wrong buttons, a vest soiled with droppings of food, may indicate the mental enfeeblement of a psychosis or chronic alcoholism. Clothing wet and of an ammoniacal odour is found in cases of incontinence of urine and cystitis. Diabetic urine in drying may leave a white deposit of glucose. The patient may be so crowded and bulging in his clothes as to suggest a recent rapid increase of bulk from obesity or general dropsy. The shoes may be left partly or entirely unfastened from forgetfulness, gout, rheumatism, or œdema; or slit for similar reasons, or because of corns, bunions, or injury; or worn more on one side, or in front, or at the heel, because of paralysis, deformity, or disease of the joints.

In meeting people unprofessionally one forms an unconscious judgment of character and peculiarities based upon the observation of small details of behaviour. These judgments are often useful outside of strictly technical lines in estimating the value of information received, its reliability, completeness, and freedom from exaggeration. The furtive look and the inability to meet squarely the eye of the physician may indicate mental weakness, morbid suspicion, or an intention to deceive. This manner, however, is not always to be interpreted as stated, for there are absolutely upright people who, because of ingrained timidity or bashfulness, will present a most striking but quite baseless hangdog manner and expression.

II. **Height and Weight** should be ascertained, if practicable, by actual measurement, but the statements of the patient, and the rough estimate which may be made by the eye, are sufficient for diagnostic purposes. Height is qualified by the adjectives dwarfish, short, medium, tall, very tall. Similar qualifying terms with reference to weight comprise emaciated, thin, spare, medium, stout, obese. When emaciation is extreme, and attended with a general failure of strength and vitality, it is called marasmus.

The relation of height to weight is by no means a fixed ratio, and is of little consequence in diagnosis. Very considerable variations may exist without indication of disease and without creating a predisposition thereto. An abnormally large disparity, other things being equal, shows that the balance of nutrition is disturbed, and in consequence the chances of longevity are lessened.

HEIGHT-WEIGHT RATIO

5	feet	0	inches	in height	should weigh	115	pounds.
5	“	1	“	“	“	120	“
5	“	2	“	“	“	125	“
5	“	3	“	“	“	130	“
5	“	4	“	“	“	135	“
5	“	5	“	“	“	140	“
5	“	6	“	“	“	145	“
5	“	7	“	“	“	150	“
5	“	8	“	“	“	155	“
5	“	9	“	“	“	160	“
5	“	10	“	“	“	165	“
5	“	11	“	“	“	170	“
6	“	0	“	“	“	175	“
6	“	1	“	“	“	180	“
6	“	2	“	“	“	185	“
6	“	3	“	“	“	190	“

Although the weight of the body at any one time has comparatively little diagnostic value, it is otherwise with increase or loss of weight as compared with former measurements. Change in the weight of the body, especially in some chronic diseases, such as tuberculosis, is in most cases a reliable index of the tendency of the malady, progressive loss indicating an advance of the disease, while progressive increase in weight follows a lessened activity. In this connection it is important to remember that general anasarca, ascites, and bulky abdominal or other tumours may cause a misleading increase of body weight.

In general, persons of moderate height and weight are best adapted to pass successfully through the ordinary trials of life and the extraordinary ordeals of disease. But, just as the thin, "wiry" individual may accomplish an enormous amount of work without excessive fatigue and emerge triumphantly from severe illness, so there are stout (in the sense of obese) and hearty people who decline to grow thin or to be worn out or to degenerate, and will live to old age in spite of serious acute diseases.

III. **Amount and Character of Adipose and Muscular Tissue.**—The subcutaneous fat is a large element in the weight. Normally it is distributed quite equally over the body, but may accumulate excessively on some particular part, especially the abdomen. In women after the menopause, and in men after the age of 45, there is frequently a marked increase in its amount. This increase is most commonly seen in sedentary persons who consume large quantities of food, although not uncommonly it appears to be an inherited tendency. The quality of the subcutaneous fat is to be estimated by the touch: good, if firm and elastic; poor, if soft and flabby.

Loss of weight is ordinarily first observed as a diminution of the subcutaneous fat. When this diminution is extreme, the skin becomes loose, owing to the loss of its foundation, is wrinkled, and can be raised in folds. On the other hand, in obese persons the skin may be overstretched, so that lines resembling the familiar lineæ albicantes of pregnancy can be seen upon the buttocks and abdomen.

The *muscles* may be large or small as compared to the bones. Size, however, is not of so much importance as quality. It is better to possess muscles which are soft and of moderate bulk, but yet firm, elastic, and quick-acting. Flabby, relaxed, firm, large, small, are the terms employed in describing the quality of muscles.

The *bones*, by comparison, may be large, thick, and prominent, or small, slender, and inconspicuous. To a considerable extent, the visibility of bony angularities depends upon the amount of subcutaneous fat. The characteristic rounded curves of well-developed women and children arise not only from the smaller bones, but also from the thicker covering of fatty tissue which belongs normally to the sex and age.

Special deformities and alterations in the shape of the skeleton, either in whole or in part, are caused by certain morbid processes, and, with associated symptoms, constitute recognised forms of disease.

The Diagnostic Import of Changes in Weight

As alterations in weight are largely dependent upon changes in the volume of the subcutaneous fat and the muscles, it is proper to summarize the principal conditions in which the weight progressively increases, progressively decreases, or remains stationary, omitting normal conditions (rich food, sedentary life, middle age, menopause; or hardships and poor food).

It is *stationary* or increases slightly in chloro-anæmia, and slight continuous or frequently recurring hemorrhages, as from bleeding hemorrhoids.

It *progressively increases* in pathological obesity; often also in alcoholism, rachitis, adiposis dolorosa, myxœdema, cretinism, dementia, pseudo-hypertrophic paralysis, and disseminated sclerosis.

It *progressively diminishes* in

Addison's disease,
Hysterical anorexia,
Cancer,
Diabetes (especially),
Chronic diarrhœa,
Long-continued fevers,
Prolonged lactation,
Marasmus,
Stricture of esophagus,
Obstruction of pylorus,
Ulcer of stomach,
Chronic suppurations,
Obstruction of thoracic duct,
Tuberculosis (all varieties).

As a symptom, progressive emaciation is most important in chronic diseases, and during convalescence from acute diseases. Continuing loss of weight in the latter case may point to approaching or co-existing chronic disease.

IV. The Conformation of the Body.—From the purely æsthetic point of view the painter or sculptor rarely, if ever, finds a figure corresponding to the ideal which is in his mind. Certain of the imported photographs used as a basis for illustrative diagrams in this volume have been made from professional models, and yet in nearly all there are obvious defects when a comparison is made with the acknowledged masterpieces of figure painting.

From the medical point of view one may rather easily recognise certain abnormalties in the configuration of the body which are (*a*) congenital and predispose toward disease, or (*b*) acquired as results, and are signs of disease.

(*a*) There are two congenital types of body which predispose to disease.

1. Tall, thin subjects, with small bones, slender ribs, and a long, narrow thorax, are predisposed toward tuberculous disease of the lungs.

2. Short, stout, thick-boned persons are predisposed toward obesity and its attendant evils. If the thorax is wide and round, emphysema is liable to occur, although this shape of chest is more often a consequence than a cause.

(*b*) Certain abnormalities of shape result from the following diseases (*q. v.*): Rachitis, acromegaly, myxœdema, pulmonary osteoarthropathy, osteitis deformans, and osteomalacia.

The colour of the hair and eyes is of little importance, but it is of service in forming a complete conception of the case to note the "complexion." Light or blonde, dark or brunette, indeterminate, may be employed as qualifying terms in the white races. The colour of the hair and its amount (*q. v.*) as symptoms are considered elsewhere.

V. **Diatheses and Cachexias.**—There are some differences among lexicographers as to the meaning of the terms diathesis and cachexia. Following and defining the modern usage, it is to be understood that *diathesis* refers to a congenital habit of body, and *cachexia* to a condition of anæmia and debility, as follows:

1. **Diathesis.**—A diathesis is a congenital condition or habit of body which predisposes to certain constitutional or local manifestations of disease.

The recognition of a diathesis is, as a rule, dependent upon the presence of a certain disease, or a history of its past occurrence in the individual. After the history is taken and the examination completed, it is of service in conveying an idea of the patient and the general trend of his pathological life, to state the diathesis with its qualifying adjective. The qualifications are as follows:

(*a*) *Gouty, Arthritic, Uric-acid, or Lithæmic Diathesis.*—A disposition to gout and its sequent renal and cardio-vascular changes—aneurism, angina pectoris, and cerebral hemorrhage. The physical characteristics of the individual of this diathesis are said to be a robust, well-developed body, florid face, thick hair, and good teeth, hearty appetite, good digestion, and a strong heart with high-pressured arteries.

(*b*) *Tuberculous* (*Phthisical, Strumous, Scrofulous*) *Diathesis.*—A habit of body which is vulnerable or predisposed to tuberculous disease of glands, bones, or other forms of tuberculous infection. The older writers recognised two types which are not infrequently seen in classical perfection—viz., the tuberculous, with oval face, bright eyes, delicate skin and colouring, and long, slender bones; and the strumous, with a heavy, round face, thick, muddy skin, lumpy figure, and thick bones.

(*c*) *Catarrhal Diathesis.*—There are many persons who appear to be so liable to various chronic inflammations of mucous membranes that it is convenient to characterize them as belonging to the catarrhal diathesis.

(*d*) *Fatty Diathesis* (Bazin).—A term applied to those who are obliged to battle, oftentimes unsuccessfully, against a tendency to pathological obesity and fatty overgrowth.

(*e*) *Rheumatic Diathesis.*—A predisposition to various rheumatic affections—an indefensible but useful term.

(*f*) *Hemorrhagic Diathesis.*—The equivalent of hæmophilia (*q. v.*).

(*g*) *Neuropathic Diathesis.*—A predisposition, frequently hereditary, to diseases of the nervous system, most commonly the various neuroses. It is among this class that drug idiosyncrasies are most apt to be encountered. The diatheses *a*, *d*, *e*, *f*, and *g* can not be said to present physical traits which are in any degree characteristic.

(*h*) *Status Lymphaticus.*—Under diatheses may be classed the status lymphaticus (lymphatism), a condition not often encountered, occurring mainly in children and young persons. The lymph glands, especially the pharyngeal, thoracic, and abdominal, are universally enlarged; the lymphoid marrow of the bones is increased in amount and red marrow may replace the yellow marrow in young adults; the thymus gland and the spleen are enlarged; and there is deficient development (hypoplasia) of the heart and aorta. The body as a whole is undeveloped, and in shape retains many of its infantile characteristics. Rachitis frequently co-exists. Such individuals have a very small power of resistance, and sudden death may occur either without apparent cause or as a result of ailments or causes which are ordinarily attended by danger—e. g., unexpected death during convalescence from infectious diseases; sudden and inexplicable deaths in children; or while bathing; or during anæsthesia; or following the injection of diphtheria antitoxine.

2. **Cachexia.**—A yellow, waxy face, associated with anæmia, general debility, and more or less emaciation, are the characteristic signs of a condition which is spoken of as "cachetic," or a "cachexia." The anæmia and the yellow or brownish-yellow colour of the skin are the indispensable signs of this condition. It is usually associated with some grave organic disease or a chronic poisoning of the blood. While the meaning of the word is somewhat vague, yet to the physi-

cian of considerable experience, like some other terms, as "very ill," "sinking," "collapse," it expresses very graphically a state which would otherwise require many words of description, and the recognition of which is often of diagnostic value.

The various cachexiæ of a more or less well-defined character and importance are as follows:

(*a*) *Cancerous Cachexia.*—Debility, emaciation, anæmia, and a dirty yellowish, yellowish-brown, or brownish-green complexion.

(*b*) *Syphilitic Cachexia.*—Pronounced anæmia, with a muddy pallor, and perhaps a light yellowish tint of the skin and conjunctivæ.

(*c*) *Malarial or Paludal Cachexia.*—Puffy, pallid face, profound anæmia, and a greatly enlarged spleen (ague cake). There may be bronzing and discoloration of the skin and general œdema.

(*d*) *Cachexia Strumipriva* (Kocher).—A condition of anæmia and myxœdema resulting from the total extirpation of the thyroid gland, and attended by peculiar nervous phenomena.

SECTION IV

POSTURE IN BED—MODE OF MOVING—GAIT—STATION

Inspection may reveal certain facts in regard to the posture and the movements of the patient, which may be of little value or may furnish important suggestions as to the general condition of the patient or the nature of the disease.

I. **Posture in Bed.**—The patient usually takes to bed in acute illness or in chronic ailments, because of general weakness or some special interference with the use of the limbs, as in certain diseases of the nervous system. Remembering that many persons in health habitually assume certain attitudes while in bed, and that their customs in this respect may not be changed by illness, the postures assumed in disease and their diagnostic associations are:

(*a*) The *dorsal strong* or active posture, in which the patient lies upon the back comfortably and without constraint. It is seen in health, and in slight illness unattended with great pain.

(*b*) The *dorsal inert* or passive posture. The patient lies upon the back, but is constantly slipping toward the foot of the bed, thereby putting the body in a posture which is uncomfortable, and which interferes with the respiratory movements. It is observed in conditions of great weakness, most frequently in the acute infectious diseases. It is especially characteristic of typhoid fever, even at an early period, because of the marked muscular weakness and mental apathy so characteristic of this disease.

(*c*) A *rigid dorsal* position, with both legs drawn up, in order to diminish abdominal tension, is the rule in general peritonitis and in many cases of pelvic peritonitis. In appendical peritonitis the right leg alone may be flexed. In hip disease also there is flexion of one knee.

(*d*) The patient may lie *upon the side*, and the manner of lying may be active or passive, as in the dorsal position.

Patients having acute affections of the chest usually lie upon the affected side to limit the movements of the affected side and lessen the pain caused by pleural friction, as well as to afford greater freedom of compensatory motion to the healthy side. Moreover, if a large pleural effusion exists, the pressure due to its weight will not burden the heart and the healthy, uncompressed lung. The posture of a patient with a cavity in the lung is of some significance, that position being chosen which brings the opening of the cavity uppermost, thus allowing secretions to accumulate and be discharged at infrequent intervals instead of constantly trickling into the bronchial tubes and causing an incessant irritating cough. The rule of lateral decubitus is not invariable, as the patient may find other positions preferable. In sciatica the subject usually lies upon the unaffected side.

The lateral position with the legs drawn up to meet the trunk (the "coiled" posture) is seen in meningeal, cerebral, and cerebellar disease (due to spasm), hepatic, renal, and intestinal colic.

(*e*) *Opisthotonus.*—This is the name given to an uncommon dorsal position in which the body rests upon the head and heels, the trunk being arched upward. It is observed in strychnine poisoning, tetanus, and uræmia, as well as in some peculiar manifestations of hysteria and hystero-epilepsy. A modification of this position is observed in the characteristic contraction of the posterior neck muscles in meningitis, whereby the back of the head bores into the pillow.

(*f*) *Emprosthotonus.*—An attitude in which the upcurved body rests face downward upon the forehead and feet. It is the opposite of opisthotonus and is rarely seen, but may be observed in tetanus, strychnine poisoning, paralysis agitans, and cerebro-spinal meningitis.

The prone position, without tonic contraction of the muscles, is occasionally witnessed in the various forms of colic, the patient usually lying with the abdomen resting upon a pillow or bolster in order to secure firm abdominal pressure for mitigation of pain. This posture, without the pillow, is sometimes assumed because of the relief afforded in erosion of the vertebræ resulting from aneurism, or in tuberculous disease of the spine. Less frequently it may be seen in gastric ulcer and mediastinal disease.

(*g*) *Orthopnœa.*—In diseases attended with excessive dyspnœa the patient instinctively sits upright, with the hands or elbows resting upon some point of support, in order, by fixing the shoulders, to facilitate the action of the accessory muscles of respiration. If an attempt is made to assume the horizontal position, the feeling of dyspnœa becomes so intense that the upright attitude is quickly retaken. Orthopnœa attends spasmodic asthma, emphysema, and diseases of the heart in their later stages. It is common with large effusions into the pericardial and pleural cavities, and is often encountered in abdominal dropsies of sufficient size to press the diaphragm upward. If the diaphragmatic pleura is inflamed, the patient is apt to sit erect with the trunk leaning toward the affected side. Extreme dyspnœa of the inspiratory type, requiring the sitting posture, attends obstructive or paralytic disease of the larynx, as in laryngeal diphtheria and paralysis of the dilators of the glottis.

II. **Mode of Moving.**—(*a*) In certain ailments, some of which have been mentioned in the preceding paragraphs, there is unusual *immobility* and a striking look of helplessness, due to the increase of pain upon motion, as in rheumatism, scurvy, and rachitis, or a disinclination to move because dyspnœa is made worse by exertion. Paralysis or tonic spasm of large muscular groups is another cause of enforced quietude.

(*b*) An opposite condition, *restlessness*, exists in many diseases, as in fevers and large hemorrhages. Agitated and irregular movements are seen in chorea, in hysteria with its manifold manifestations, and in other diseases of the nervous system. Severe griping or colicky pain, gallstone, or renal colic will induce the patient to throw himself about in the hope of relieving his suffering.

III. **Station.**—One should observe the manner in which the patient stands; whether or not he is firm upon his feet; the shape and carriage of the head and shoulders; whether he is erect or bends forward (as in paralysis agitans) or backward (as in ascites or abdominal tumour); and the position and shape of the limbs. *Station*, the power of standing more or less steadily, is greatly disturbed in some forms of nervous disease. A healthy person standing with the feet close together and the eyes open will sway forward and back 1 inch and from side to side ¾ of an inch. In locomotor ataxia the swaying is extreme, owing to the loss of muscular, articular, and tendonous sense, and if the eyes are closed the patient may fall. Disease of the middle cerebellar lobe and aural vertigo (Ménière's disease) will also cause swaying, and in paroxysms of the latter malady the patient may be absolutely incapable of standing or walking. Loss of the power to stand steadily is called static ataxia (*q. v.*).

IV. **Gait.**—The manner of walking is closely associated with station, and when possible should be attentively observed, as it is of much importance in the diagnosis of certain conditions or diseases.

When observing the manner in which the patient walks, it is very desirable, if circumstances permit, to have the legs stripped and the patient in his bare feet. In women patients the nightdress may be pulled through from the back between the legs, snugged up, and pinned in front; or a T-bandage may be improvised out of a couple of towels.

First eliminate, by inquiry and examination of the abdomen, bones, and joints, the various causes of abnormal progression enumerated in (*a*) and (*b*) below. Then desire the patient to walk away from and back to the observer; to walk at right angles to the line of sight; to walk along a crack between the floor boards or a seam of the carpet. During this time he is to be attentively watched in order to discover any peculiarities of gait, the manner of putting down and raising the feet, reeling, unsteadiness, or deviation from the straight line.

The peculiarities in the manner of walking, and the conditions in which they possess more or less diagnostic importance, are as follows:

(*a*) In pregnancy, ascites, large abdominal tumours, cretinism, pseudo-hypertrophic paralysis, and obesity, the body leans backward and the feet are widely separated while walking.

(*b*) Painful or disabling affections of one or both lower extremities, such as corns, rheumatism, gout, sciatica, metatarsal neuralgia, hip- or knee-joint disease or injury (recent or old), sacro-iliac disease, sprains, inflammatory disease of extremity, short leg, and paralysis of one leg, give rise to a limping or hobbling gait. So also may abdominal aneurism, and subacute or chronic appendicitis. *Intermittent claudication* is a condition in which there are pains and paræsthesias in the feet and legs on walking. The gait becomes limping, and finally a rest must be taken, after which the progress is resumed until a similar pause becomes necessary. Variously there are sensations of tension and stiffness in the calves; heat and redness, or coldness and blueness of the feet. The principal objective sign is the presence of sclerotic changes, with a small or absent pulsation in the arteries of the foot. The majority of cases are in men over forty years of age. In some instances the condition is due to angio-spasm, without sclerosis of the vessels.

(*c*) The most characteristic methods of progression are seen in diseases of the nervous system:

(1) *Ataxic Gait.*—In walking, the foot is raised suddenly and too high, the leg is thrown forward with unnecessary vehemence, and the foot is again brought to the ground heel first, or flat-footed with a stamp. The feet are usually planted wide apart, and while they are

in the air they move as if the patient was doubtful where to put them. The body is bent forward, and the eyes are fixed upon the ground in order to supplement as far as possible the loss of muscular and articular sensation. This gait is extremely characteristic of locomotor ataxia.

(2) *Cerebellar Ataxic Gait.*—The manner of progression resembles that of an intoxicated person. The patient walks with short steps, and with his feet wide apart, staggers, reels, sways to and fro, and reaches a set point by zigzagging toward it. The swaying is relieved if he is supported by the hands of the observer placed under the armpits. This gait is significant of a tumour of the vermis or middle lobe of the cerebellum, and is often called the titubating gait, or simply cerebellar ataxia (*q. v.*). A somewhat similar gait is seen in Friedreich's disease, hereditary cerebellar ataxia, dementia paralytica, ataxic paraplegia, labyrinthine disease, and, to some extent, in vertigo from any cause.

(3) *Steppage Gait.*—This variety of gait is due to paralysis of the extensor muscles of the foot, whereby, when the foot is lifted, its anterior part tends to hang or drop down. In order to prevent the toes catching and tripping against the ground, the leg carries the foot somewhat forcibly forward, raising it at the same time unusually high, thus throwing the toes upward and bringing the foot to the ground heel first. It resembles the gait of a man who is walking through thick grass or brushwood, and has been described as "prancing" or "high-stepping." It is evidence of peripheral neuritis (diabetic, arsenical, alcoholic, etc.) of the anterior tibial nerve, and, because of a certain resemblance to that of locomotor ataxia, is sometimes termed the pseudo-tabetic gait.

(4) *Spastic Gait.*—The legs are rigid, move stiffly, and there is apparent difficulty in bending the knees. In consequence the foot is dragged along, the toes catching and scraping on the ground. In some instances, owing to spasm of the adductors of the thigh, the legs and knees touch and cannot be separated, causing cross-legged progression—i. e., the legs and feet overlap at each step. This gait depends upon the excessive tension and spasticity of the muscles arising from lesions of the upper motor neurones. It is therefore, when bilateral, significant of sclerosis affecting the lateral pyramidal columns of the cord. The mode of walking in hemiplegia (*q. v.*) is a unilateral form of the same gait. The paralyzed leg, by a tilting of the pelvis, is swung outward and around to the front ("mowing" gait), the toes often scraping the ground.

(5) *Festination.*—The head and body are bent forward and the patient takes short, shuffling, hurried steps, his speed tending to

increase as he progresses, exactly as if he was being constantly pushed forward and was trying to prevent it. This gait is termed festination or propulsion. In some instances, if the patient is pulled rather suddenly backward, he will take a number of backward steps (retropulsion), although the body remains in its forward-leaning attitude. This gait is characteristic of paralysis agitans.

(6) *Waddling Gait.*—The shoulders are thrown back, the back is hollowed (lordosis), and the abdomen protuberant, the body sometimes actually leaning backward. In walking, the feet are planted wide apart and the body swings from side to side at each step—the "waddling" or "goose" gait. It resembles somewhat the gait described under (*a*), and is a very characteristic symptom of pseudo-hypertrophic muscular paralysis. It is seen also in disease, or congenital dislocation of both hip-joints.

(7) In *Thomsen's disease*, on attempting to rise or walk the leg muscles immediately become stiff and cramped, without pain. The cramp relaxes in a few seconds, but returns when the muscles are again called into use. In *saltatory spasm* (very rare), when the weight of the body is put upon the feet, strong and rapid contractions of the leg and thigh muscles take place, causing the patient to jump violently.

In hemiplegia, *one foot* is dragged. Dragging of *both feet* is seen in multiple neuritis, hereditary peroneal atrophy, spasmodic spinal paralysis, and in spinal and syphilitic spinal paralyses. The gait is *tottering* in those who have taken large doses of the bromides for long periods; so also in hydrocephalus, psychosis polyneuritica (Korsakoff's disease), paralysis agitans, and idiopathic muscular atrophy.

SECTION V

PAIN; TENDERNESS; PARÆSTHESIAS

There are certain subjective sensations which are of much, although varying, importance in diagnosis. Although a subjective symptom does not constitute decisive evidence, it may furnish an important clew to the nature of the disease—e. g., the "tender point" in appendicitis. On the other hand, there may be an absolute absence of tenderness over the kidney in pyelitis and an occasional lack of pain in peritonitis, gastric ulcer, perforating duodenal ulcer, latent pleurisy, and various pelvic lesions, by which a most important guide symptom is missing.

The subjective symptoms which are considered in this section comprise pain, tenderness, and paræsthesias. Other disorders of sensation, such as anæsthesia, hyperæsthesia, etc., are dealt with elsewhere (Examination of the Nervous System). It is, of course, impossible within reasonable limits to describe all the multiple forms and sites of pain and other subjective sensations. Only those are considered which may prove helpful in diagnosis.

I. PAIN

1. **Differences in Susceptibility.**—As pain is a purely subjective symptom, its intensity must be estimated by the statements of the sufferer, by the manifestations of its presence, and by the nature of any lesion which may be discovered as its probable cause. Much depends upon the skill and experience of the observer in judging individual susceptibility. The variations in pain sensibility are very great, and are racial as well as individual. The Semitic stock, and the Celtic and Italic groups, appear to possess an average greater sensibility to pain than the Teutonic and Slavonic groups. The most important variations, however, are personal or individual. The congenitally neurotic patient will complain bitterly of pain from a cause which in one of dull sensibilities will give rise to simple discomfort. It is to be remembered that the pain suffered by the abnormally sensitive person has as real an existence in consciousness as the slight discomfort felt, from the same cause, by those of less acute pain perception.

2. **Modifications of Susceptibility.**—The manner of life and occupation may modify the susceptibility to pain. The habitual endurance of hardship blunts the pain sense, and, conversely, the person guarded from rude mental or physical contact, will be more acutely sensitive to pain. A strong mental prepossession (religion, excitement of battle, etc.) may interfere with the registration of painful impressions upon the consciousness. The sensibility to pain is apt to be increased by its long continuance, and it is a common observation that each recurrence of pain, during the course of a disease, finds the patient less able to bear it. Fright or expectant apprehension invariably increases pain, and sometimes originates it.

3. **Manner of Statement.**—There are also differences in the manner of statement. Some patients as a matter of pride practise understatement of their subjective sensations, while others from various motives habitually magnify their sufferings, and in most instances without

the slightest intention of deceiving the physician. It arises largely from the unconscious egotism of illness and a desire to obtain relief by impressing the medical attendant with its pressing necessity. In estimating the severity of pain, the facial expression and bodily manifestations of pain are of much value. A statement made with a cheerful countenance, that the speaker is at the present moment suffering "horrible agony," does not square with the facts, and this combination is of diagnostic value as indicative of self-deception, hysteria, or a habit of chronic emphasis. Women, perhaps more than men, are inclined to exaggerate in recounting their symptoms. The reason may be found in the greater susceptibility of the feminine nervous system, and the larger measure of sympathy which a woman habitually receives. This is said without prejudice to the courage, endurance, and self-sacrifice which are so brilliantly exhibited in many sick-rooms.

In the majority of cases in which really severe pain is present the respiration is rapid, the pupils are dilated, the skin is wet with perspiration, the pulse is apt to be tense, there is a feeling of faintness, and not infrequently a large amount of limpid urine is passed within a brief period—symptoms some of which can not be simulated.

In all cases in which pain is a symptom a careful investigation should be made in order to discover any existing objective condition which may constitute corroborative evidence of the truth of the patient's statement. In view of the fact that sad mistakes have occurred, it is best not to err on the side of scepticism, but to credit subjective testimony until some anatomical incongruity of distribution is found or some sudden shifting of the seat of pain occurs which is incompatible with the ascertained objective symptoms and signs.

4. **Varieties of Pain.**—Pain varies in intensity from sharp or acute to dull or aching pain. It may be radiating, darting from its point of origin along the branches of a nerve trunk; or paroxysmal, remitting, coming and going; or shifting, moving from one locality to another; or possess the character indicated by the terms gnawing or colicky. It may be increased by motion or relieved by pressure. Two or more varieties may co-exist as a single pain symptom.

5. **Diagnostic Import of the Character of Pain.**—(*a*) *Acute pain* is characteristic of acute inflammations of serous and synovial membranes, as in pleurisy or joint inflammations. Acute radiating pain marks the idiopathic neuralgias or the nerve pain due to inflammation or pressure, as in neuritis or thoracic aneurism.

(*b*) *Dull pain*, like that of a bruise, usually attends inflammations of mucous membranes and the parenchymatous viscera (which are poorly endowed with sensory nerves), and many chronic inflammations.

(*c*) *Paroxysmal* or *remitting pain* is characteristic of the neuralgias and colics, and the major portion of the radiating pains just mentioned are paroxysmal in type.

(*d*) *Shifting pain*, more or less sharp, occurs in connection with rheumatism, hysteria, locomotor ataxia, and trichinosis.

(*e*) Pain of a *gnawing* or *boring* character is encountered in disease of the spinal column, thoracic and abdominal aneurism, periosteal or osteal inflammations, gastric carcinoma, and sometimes in gouty lesions and lithæmic states.

(*f*) *Cramp* is a sudden and painful spasm of certain muscles or muscle groups. Aside from the cramp due to overuse of special muscles (writer's cramp, occupation neuroses), and the cramp affecting the muscles of the calf and the toes, the term is frequently applied to painful abdominal spasms (colic) due to excessive action of the muscular walls of the stomach and intestines. Abdominal cramp or colicky pain is a frequent accompaniment of flatulence and gastro-intestinal disease in general; it occurs in cases of intestinal obstruction from any cause, and as a result of irritant poisoning.

(*g*) Other qualifying terms which are employed to indicate the character of pain are: burning, as in herpes zoster; aching, as in a moderate lumbago or other myalgia; throbbing or pulsating with the heart beat, as in a circumscribed phlegmon or suppurative inflammation. *Tenesmus*, or tenesmic pain, is that which attends urination or defecation from an inflamed bladder or rectum, or the expulsion of membrane or clots from the uterus, accompanied with a sensation of straining or bearing down. The adjectives stabbing, darting, and lancinating are equivalent to sudden, sharp, and acute radiating pain.

(*h*) Pain, *increased by motion*, is found in all inflammatory diseases, myalgias, serous inflammations, the various forms of articular rheumatism, and disease of the joints and vertebræ. Some forms of colic and hysterical pain may be relieved by firm and even pressure.

(*i*) The *acuteness* or *chronicity* of pain corresponds largely to the suddenness of occurrence and the persistence of its cause. Pain may persist after the removal of its factors, apparently because the pain habit has been formed by the affected nerves and their associated centres; but, as a rule, pain extending over a long period of time indicates a continuance of the pathological conditions from which it originated. Pain may be recurrent or periodic, days or weeks elapsing between successive attacks, as with migraine; or may be continuous, with occasional exacerbations, as in headache from eye strain.

6. **Diagnostic Import of the Seat of Pain.**—As a general rule, the seat of pain corresponds to the location of the causative lesion. In certain cases the pain is reflex or, more properly, transferred, being assigned to the furthest peripheral termination of a nerve, when the causative lesion is situated at one of its terminations much nearer the origin of the nerve, or an irritation at the termination of one branch is felt also at the termination of a branch situated in a different locality, or the irritation may be at the origin of the nerve trunk and the pain referred to its entire peripheral distribution. If the pain is extremely intense, it may be felt not only in the direct nerve supply of the affected area but also in areas indirectly connected, a phenomenon assumed to be caused by irradiation, or an overflow of the painful impression from its accustomed channels.

Even though the pain caused is not intense, a source of irritation may exist at one point and be felt at another widely separated from the actual seat of the lesion. In all such cases the sensation is in reality a transferred or referred pain, and strictly speaking can not be termed reflex. The latter word implies the travelling of an afferent (sensory) impression to a centre, which centre, in consequence of the received impulse, sends out an efferent (motor) impulse; whereas a transferred sensation is one perceived by the sensorium, not as belonging to its real source of origin, but which, because of the existence of indirect sensory connections along which the impression travels, is referred to an entirely different portion of the periphery. The pain sometimes felt in the mammary gland when disease of the uterus exists is an illustration of this fact. The well-known diagrams of Dana represent graphically the more important varieties of transferred pains (Figs. 5 and 6). Not infrequently there may be pains felt in the periphery which are due to central disease of the brain or cord, as in meningitis.

Diseases of the different viscera may manifest themselves by pain and disturbed sensation referred to certain cutaneous areas. The elaborate researches of Head make it probable that when a painful stimulus is applied to a tissue or an organ which normally possesses a low degree of sensibility, and which is centrally in close connection with a tissue or organ possessing a much higher degree of sensibility, the pain produced is felt in the part which is relatively more sensitive. Consequently the tenderness of the skin in visceral disease is due to the passing of sensory impulses from a diseased organ to its corresponding spinal segment, these impulses causing a disturbance in the segment of such a nature that any additional impulses coming from the skin surface with which this is connected will be increased and modified so as to produce abnormal or painful impressions, which are referred in consciousness to the part of higher sensitiveness, the skin.

These painful cutaneous areas are best demonstrated by using a roundaded pin of sufficient size to feel distinctly blunt to the normal skin of the server. The head of the pin is then pressed with moderate force upon the

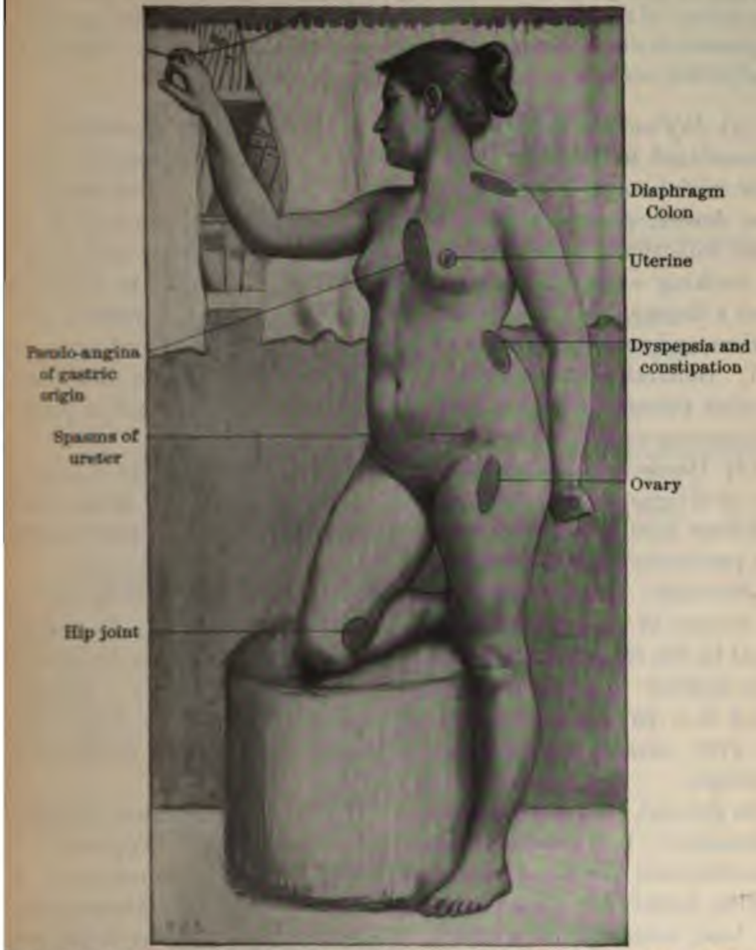


Fig. 5.—The location of transferred pains (Dana). Figure redrawn in charcoal after an imported photograph.* These pains, through the existence of more or less indirect or roundabout sensory nerve connections, are felt at points of varying remoteness from the source of the irritation which produces them. Thus, pain in the knee may be due to disease of the hip joint, the lesion affecting one of the terminations of the nerve much nearer to the origin of the latter than that in which the pain is felt; or the mammary gland may swell and become painful when a distant organ—the uterus—is the seat of a pain-producing process. See also Fig. 6.

urface to be examined, using it as if testing with the point for anæsthesia. If tender area is present, the patient will at once complain of soreness resembling that of a bruise when touched; and if the point of the pin is employed, it causes pain greatly exceeding that which would be felt in a normal skin. A rough test may be made without using the pin by gently pinching up a fold of skin. The absence of tenderness does not negative the existence of visceral disease; its presence is simply confirmatory, or in some cases suggestive. The clinical value of this test is at present very slight.

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* With few exceptions, the photographs used for diagrammatic purposes in is book have been imported.

(*a*) *Diffuse Pain.*—Pain or aching, general in its distribution, is encountered in the majority of febrile diseases, especially during their initial stage. Although present to a greater or less degree in most fevers, general aching is apt to be most pronounced in the acute infections, of which epidemic influenza, variola, and dengue are striking examples. Lacunar tonsilitis exhibits it to almost as great a degree. It may be associated with syphilis, lithæmia, rheumatism, and some of the intoxications, as in poisoning by mercury and lead. General aching results, as a rule, from the action of a toxine or other poison in the circulating blood upon the central or peripheral nervous system.

(*b*) *Headache and Pain in the Head.*—Pain in the head is a symptom of diverse meaning and origin. *Headache* is defined as an attack of diffuse pain affecting different parts of the head, and not confined to a particular nerve.

Neuralgia (toxic, referred, pressure) is characterized by pain in the course of a nerve or nerves, generally unilateral. It is functional in the sense that no disease of the nerve itself may be present. Pain limited to a nerve tract may be due to neuritis. *Migraine* (*q. v.*) is a painful, periodical neurosis, involving the trigeminus, but with certain symptoms which distinguish it from headache or neuralgia.

In general, the *causes* of pain in the head are as follows, excluding traumatism: 1. Anæmia and sudden hemorrhages. Nephritis. 2. Constitutional diseases; diabetes, gout, lithæmia, rheumatism. 3. Specific infectious diseases, mainly fevers. 4. Intoxications; alcohol, lead, mercury, tobacco. 5. Neuroses; epilepsy, hysteria, neurasthenia, exophthalmic goitre. 6. Inflammations or organic diseases of, or affecting, the nervous system; embracing arteriosclerosis, diseases of cranial bones, meningitis, encephalitis, neuritis, syphilis, and tumour or abscess. 7. Reflex or referred pain from disease of the ear, eye, nasopharynx, stomach, and sexual organs. 8. Fatigue, bodily or mental; impure air, acclimation.

The *character* of head pain varies with the individual and the cause. We may distinguish: 1. Sharp, lancinating, paroxysmal pain; characteristic of neuralgia. 2. Pulsating or throbbing headache; if unilateral and in connection with other vasomotor disturbances, indicative of migraine or hemicrania. 3. Dull, heavy, diffused headache; found in gastro-intestinal and febrile diseases of

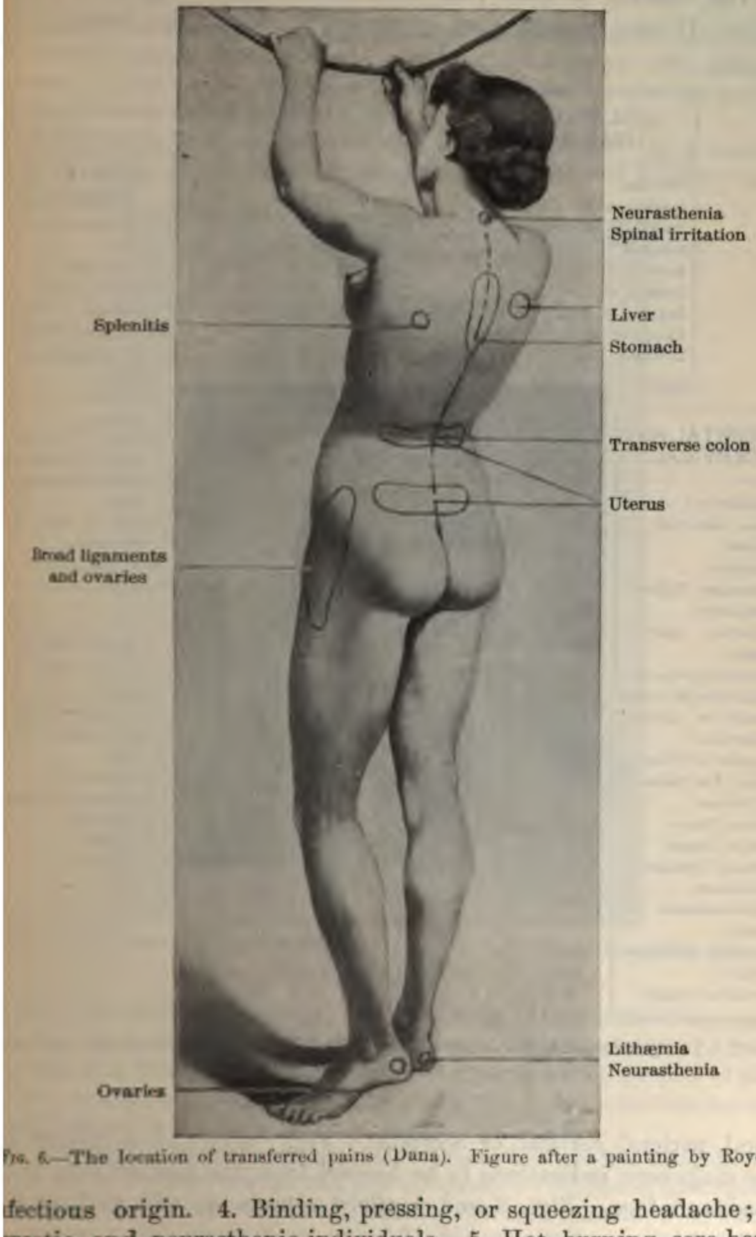

Fig. 6.—The location of transferred pains (Dana). Figure after a painting by Royer.

fectious origin. 4. Binding, pressing, or squeezing headache; in eurotic and neurasthenic individuals. 5. Hot, burning, sore headache; associated with rheumatism and anæmia. 6. Sharp and boring head pains; encountered in epilepsy and hysteria.

The location of head pain is of considerable diagnostic importance. It may be diffuse or in varying combinations, frontal, temporal, parietal, vertical, or occipital. Fig. 7 shows in a general way the diagnostic indications to be derived from the seat of pain in the head and face. Figs. 8 and 9 afford a more specific and detailed representation. See also Fig. 10.

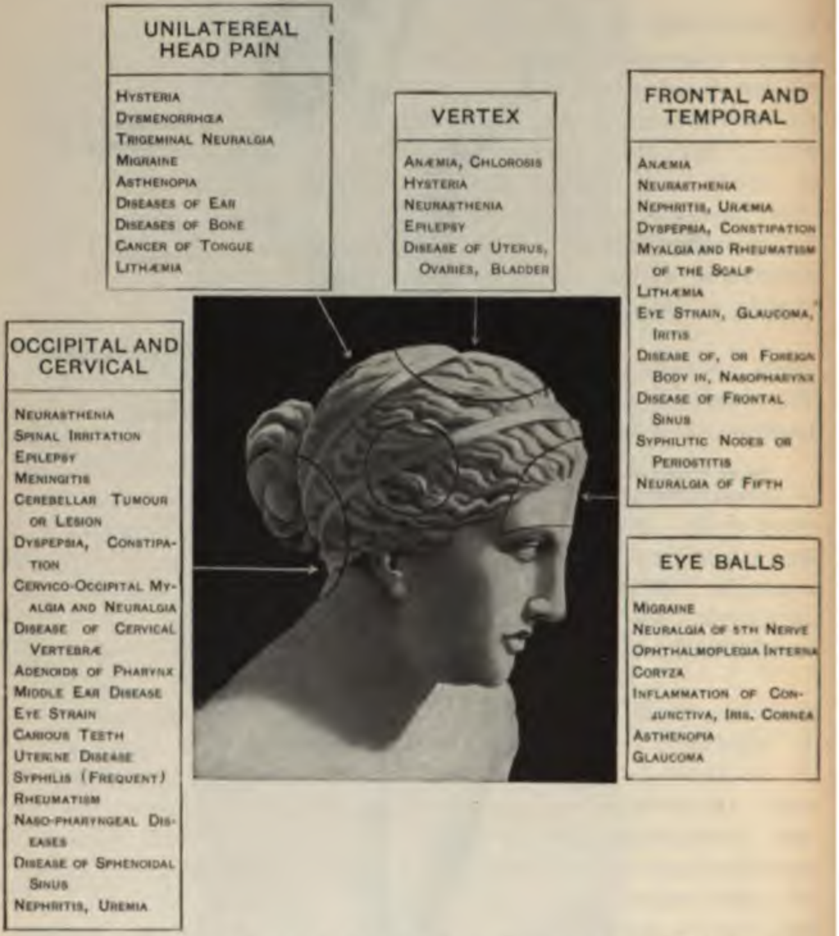

Fig. 7.—The general diagnostic indications to be derived from the seat of pain in the head and face.

The more important *varieties* of headache which possess somewhat distinctive characteristics are as follows:

1. *Anæmic headache* is a sore and pressing pain, usually felt in the forehead and orbital region or in the vertex, and is often associated with occipital pressure. As its name indicates, it is found in connection with the general and special forms of impoverished blood.

2. The headache of *nephritis*, excepting the sudden attacks due to uræmia, is in most cases caused by the arteriosclerosis which so often forms an essential part of the chronic nephritides. The pain is apt to be of a throbbing character, somewhat shifting, often accompanied by vertigo and tinnitus. I have more than once been struck by the presence of a vague fear on the part of the patient as to the final result—forebodings which have in two cases been unconsciously realized by the supervention of an apoplectic attack.

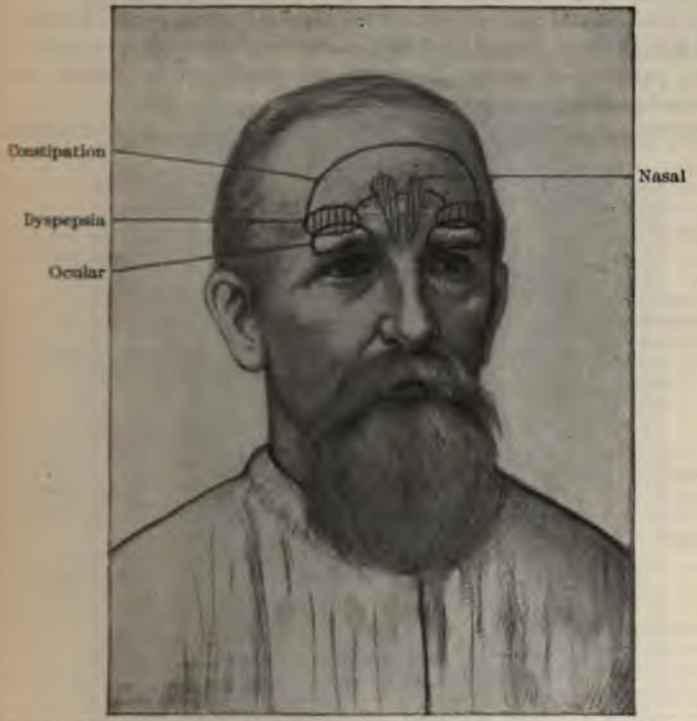

Fig. 8.—The causes of localized headache, according to the exact site of the pain. Redrawn after Collins.

3. The typical headache of *hysteria* is a pain as if a nail was being driven into the top of the head (clavus), but is of comparatively infrequent occurrence.

4. The headache of *neurasthenia*, probably the most frequent of all headaches which require treatment, is of a pressive character, usually vertical, but sometimes described as a band around the head. It is very characteristic in

being almost invariably worse in the morning, becoming lighter or disappearing toward the latter part of the day.

5. Headache from *turbinal pressure*, either acute from rapid swelling or chronic from hypertrophy or septal deviations, or perhaps from distention of the accessory sinuses of the nose, is felt as a pain beginning at the root of the nose and running directly backward to the occiput. It is greatly increased in severity by coughing or bending over. This variety of headache is not at all uncommon.

6. *Ocular headaches* are either frontal or occipital; the pain comes on in the majority of cases after use of the eyes in close work, such as sewing or reading, and the nightly rest of the eyes renders the patient free from pain on arising.

7. Headache from *constipation* and *disorders of digestion* is usually of a throbbing, pulsating character, affects the frontal and orbital regions, and is made worse by sudden movements of the head.

8. The headache of *uterine disease* is usually occipital, sharp, and radiating.

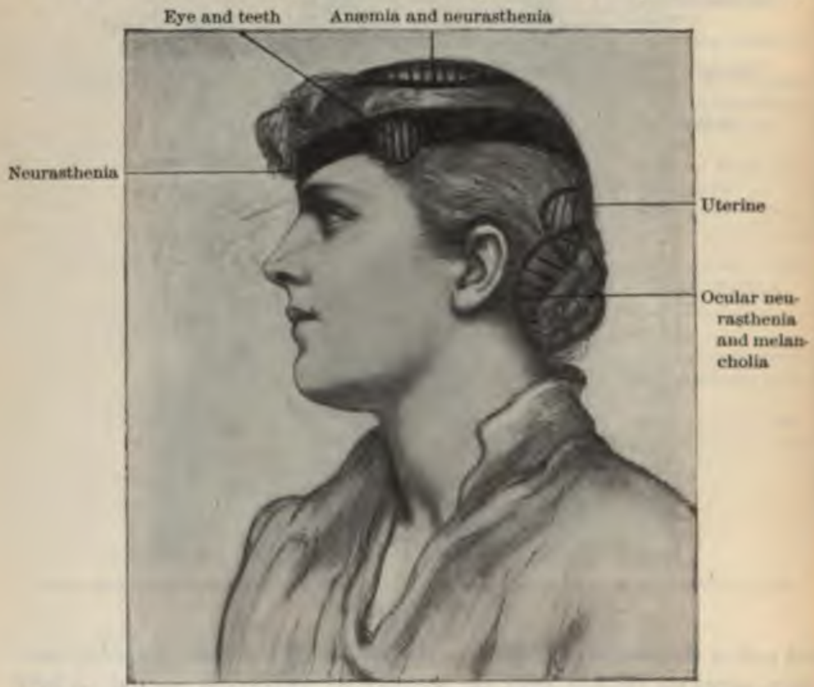

Fig. 9.—The causes of localized headache, according to the exact site of the pain. Redrawn after Collins.

As headache or head pain is merely a symptom, a careful search should be made to find the *cause*. It is of especial importance to examine with reference to rheumatism of the scalp, periostitis or caries of the cranial bones, the existence of nasal disease (particularly inflammation of the frontal or ethmoidal sinuses), ocular defects, gastro-intestinal disorders, anæmia, disease of the blood-vessels (arteriosclerosis), nephritis, syphilis, neuralgia, migraine, neurasthenia, hysteria, epilepsy, and finally organic disease of the nervous system (abscess, tumour, meningitis, encephalitis). Chronic headaches are usually due to neurasthenia, less frequently to ocular defects, anæmia, syphilis, or pachymeningitis, and, rarely, the cause remains conjectural.

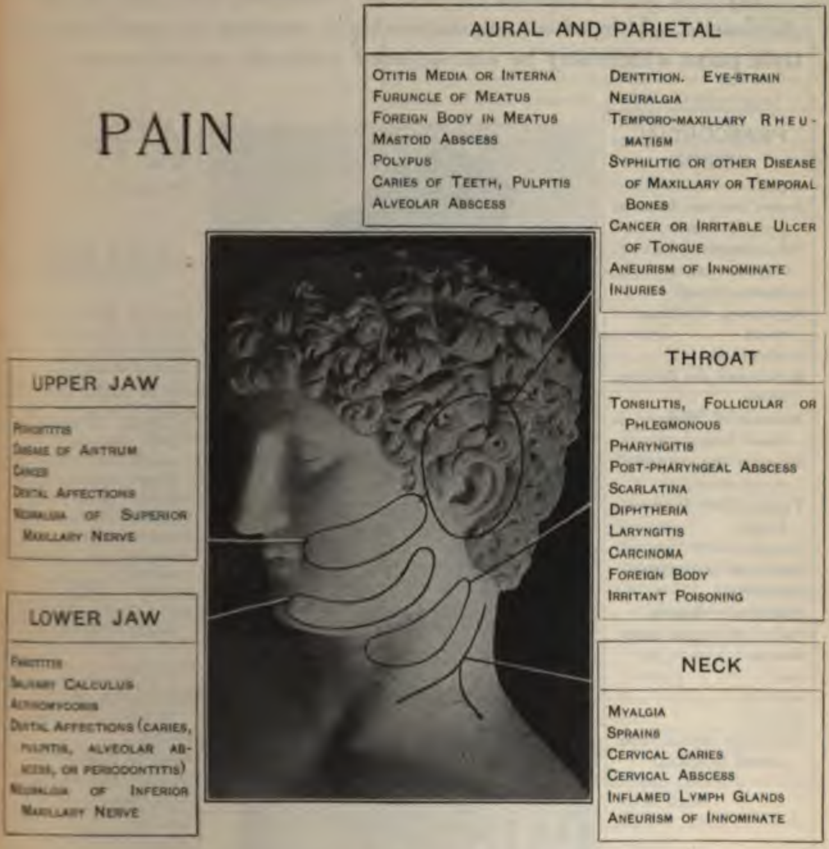

Fig. 10.—The general diagnostic indications to be derived from the seat of pain in the face and neck.

(c) *Pain in Chest, Abdomen, and Extremities.*—The diagnostic indications derived from the seat of pains in the thorax, abdomen, back, and extremities are shown in Figs. 10 to 21. As with headache, the cause of a given pain is to be discovered by a study of the

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associated signs and symptoms, determining by means of the latter the presence or absence of the diseases or conditions which are assigned as capable of producing the pain of which complaint is made.

(*d*) *The More Common Diseases or Conditions with which Pain is Associated.*—It is, of course, impossible to mention in detail the multiple pains which may be encountered clinically as evidences of disease of various organs or systems. Those here dealt with are of more or less importance from a diagnostic point of view. There are many examples of pain which the clinician can not rationally or to his own satisfaction explain.

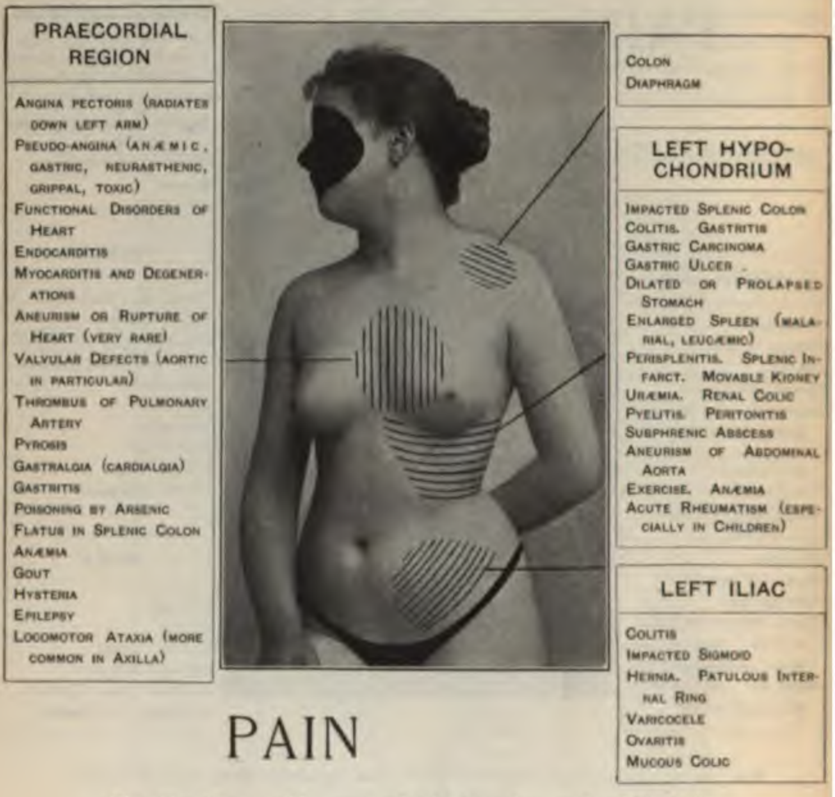
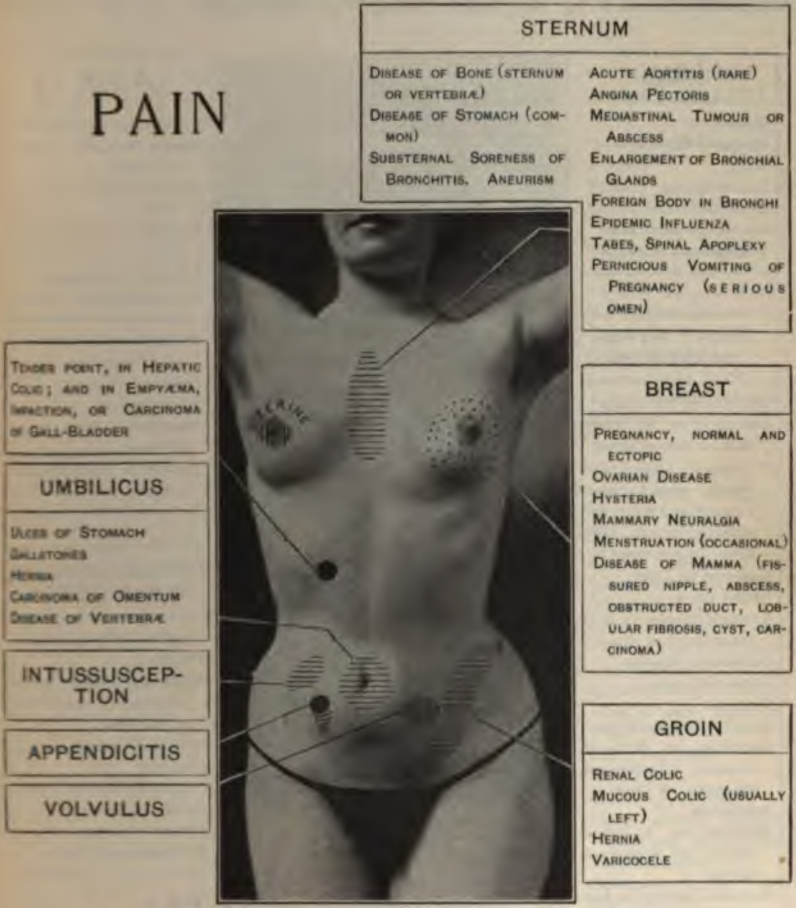

PRAECORDIAL REGION
Angina pectoris (radiates down left arm)
Pseudo-angina (anæmic, gastric, neurasthenic, grippal, toxic)
Functional Disorders of Heart
Endocarditis
Myocarditis and Degenerations
Aneurism or Rupture of Heart (very rare)
Valvular Defects (aortic in particular)
Thrombus of Pulmonary Artery
Pyrosis
Gastralgia (cardialgia)
Gastritis
Poisoning by Arsenic
Flatus in Splenic Colon
Anæmia
Gout
Hysteria
Epilepsy
Locomotor Ataxia (more common in Axilla)
Colon
Diaphragm
LEFT HYPOCHONDRIUM
Impacted Splenic Colon
Colitis. Gastritis
Gastric Carcinoma
Gastric Ulcer
Dilated or Prolapsed Stomach
Enlarged Spleen (malarial, leucæmic)
Perisplenitis. Splenic Infarct. Movable Kidney
Uræmia. Renal Colic
Pyelitis. Peritonitis
Subphrenic Abscess
Aneurism of Abdominal Aorta
Exercise. Anæmia
Acute Rheumatism (especially in Children)
LEFT ILIAC
Colitis
Impacted Sigmoid
Hernia. Patulous Internal Ring
Varicocele
Ovaritis
Mucous Colic
PAIN

FIG. 11.—The possible causes of pain in various parts of the body.

Nervous System.—There is the sharp, radiating, generally unilateral pain of the various neuralgias (*q. v.*). There is, furthermore, the pain of neuritis, multiple or localized; of neurasthenia or hysteria in the back of the neck, along the spine, and sometimes in the heel; of locomotor ataxia, most atrocious and agonizing; and the rare sharp, solitary pain below the knee which has been known as a premonition of cerebral hemorrhage.

Heart and Vessels.—The cardiac pain which is important beyond all others is that of true angina pectoris, closely simulated by the pseudo-angina of anæmic, gastric, hysterical, or toxic origin. Valvular disease, especially of the aortic cusps, may cause pain in the right hypochondrium and right shoulder, and there may or may not be some sternal pain in pericarditis. Thoracic aneurism may give rise to pain beneath the sternum or between the shoulders, and in rare cases acute aortitis is responsible for breast pain. Abdominal aneurism may produce general abdominal pain, sometimes acute, as well as pain in the lumbar back and between the lower angles of the scapulæ. Sharp abdominal pain may be due to embolism of the superior mesenteric artery.



PAIN

STERNUM

Disease of Bone (sternum or vertebræ)
Disease of Stomach (common)
Substernal Soreness of Bronchitis. Aneurism
Acute Aortitis (rare)
Angina Pectoris
Mediastinal Tumour or Abscess
Enlargement of Bronchial Glands
Foreign Body in Bronchi
Epidemic Influenza
Tabes, Spinal Apoplexy
Pernicious Vomiting of Pregnancy (serious omen)

Tender point, in Hepatic Colic; and in Empyæma, Impaction, or Carcinoma of Gall-Bladder

UMBILICUS

Ulcer of Stomach
Gallstones
Hernia
Carcinoma of Omentum
Disease of Vertebræ

INTUSSUSCEPTION

APPENDICITIS

VOLVULUS

BREAST

Pregnancy, normal and ectopic
Ovarian Disease
Hysteria
Mammary Neuralgia
Menstruation (occasional)
Disease of Mamma (fissured nipple, abscess, obstructed duct, lobular fibrosis, cyst, carcinoma)

GROIN

Renal Colic
Mucous Colic (usually left)
Hernia
Varicocele

FIG. 12.—The possible causes of pain in various parts of the body.

Lungs and Pleura.—Practically the pain in diseases of the lung is due to involvement of the pleura, the lungs themselves possessing a very low grade of pain sensibility. Leaving out of consideration the substernal soreness of bronchitis and the ordinary pleuritic pain in the lateral portions of the chest one may note the epigastric pain of diaphragmatic pleurisy (sometimes felt also in the left shoulder and above the clavicle), and the upper. abdominal pain so often complained of by the child suffering from pneumonia.

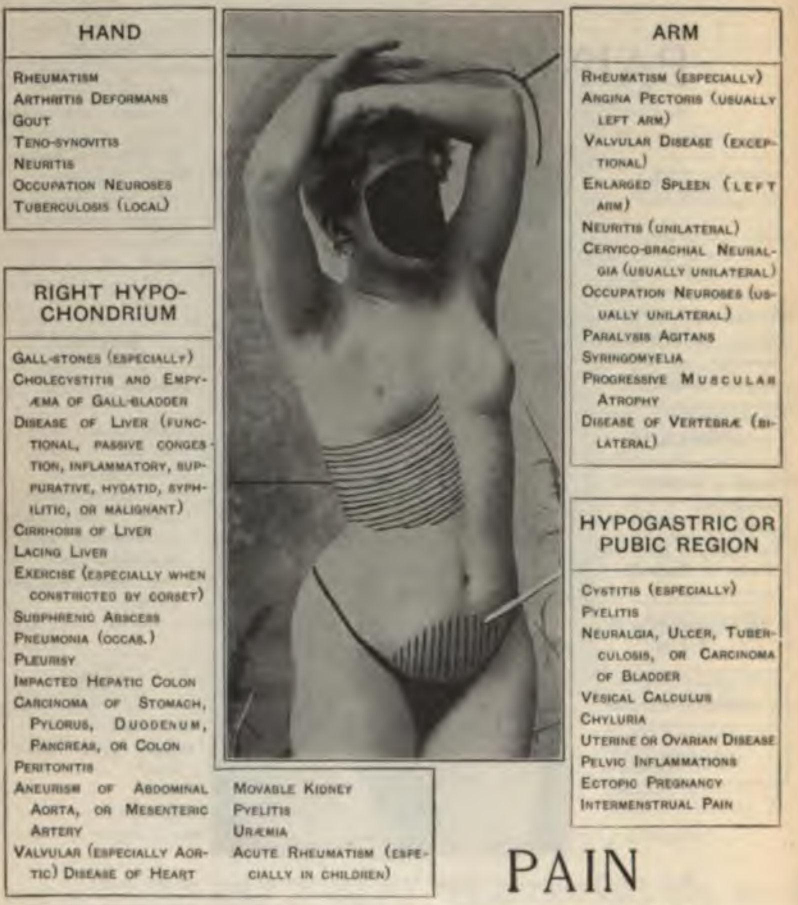
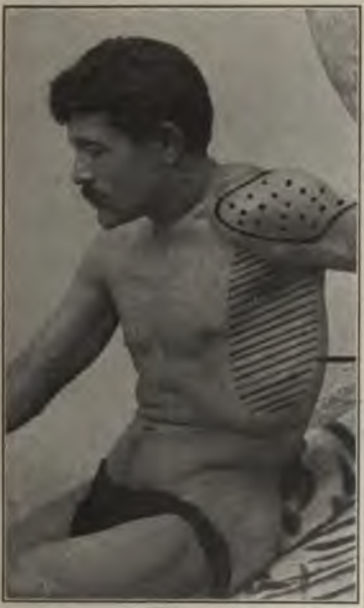

HAND
Rheumatism
Arthritis Deformans
Gout
Teno-synovitis
Neuritis
Occupation Neuroses
Tuberculosis (local)
RIGHT HYPOCHONDRIUM
Gall-stones (especially)
Cholecystitis and Empyæma of Gall-bladder
Disease of Liver (functional, passive congestion, inflammatory, suppurative, hydatid, syphilitic, or malignant)
Cirrhosis of Liver
Lacing Liver
Exercise (especially when constricted by corset)
Subphrenic Abscess
Pneumonia (occas.)
Pleurisy
Impacted Hepatic Colon
Carcinoma of Stomach, Pylorus, Duodenum, Pancreas, or Colon
Peritonitis
Aneurism of Abdominal Aorta, or Mesenteric Artery
Valvular (especially Aortic) Disease of Heart
Movable Kidney
Pyelitis
Uræmia
Acute Rheumatism (especially in children)
ARM
Rheumatism (especially)
Angina Pectoris (usually left arm)
Valvular Disease (exceptional)
Enlarged Spleen (left arm)
Neuritis (unilateral)
Cervico-brachial Neuralgia (usually unilateral)
Occupation Neuroses (usually unilateral)
Paralysis Agitans
Syringomyelia
Progressive Muscular Atrophy
Disease of Vertebræ (bilateral)
HYPOGASTRIC OR PUBIC REGION
Cystitis (especially)
Pyelitis
Neuralgia, Ulcer, Tuberculosis, or Carcinoma of Bladder
Vesical Calculus
Chyluria
Uterine or Ovarian Disease
Pelvic Inflammations
Ectopic Pregnancy
Intermenstrual Pain
PAIN

Fig. 13.—The possible causes of pain in various parts of the body.

Stomach.—The painful diseases of the stomach are ulcer and erosions in particular, less frequently cancer; and with varying incidence gastritis, gastroptosis, gastrectasia, and various neuroses. The pain of gastralgia (cardialgia) is excessive. The pain of a diseased stomach is felt in most cases primarily in the epigastrium, very frequently behind the lower sternum and between the shoulders; often in the right or left hypochondrium or posterior lumbar region; and gastralgic attacks may involve almost the entire abdomen.

Intestines.—Impaction of the colon, according to its seat, may cause pain in the right or left hypochondrium, or in the anterior, lateral, and posterior aspects of the thigh; mucous colic and inguinal hernia, in the groin; obturator hernia, in the knee; cancer or ulcer of the rectum, and hemorrhoids, in the sacral and coccygeal regions. The localized pain and tenderness of appendicitis is well known. Flatus in the splenic flexure of the colon may cause præcordial pain. A duodenal ulcer may or may not be painful until perforation occurs, and even then pain may be absent. Flatulence, poisoning by lead, mercury, and arsenic, as well as acute enteritis and intestinal obstruction or perforation, may initiate more or less general and severe abdominal pain.

PAIN



SHOULDER, RIGHT OR LEFT

- Rheumatism (especially)
- Synovitis
- Neuritis
- Cervico-brachial Neuralgia
- Diaphragmatic Pleurisy
- New Growth of Adrenals (top of Shoulder Blade)
- Dilated Stomach
- Duodenitis
- Colitis
- Loaded Colon (left)
- Acute Pleurisy (exceptional)

LATERAL WALL OF CHEST

- Pleurisy (especially)
- Pleural New-growths
- Pleurodynia
- Intercostal Neuralgia (usually left side, sixth to ninth rib)
- Disease or Injury of Chest Walls (ribs, soft tissues)
- Acute Pneumonia
- Pericarditis
- Aneurism (thoracic)
- Herpes Zoster (usually right side, pain may precede eruption)
- Overloaded Colon
- Flatulence

- Phthisis (accompanying Pleurisy)
- Disease of Vertebræ
- Mediastinal Tumour
- Enlarged Bronchial Glands
- Anæmia, Hysteria

Fig. 14.—The possible causes of pain in various parts of the body.

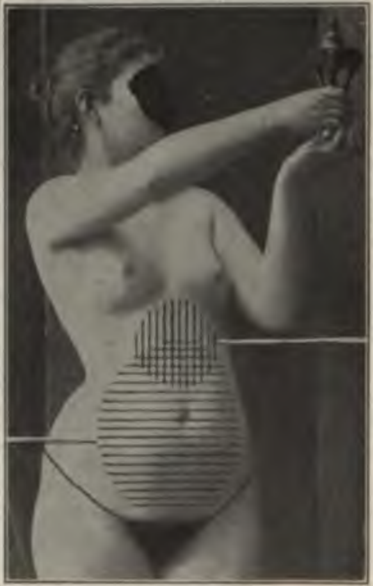
Liver and Gall Bladder.—The pain-producing diseases of the liver are the functional disorders and cirrhosis, which may be moderately painful; inflammation, abscess, and cancer, which may give rise to severe suffering.

It is from the gall-bladder and its associated ducts that the severest form of liver pain originates—viz., hepatic colic. A particularly tender point, which is of much diagnostic importance, is found at the ninth right costal cartilage as an evidence of an inflamed, impacted, or cancerous gall-bladder, or a gallstone lodged in the common duct.

ABDOMINAL PAIN, MORE OR LESS GENERAL, OFTEN SHARP OR COLICKY

- Gastralgia (usually Hyperacidity, or Disease of Cord)
- Arsenical or Mercurial Poisoning
- Cancer or Ulcer of Stomach
- Enteroptosis
- Enteralgia
- Fæcal Impaction, or Concretion
- Lead Colic
- Flatulence
- Mucous Colic. Enteritis
- Appendicitis (onset)
- Intestinal Obstruction
- Intestinal Ulcer or Perforation
- Intestinal Tuberculosis
- Peritonitis
- Hyperæsthesia
- Rheumatism of Diaphragm or Abdominal Walls
- Hernia
- Angina Pectoris (spasm of Sclerosed Abdominal Arteries)
- Embolism of Superior Mesenteric Artery
- Cysts of Mesentery
- Hysteria or Neurasthenia (infrequent)
- Lumbo-Abdominal Neuralgia
- Tabes (gastric, or intestinal crisis)
- Floating Kidney (Dietl's crisis)
- Large Hydronephrosis
- Thoracic or Abdominal Aneurism (occasional)
- Disease of Pancreas
- Gastro-Intestinal Influenza
- Henoch's Purpura
- Visceral Crisis of Erythema Multiforme
- Leucæmia
- Dysmenorrhœa
- Diabetes
- Raynaud's Disease
- Pott's Disease (in child)
- Spondylitis Deformans
- Pneumonia (in child)
- Pleurisy (especially diaphragmatic)
- Pericarditis or Valvular Disease

PAIN



EPIGASTRIUM

- Hyperæsthesia
- Neurosis, Catarrh (acute or chronic), Ulcer, Erosions, Carcinoma, Dilatation, or Gout of Stomach
- Gastralgia, Hyperchlorhydria
- Irritant Poisoning
- Appendicitis
- Ulcer of Duodenum
- Enteroptosis
- Impaction of Transverse Colon
- Congested Left Lobe of Liver, Hepatoptosis
- Gallstones
- Carcinoma or Inflammation of Pancreas
- Disease of Vertebræ
- Pneumonia (especially in children)
- Diaphragmatic Pleurisy
- Addison's Disease
- Cholera Asiatica
- Uræmia
- Acute Rheumatism (especially in children)

Fig. 15.—The possible causes of pain in various parts of the body.

Pancreas.—Acute inflammation of the pancreas is usually attended with epigastric pain, so also with cancer of the same viscus.

Kidney.—The typical painful disorder of the kidney is renal colic. Pyelitis may cause pain, not only in the lumbar region but also, as the only algetic symptom, above the pubes. A movable or floating kidney, under circumstances not as yet definitely ascertained, is the source of much pain, which may become excessive if the ureter is twisted or kinked (Dietl's crisis). A perirenal abscess may be responsible for pain and swelling in one posterior lumbar region.

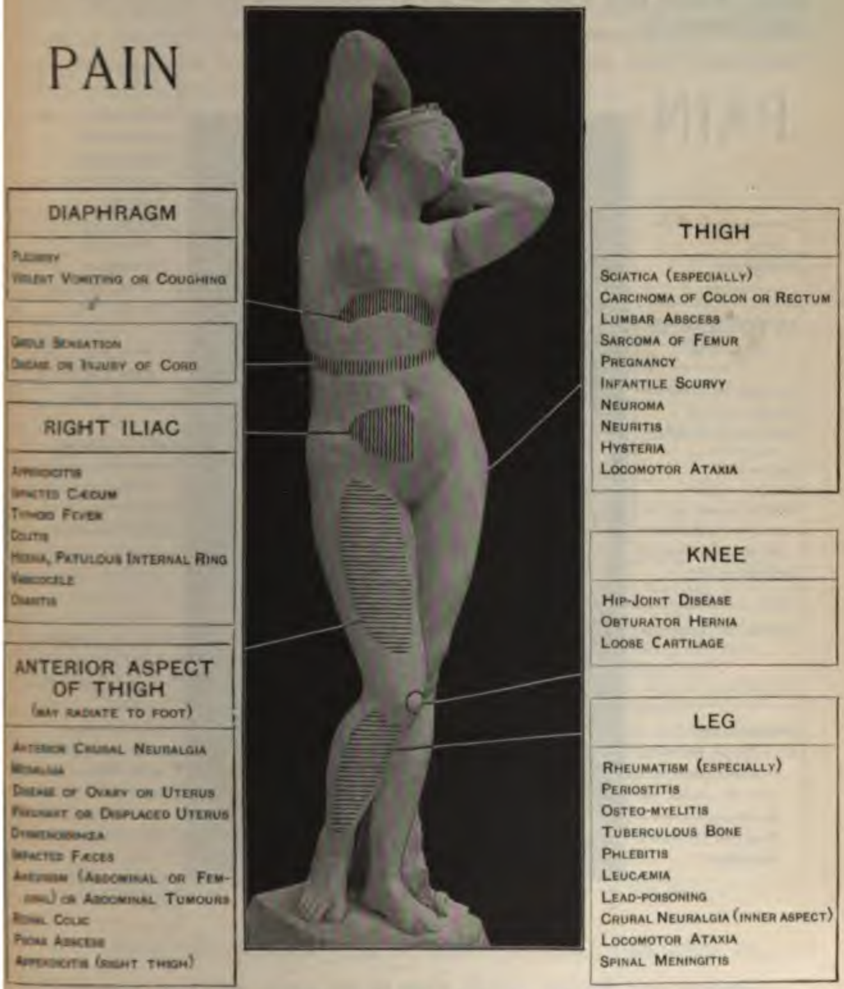


Fig. 16.—The possible causes of pain in various parts of the body.

Spleen.—Some enlargements of the spleen are painful because of the dragging weight of the organ, as in the malarial and leucæmic forms; others give pain because of the rapid swelling which overstretches its fibro-elastic framework, as in the acute enlargement of certain febrile diseases. Pain is also present as an evidence of inflammation of its peritoneal investment (perisplenitis). Splenic pain is felt in the left hypochondrium and the left side of the chest posteriorly.

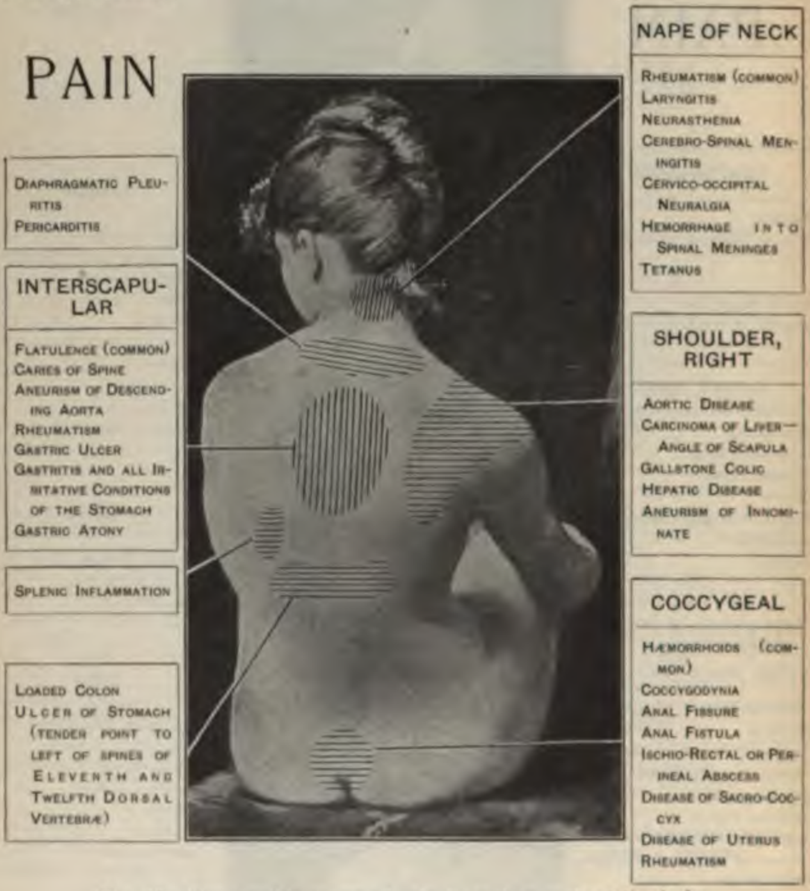


Fig. 17.—The possible causes of pain in various parts of the body.

Genitalia and Bladder.—Pain, more or less acute, is felt as a symptom of diseases or displacements of the uterus and ovaries, pelvic inflammations, the pregnant uterus, ectopic gestation, and dysmenorrhœa. According to the nature and severity of the lesion, the pain is referred to the sacral and pubic regions (most frequently the former), the mammary gland, the anterior aspect of the thigh, and the external and posterior surfaces of the hip. Rarely pain in the heel and the wrist is due to ovarian disease, and in the hand to uterine disorders.

Prostatic disease may be responsible for pain in the sole of the foot; disease of the testicle and excessive venery, for sacral pain; varicocele, for pain in the groin; and cystitis and pyelitis, for pain in the pubic region.

Bones and Muscles.—The painful diseases of bone which give rise to some confusion in the practice of internal medicine are spinal caries, rachitis, or arthritis deformans, causing pain in the back, the lateral walls of the chest, the epigastric region, or generally diffused over the abdomen. A psoas abscess or malignant disease of the femur makes a painful thigh; a diseased sternum causes breast pain; disease of the hip joint, a painful knee; diseased ribs, lateral chest pain; and disease of a sacro-iliac joint, sacral backache.

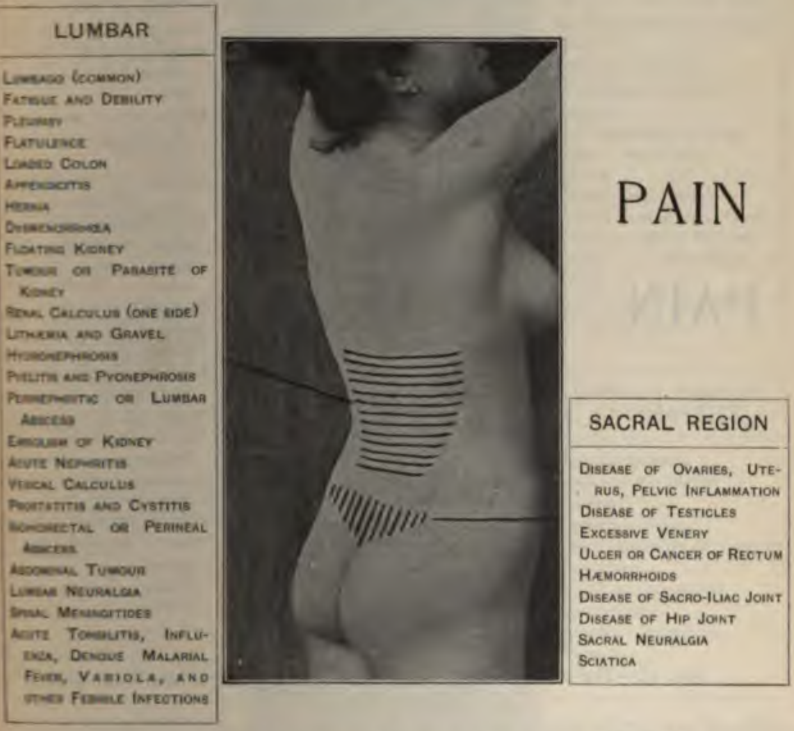


Fig. 18.—The possible causes of pain in various parts of the body.

The painful diseases of the joints are sufficiently catalogued in the diagram (Fig. 18).

The muscles are painful in pleurodynia (intercostal muscles), lumbago (lumbar region), and other varieties of muscular rheumatism; as a result of overexertion or debility, and in trichinosis. Cramps in the calves may be explained by too much walking or by the presence of chronic nephritis, and congestion or toxic irritation of the nerves supplying the calf muscles.

It can not be too strongly emphasised that the so-called "growing pains," if a not uncommon occurrence in children, are, in the great majority of cases, if not in all, of rheumatic origin. In every such case a careful watch should be kept for the evidences of endocarditis or pericarditis. Neglect in this respect may result in the unnoted development of serious valvular defects. These pains are in the muscles, rather than in the joints, and, as a rule, are present only, or are worse, during damp weather.

Patients frequently complain of a general aching, soreness, or painful stiffness of all of the muscles of the body. It is a symptom common to a large number of diseases and conditions, some of which are here mentioned. Thus, it may be present in the initial stages of the acute specific infections, especially typhoid fever, cerebro-spinal meningitis, epidemic influenza, acute rheumatic fever, relapsing fever, cholera, and the exanthemeta, as well as in septicæmia and all acute febrile diseases. It may occur in coryza and tonsilitis; and in alcoholism, syphilis, and anæmia. It is a prominent symptom in scurvy, trichiniasis, and polymyositis. It is a not uncommon subject of complaint in gastro-duodenal catarrh with involvement of the bile-ducts; in the rare acute infection jaundice (Weil's disease); and in locomotor ataxia. Finally it may attend convalescence from any acute illness.

PAIN

PAIN OR TENDERNESS AT VARIOUS POINTS ALONG THE SPINE

Hysteria, or Neurasthenia, especially Traumatic
Meningitis or Meningeal Hemorrhage (various forms)
Myelitis or Tumour of Cord, Syringomyelia
Pott's Disease, Osteomyelitis, Spondylitis Deformans, Spinal Curvature
Carcinoma of Liver
Ulcer of Stomach or Duodenum
Aneurism of Descending or Abdominal Aorta
Mediastinal Tumour
Abscess, Subphrenic, Perinephric, or Lumbar
Rachitis
Scurvy
Febrile Diseases, especially Influenza, Tonsilitis, Variola, and Dengue (general and acute)

Fig. 19.—The possible causes of pain in various parts of the body.

Miscellaneous Causes of Pain.—Mediastinal tumour or abscess, or enlarged bronchial glands may produce pain in the right hypochondriac region and either lateral wall of the chest; subphrenic abscess, in either hypochondrium. The sharp pain under either costal margin after some violent exercise, especially running, is probably familiar; so also the epigastric and costal arch pain after violent coughing or vomiting.

Anæmia and debility are frequently attended by a more or less severe aching pain along the spine and the left lateral wall of the chest, extending into the left hypochondriac region. A general backache is common in acute febrile diseases, and is of diagnostic severity in epidemic influenza, tonsilitis, smallpox, and dengue. Diffuse soreness of the whole body in rickets, and of the back and lower extremities in infantile scurvy, upon voluntary or assisted movement, is in either case a very characteristic symptom. As in locomotor ataxia, so in diabetes, Raynaud's disease, and, more rarely, in abdominal aneurism, there may be sudden, perhaps violent, attacks of general abdominal pain.

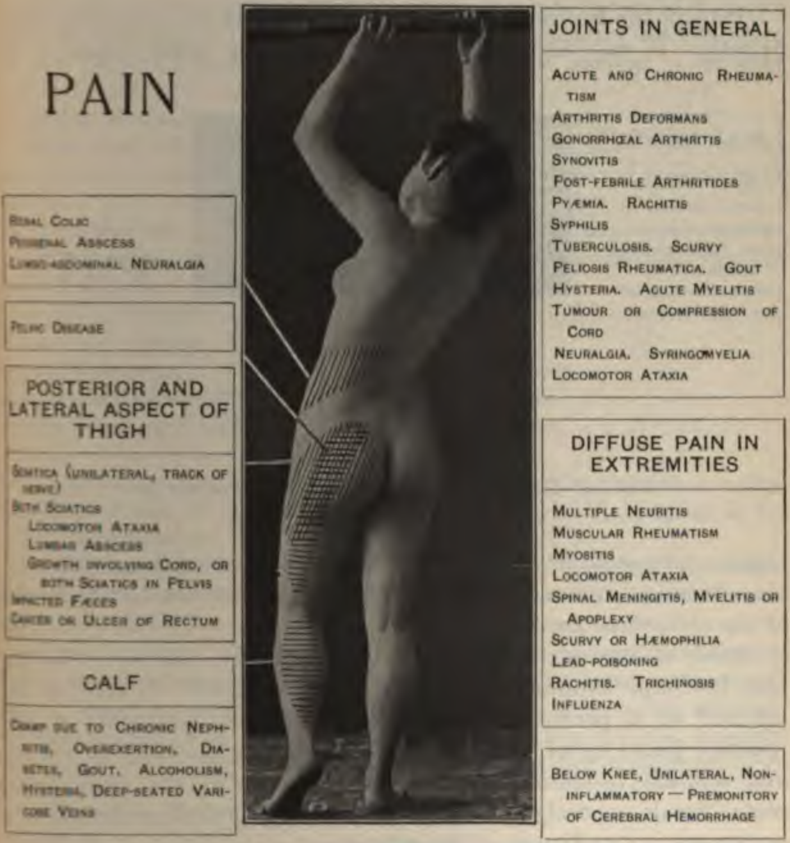

PAIN
Renal Colic
Perineal Abscess
Lumbo-abdominal Neuralgia
Pelvic Disease
POSTERIOR AND LATERAL ASPECT OF THIGH
Sciatica (unilateral, track of nerve)
Both Sciatics
Locomotor Ataxia
Lumbar Abscess
Growth involving Cord, or both Sciatics in Pelvis
Impacted Fæces
Cancer or Ulcer of Rectum
CALF
Cramp due to Chronic Nephritis, Overexertion, Diabetes, Gout, Alcoholism, Hysteria, Deep-seated Varicose Veins
JOINTS IN GENERAL
Acute and Chronic Rheumatism
Arthritis Deformans
Gonorrhœal Arthritis
Synovitis
Post-febrile Arthritides
Pyæmia. Rachitis
Syphilis
Tuberculosis. Scurvy
Peliosis Rheumatica. Gout
Hysteria. Acute Myelitis
Tumour or Compression of Cord
Neuralgia. Syringomyelia
Locomotor Ataxia
DIFFUSE PAIN IN EXTREMITIES
Multiple Neuritis
Muscular Rheumatism
Myositis
Locomotor Ataxia
Spinal Meningitis, Myelitis or Apoplexy
Scurvy or Hæmophilia
Lead-poisoning
Rachitis. Trichinosis
Influenza
Below Knee, Unilateral, Non-inflammatory — Premonitory of Cerebral Hemorrhage

Fig. 20.—The possible causes of pain in various parts of the body.

(*e*) *Pain in penis, testicle, and perinæum.*—Pain in the *penis* arises mainly from the presence of a vesical calculus, or the passage of uric acid crystals (gravel). It may however result from other sources of irritation such as a calculus in the ureter or the urethra, or a urethral stricture; from inflammation or ulceration of the bladder; from an enlarged, tuberculous, or inflamed prostate gland; or from excessive venery.

Pain in the *testicle* is most frequently due to an orchitis or epididymitis. It may also be caused by cancer or tuberculosis of the organ; varicocele, or excessive venery; vesical or renal calculus; hernia, abdominal tumours, especially tumour of the colon; and aneurism of the abdominal aorta.

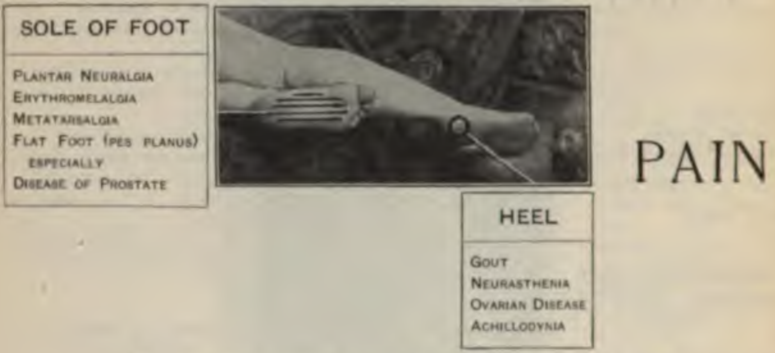


Fig. 21.—The possible causes of pain in the foot and heel.

Pain in the *perinæum* is most commonly due to inflammation (perhaps suppurative) of the prostate gland. It may be significant of a perineal or ischiorectal abscess, carcinoma of the prostate gland or of the bladder; or stone in the bladder, cystitis, and seminal vesiculitis; hæmorrhoids, fissure of the anus, ischiorectal fistula, neuralgia of the rectum or a rectal crisis of locomotor ataxia; or disease of the uterus.

II. TENDERNESS

Tenderness is pain or soreness produced by pressure. The greater portion of diseases attended by spontaneous pain will manifest tenderness on pressure over the seat of pain; but this is not an invariable rule, as either pain or tenderness may exist separately. More or less tenderness exists over the seat of all *inflammations*. Prolonged or repeated attacks of neuralgia may be attended by tender points corresponding to the exit of the affected nerve through bone or fascia.

The diagnostic indications to be derived from the *localization of tenderness* are as follows:

Head and Scalp.—Neuralgia of the cervico-occipital nerves, hemicrania, and periostitis (the latter usually syphilitic), or hysteria may be suspected if tenderness of the scalp is found to exist. After or during any severe headache there may be a temporary hyperæsthesia of the scalp, so that combing or brushing the hair is painful.

Mastoid Process.—Suppurative mastoiditis.

Malar Bone.—Tenderness below the inner part of the malar bone over the superior maxilla is found in suppurative or malignant disease of the antrum or maxilla.

Neck.—Swollen or inflamed lymphatic glands from any cause, cervico-occipital neuralgia, cervical myalgia, and cervical caries are the most frequent causes of tenderness in the neck.

Back.—1. Tenderness along the *whole length* of the spine, either continuous or in a series of points may be due to

Arthritis deformans,
Cerebro-spinal meningitis,
Hysteria and Neurasthenia,
Myelitis,
Periostitis (spinal),
Spinal irritation,
Spinal meningitis,
Rheumatism.

2. Tenderness over the *dorsal spine* may be caused by thoracic aneurism, tuberculous disease of the spine, enlarged bronchial glands, or tumor in the posterior mediastinum.

3. Tenderness over *lumbar spine*, by abdominal aneurism, subphrenic abscess, perinephritic abscess, suppurative and acute nephritis, lumbar abscess, and tuberculous disease of the spine.

Thorax (in front).—Tenderness may be due to ostitis or periostitis of sternum or ribs, or inflammation of the costal cartilages; tender breast, to hysteria or tumour; tender points, to intercostal neuralgia, projecting or eroding aneurism, or abscess of chest wall. A tender point, evoked by deep pressure, especially along the line of the inter-lobar fissures, is an important sign in suspected localized empyema.

Præcordial tenderness may arise from pericarditis.

Abdomen.—1. General tenderness of the abdomen may be caused by

Dysentery,
Dysmenorrhœa,
Strangulated hernia,
Hysteria,
Cancer of intestine,
Ulceration of intestine,
Simple peritonitis,
Suppurative peritonitis,
Tuberculous peritonitis,
Diaphragmatic pleuritis,
Irritant poisons,
Rheumatism of abdominal walls.

2. Hypochondrium.—Tenderness in the right hypochondrium may be due to

Empyema of gall bladder,
Gallstones,
Influenza (epidemic),
Abscess of liver,

Acute inflammation of liver,
Acute yellow atrophy of liver,
Cancer of liver,
Inflamed hydatids of liver,
Syphilis of liver,
Malarial fever (both sides),
Perihepatitis,
Relapsing fever.

3. Epigastrium.—Tenderness in the epigastrium may be indicative of

Addison's disease,
Gallstones,
Acute gastritis,
Chronic gastritis,
Hypochondriasis,
Hysteria,
Acute yellow atrophy of liver,
Pancreatitis,
Acute pericarditis,
Diaphragmatic pleurisy,
Irritant poisons,
Ulcer of stomach.

4. Iliac Region.—Tenderness in one or both iliac regions may arise from pelvic peritonitis and pelvic inflammations in general, hysteria, fecal impaction, and membranous enteritis (usually in left side). Tenderness in the right iliac region occurs with typhoid fever and appendicitis.

5. Hypogastrium.—Tenderness in the hypogastric region may originate from cystitis, pelvic inflammations, or congestion, hysteria, and dysmenorrhœa.

Extremities.—Tenderness found in the extremities may be due to neuritis, in which case it will be found along the course of the nerve; to phlebitis, the tender vein being felt as a hard cord; to ostitis or periostitis, or malignant disease of the bone; to myalgia, rachitis, scurvy, trichinosis, or tetanus; to disease of the joints. Tender joints are usually due to rheumatism, acute, chronic, or gonorrhœal; to arthritis deformans, synovitis, sprain, gout, tuberculosis, pyæmia, or hysteria.

III. PARÆSTHESIAS

The word paræsthesia is here used as embracing a number of subjective sensations, largely of cutaneous origin. Among the paræsthesias are: Sensations of bearing down, of coldness, faintness, formication, itching, fulness, the girdle sensation, sensation of heat, numbness, tingling, burning, oppression or weight, præcordial constriction and sinking, throbbing, tightness, and weakness or debility.

Diagnostic Significance of Paræsthesias in General.—Leaving out of account the hyperæsthesias and anæsthesias, there is a large class of cases in which some of the peculiar and annoying sensations just mentioned are the only symptoms of which complaint is made. Such cases, mainly in women, form a considerable proportion of the office practice of the physician, especially among those in the better grades of society. The sensations of most frequent occurrence in these patients are weakness, numbness, flashes of heat or cold, throbbing, general or localized, tingling, burning, and sudden perspirations, as well as other sensations which are described in the most bizarre manner, or, possibly, said to be indescribable. A careful examination in such cases will reveal, as a rule, nothing beyond a moderate anæmia, slight digestive disturbance, constipation, irregular innervation, and poor nutrition. The previous history will tell of overwork of some kind, grief, worry, anxiety, or a congenital deficiency in what, for lack of a better term, is called "vitality." Such cases must be classified as examples of a slight degree of neurasthenia.

If, however, certain of these paræsthesias, such as numbness, prickling, formication, tingling, or burning, become localized in a certain nerve or a definite part of the body, they become comparable to a definite disease (DANA). In the majority of cases such localized subjective sensations are due to the same kind of irritation as that which gives rise to a neuralgia, except that it is of lesser intensity and the sensation is not sufficiently severe to conform to the usual conception of pain. The most frequent perversion of sensation is that of a prickling numbness, less frequently a sensation of burning. When such paræsthesias affect the head, there are sensations of pressure, burning, or constriction—incomplete headaches—and their most common cause will be found in the presence of neurasthenia or lithæmia. If situated in individual nerves, occurring especially in the brachial, ulnar, crural, and plantar, they may be regarded as incomplete neuralgias. If the feet or hands, or both, are generally involved, it constitutes acro-paræsthesia.

Diagnostic Significance of Special Paræsthesias.—

1. *Bearing Down.*—The sensation of bearing down is felt chiefly in the pelvis. The causes of this sensation are for the most part diseases or conditions affecting the uterus, especially membranous dysmenorrhœa. Hemorrhoids, undue fulness of the bladder, and sometimes prolonged standing may also be responsible.

2. *Coldness or Chilliness.*—Subjective sensations of coldness, existing with a normal or subnormal temperature, may be due to hysteria, lateral sclerosis, myxœdema, neurasthenia or syringomyelia; and chilliness may be present as one of the prodromal symptoms of an attack of migraine.

Cold sensations (psychro-æsthesias) may implicate a whole extremity, or all four extremities, without being limited to certain areas or the distribution of a nerve (DANA). Not infrequently these are associated with other paræsthesias (prickling and numbness), with pain, and with local disturbances of the circulation. This form of cold sensation is usually indicative of a mild neuritis due to the abuse of alcohol, or to a rheumatic diathesis, exposure, and toxæmias. It may also occur in locomotor ataxia and in the early stage of syringomyelia.

On the other hand, the cold sensation may be limited to some special area, usually upon the buttock or thigh, sometimes the calf, corresponding pretty accurately to the distribution of a nerve. The feeling is as though something tangible and cold were in contact with the part. The sensation may be so distressing that it is very properly termed a cold pain (psychro-algia). These circumscribed areas of coldness are found, as a rule, in patients over 40, more commonly in men. The causes may be found in pressure upon the nerves due to occupation, rheumatism, or exposure, and in many cases a neuropathic diathesis is an underlying agency. If obstinate, syringomyelia or, rarely, locomotor ataxia may be responsible for this symptom.

3. *Faintness.*—A sensation of weakness with a tendency to syncope, persistent, recurrent, or acute, may be present as an accompaniment of the various forms of anæmia, angina pectoris, fatty heart, thrombosis of the pulmonary artery, pneumothorax, thoracic aneurism, ascites (especially during tapping), and tympanites. It may be present as a result of emotion, fatigue, excessive heat, painful affections, depressing poisons, and as an evidence of shock after injuries. As an idiosyncrasy it attends the act of defecation in certain persons, and is a frequent event in Ménière's disease.

4. *Formication, Itching.*—Formication is a sensation as of ants or other insects crawling over the skin. It is a variety of itching or pruritus, and in varying degrees and in different parts of the body may result from many diseases of the skin, including the exanthemata. Affecting the external genitals, it may be due to diabetes, leucorrhœa, or neuroses; the anus, to seat worms or hemorrhoids. It is an occasional premonitory symptom of apoplexy, and may be present as a consequence of a tumour of the brain, in the part supplied from the seat of the lesion. More or less general pruritus frequently occurs in hysteria and neurasthenia, also in locomotor ataxia, chronic spinal meningitis, chronic myelitis, disseminated sclerosis, chronic lead poisoning, and tetany. It is general and frequent in jaundice, less often it is found in gout and cases of granular kidney. It is an occasional result of the administration of morphine (especially affecting the nose), copaiba, and ergot.

General itching or pruritus, if persistent, should lead to an examination for the existence of gout, lead poisoning, chronic interstitial nephritis, or diabetes mellitus.

5. *Fulness.*—A sensation of fulness in the chest or epigastrium is caused by chronic gastritis, nervous dyspepsia, or dilatation of the stomach, as well as by cardiac hypertrophy, pulmonary emphysema, chronic peritonitis, tympanites, or malarial fevers.

6. *Girdle Sensation.*—A subjective sensation as of a constricting band around the trunk is found in connection with locomotor ataxia, chronic myelitis, injury or tumour of the spinal cord, and inflammation or tumour of the spinal meninges, also from an enlarged liver. After prolonged vomiting or repeated paroxysms of violent cough, a similar sensation may be felt, corresponding to the insertion of the diaphragm, and due to a strain of that muscle.

7. *Heat.*—Subjective sensations of heat may be felt in the epigastrium and beneath the sternum in pyrosis and irritant poisoning, and to a less degree in acute bronchitis; in the back, in spinal irritation or neurasthenia. Flashes of heat, with or without flushing of the face and sudden perspirations, are common attendants of the menopause, as well as disease of the pelvic viscera and conditions of debility and nerve tire. Subjective sensations of heat may be felt in the back and epigastrium in paralysis agitans.

8. *Numbness, Tingling, Burning.*—These are of rather common occurrence, and may possess diagnostic value. Particularly in the feet, they are in some cases the first symptoms of locomotor ataxia, myelitis, or chronic spinal meningitis, and are very frequent in neurasthenia and hysteria, and (in the fingers

and toes) are not infrequent in arthritis deformans. Numbness (fingers, occasionally scalp and feet) on waking in the morning, which at first passes away, but later remains during the day, is acro-paræsthesia or waking numbness. Localized numbness is sometimes caused by a tumour of that portion of the brain which supplies the area of abnormal sensation. In tetany there is numbness of the fingers and toes, and tingling or burning is an occasional premonitory symptom of neuralgia or herpes zoster. Certain poisons also may cause numbness.

The diseases or poisons of which numbness, tingling, or burning may be indications are as follows:

Aconite poisoning (general numbness),
Apoplexy (an occasional premonitory symptom),
Appendicitis (occasionally in right leg),
Arthritis deformans (hands and feet),
Beri-beri (multiple neuritis),
Brain tumour (part supplied from seat of lesion),
Bromism,
Carbolic acid (local contact of),
Chronic spinal meningitis,
Epilepsy (occasionally as a part of the aura),
Herpes zoster (peripheral terminations of affected nerve),
Hysteria (a frequent symptom),
Injuries of nerves,
Locomotor ataxia (feet and legs),
Myelitis (early stage),
Myxœdema,
Neuralgia (premonitory),
Neurasthenia (frequent),
Neuritis,
Sciatica,
Tetany (fingers and toes).

9. *Oppression or Weight.*—When felt in the *chest*, is most frequently found in hæmoptysis, hysteria, spasmodic asthma, angina pectoris, nervous dyspepsia, and chronic gastritis; less commonly in cardiac or pericardial disease, Basedow's disease, exophthalmic goitre, and tumour of the mediastinum. When felt in the *epigastrium*, is usually caused by neurasthenia, chronic gastritis, nervous dyspepsia, or, rarely, as a premonition of gastric hemorrhage. A feeling of pressure or weight in the *head* is common in neurasthenia and hypochondriasis, and is occasionally premonitory of apoplexy or is a part of the epileptic aura. A similar sensation in the *abdomen* or *pelvis* attends the presence of abdominal or pelvic tumours.

10. *Præcordial Constriction and Sinking.*—A typical example of this distressing symptom, associated with agonizing pain, is seen in angina pectoris, and to a lesser degree in pseudo-angina. A sensation of cardiac weakness and sinking may be indicative of dyspepsia or gastritis, especially the acute forms. Severe acute diarrhœa, and especially cholera (sporadic or Asiatic), will rapidly exhibit this symptom. It is not infrequently a concomitant of tympanites, cardiac dilatation, or pericarditis, and, finally, it may be a subject of complaint in hysteria, neurasthenia, and hypochondriasis.

11. *Throbbing.*—This, as a subjective sensation, felt over the entire body or in the head and extremities, is not infrequent in hysteria, neurasthenia, vasomotor ataxia, the anæmias, and aortic regurgitation. Locally it is felt in phlegmonous inflammations and over the seat of an aneurism; in the neck and head, in cases of exophthalmic goitre; in the head, in migrainous headaches; in the præcordial area, in cardiac hypertrophy or palpitation; and the "throbbing aorta" is very common in gastritis and neurasthenia.

12. *Tightness.*—This, when substernal, is usually associated with the dry stage of an acute tracheitis and bronchitis of the larger tubes. The term is also applied to the character of the cough in the same stage.

13. *Weakness or Debility.*—A feeling of muscular weakness, debility, or lassitude attends the prodromal stage of fevers, and is a sequel, more or less prolonged, of all febrile and exhausting diseases. It may also be a concomitant of the dyspepsias; the overuse of tea, coffee, alcohol, and tobacco; and rheumatism, gout, and lithæmia.

This symptom, when persistent, has a distinct clinical value in suggesting the presence of neurasthenia (most frequent), pulmonary tuberculosis (even without physical signs), diabetes, anæmia, influenza (obstinate), latent pleurisy, and exophthalmic goitre.

SECTION VI

VERTIGO

Vertigo or dizziness is a subjective sensation of loss of equilibrium. The patient feels as if he himself were whirling, sinking, or rising (*subjective* vertigo), or surrounding objects may appear to be rapidly revolving, sinking, or rising (*objective* vertigo). *Horizontal* vertigo is felt when reclining and disappears on rising. In a severe case the sensation may be so sudden and profound as to be likened to a blow; in the slighter degrees there is a swimming lightness in the head. The severe and acute forms are usually of brief duration, but in its lighter forms it may last for weeks and months (*status vertiginosus*). The gait is reeling or staggering, like that of a drunken man. Slight nausea often co-exists, and vomiting may follow a sharp attack. Loss of consciousness, if occurring, is only momentary.

Vertigo is a cause of so much alarm to the patient that the determination of its origin in a given case is of great importance. In by far the larger proportion of cases it is due to neurasthenia, especially the lithæmic form, gastric disorders, arteriosclerosis, aural disease, or eye strain, these causes being arranged approximately in the order of relative frequency. Very rarely the vertigo is essential, without discoverable cause.

In all cases a careful examination should be instituted for the discovery of diseases or conditions which may be responsible for this symptom, as follows:

Neurasthenia and Lithæmia.—(*a*) If a patient with a history of overstrain, or congenital deficiency in nerve force, complains of constant weariness, feelings of pressure in the head, and other characteristic symptoms, with vertigo of brief duration and moderate severity, but of frequent occurrence, the vertigo is due to neurasthenia (*q. v.*).

(*b*) If in addition the urine is high coloured, strongly acid, and with a high specific gravity (1.028 to 1.032), the cause of the vertigo is neurasthenia *plus* lithæmia. The latter alone may be a cause of vertigo.

Gastric Disorders.—(*a*) If a patient of middle age complains of a sudden vertigo with headache after too free indulgence in the pleasures of the table, and the symptoms slowly disappear after vomiting or the administration of a purgative, the vertigo is due to acute indigestion.

(*b*) If there is sudden headache, dimness of vision (blind headache), marked vertigo causing a reeling gait, temporary mental confusion, and possibly vomiting of an extremely sour fluid, the vertigo is due to hyperacidity of the stomach contents (*q. v.*).

(*c*) If there is a history, extending over months or years, of oppression or distress after eating, epigastric tenderness, etc., with a rather constant but slight vertigo, it is due to chronic gastritis (*q. v.*).

Arteriosclerosis; Valvular Disease of Heart; Aneurism.—(*a*) If a man or woman, past middle age, complaining of constant slight vertigo, intensified on excitement or exertion, presents sclerosed arteries, arcus senilis, and ringing aortic closure, with or without moderate cardiac hypertrophy, the vertigo is due to arteriosclerosis.

(*b*) A similar complaint in a patient with water-hammer pulse, marked cardiac hypertrophy, and a diastolic murmur, most intense over the aortic cartilage, is due to aortic regurgitation.

(*c*) Vertigo may attend an aneurism of the thoracic aorta, because of the disturbed intracranial circulation.

Ménière's Disease.—If a patient, generally a man past 40, has sudden attacks of vertigo, with tinnitus or incomplete deafness in one or both ears, the combination is sufficient to warrant the diagnosis of Ménière's disease (aural vertigo, labyrinthine vertigo).

Vertigo accompanying other diseases or conditions is rarely associated with tinnitus or deafness. The vertigo of Ménière's disease is abrupt in its onset, and the patient may fall before he has time to grasp some near-by object. There may be a momentary loss of consciousness. He becomes pale, and the face is covered with perspiration. Nausea follows, and vomiting may take place. The tinnitus may consist of hissing, buzzing, roaring, or throbbing noises, more rarely sudden and loud reports like pistol shots. The attacks may recur several times in a day, or may be months apart. In the intervals there may be a more or less continuous giddiness. Rare symptoms during the attack are diplopia and nystagmus, double vision, or oscillatory movements of the eyeballs. The deafness is usually progressive but incomplete, affecting one or both ears.

The causes of Ménière's disease are variously stated. It has been attributed to disease of the labyrinth in particular, hemorrhagic or degenerative; to suppurative or other disease of the middle ear; to disease of the external ear (furuncle, inspissated cerumen); to a vasomotor neurosis of the vessels of the semicircular canals; to disease of the centres of hearing and equilibration.

Eye Strain.—In a patient presenting symptoms of eye strain, viz., indistinctness or blurring of sight, brow pain or occipital headache, reddening or watering of the eyes, these following use of the eyes upon near work, as reading, writing, and sewing, together with vertigo which is not severe but rather persistent, and examination of the eye reveals errors of refraction, strabismus, or exophoria, the vertigo may be due to the ocular defect. The diagnosis can be made positively only by the disappearance of the vertigo after the necessary corrections, optical or operative, have been made.

Among the less common causes of vertigo are: epilepsy, in which vertigo may be a part of the aura preceding the convulsion or, alone, usually with a momentary loss of consciousness, constitute an attack of *petit mal*, and is discriminated from Ménière's disease by the absence of tinnitus; anæmia, especially upon exertion; brain tumours; the abuse of tea, coffee, alcohol, and tobacco; auto-intoxication from absorption of decomposing substances; and chronic interstitial nephritis. Vertigo may also be a symptom of locomotor ataxia; disseminated sclerosis (rarely); great mental overwork or excitement in neurotic persons (sudden, with faintness and nausea, lasting several hours); or mechanical (sea-sickness, railway and elevator sickness). It may be transitory and caused by looking at a rapidly rotating body; *vertical*, by looking up toward or down from a height; *lateral*, occurring while the patient is walking alongside of some construction made up of similar parts, like a wall or fence; or *nocturnal*, felt in the act of going to sleep.

Laryngeal Vertigo (L. syncope, L. epilepsy).—This rare disease is either a true epilepsy or a reflex neurosis, probably the latter. It usually manifests itself in middle-aged neurotic men, who are suffering from laryngitis, bronchitis, bronchial asthma, or phthisis pulmonalis. The attack begins with laryngeal tickling or irritation, followed by a short cough, spasm of the larynx, dyspnœa, transitory syncope, and slight convulsive movements. Such attacks may recur every day or at intervals of a month or more. As a rule, the removal of the local lesions, when possible, will put a stop to the paroxysms.

SECTION VII

TEMPERAMENT—PSYCHICAL CONDITION—EXPRESSION OF FACE—DELIRIUM

DURING the general examination the physician should endeavour to form some conception of the mental characteristics of the individual, in health and as modified by disease. As a rule, the more highly trained the mind and the more elaborate and complex the social environment, the greater is the necessity for an appreciation of the personal traits of the individual. Influences which produce no apparent effect upon the labourer, toughened by hard life, will cause profound perturbation in the somewhat hyperæsthetic woman whose nervous system is in a state of tension from the demands of society, art, or literature. Delirium, for instance, is a much less significant symptom when encountered in a person possessing a sensitive nervous organization than when it is present in one of a phlegmatic and sturdy temperament.

If necessary, information may be obtained from friends or relatives concerning the mental traits of the patient in health. One should inquire especially as to the existence of extreme susceptibility to pain and exaggeration in the manner of expression; as to whether the patient is or is not easily elated or depressed, or given to undue solicitude about personal health; and other points which may be serviceable in estimating the value of the statements made by the patient, or the present deviation from the normal mental condition. Such inquiries should be made with circumspection and tact in order to avoid misconception of motives and the charge of making imputations uncomplimentary to the patient.

I. **Temperament.**—In this connection a word may be said in regard to the old classification of temperaments, into sanguine, bilious, nervous, strumous, and lymphatic. Considered as large groups, there is doubtless a partial justification for such divisions, but the attempt to place an individual patient in one of these is almost hopeless, as is clearly proved by the frequent necessity felt by the older writers for word combinations, of which *bilio-sanguine*, *lymphatico-sanguine*, are examples. As a matter of fact, the majority of men exhibit the characteristics belonging to more than one of the temperamental classes.

Nevertheless, it is often convenient to use the names given to the temperaments as descriptive terms, without the broad meaning which was formerly implied by their use. Thus it may be a material aid in the description of a particular case to say that the patient is of a *nervous temperament* (meaning thereby that his nervous system is sensitive), and that he has a *gouty diathesis*. Practically the tendency of modern writers is to retain some of the names of temperaments for the characterization mainly of mental qualities, while physical characteristics and predispositions toward disease are spoken of as diatheses (*q. v.*), a suitable qualifying adjective being prefixed.

Choleric Temperament.—Exhibits great irritability and strong passions, with very active voluntary muscles.

Phlegmatic or Lymphatic Temperament.—Characterized by a want of energy, small or slow reaction to psychic excitations, slightly developed passions, and little sensibility to bodily suffering.

Melancholic (*Atrabiliary*) *Temperament.*—Marked by slight irritability, united with strong capacity for reacting to sensory excitations, and by great persistence of the frame of mind, especially that of sullenness or dulness.

Nervous Temperament.—Characterized by marked sensitiveness of the nervous system to slight impressions.

Sanguine Temperament.—Characterized by marked irritability, great liability of the nervous system to exhaustion, and sudden changes in humour.

II. **Psychical Condition.**—In determining the present psychical condition of the patient it is necessary to test the *memory*, not only in regard to recent but also in regard to long past occurrences, as one of the first faculties of the mind to give way in conditions of weakness or disease is the power of recollection. It is also requisite to determine the power of *judgment*, the ability to follow a logical train of thought and to appreciate the relative value of facts. An abnormal increase in mental activity, with a rapid flow of ideas and a certain exaltation of the frame of mind, may be encountered, as well as its opposite, a depressed, sluggish, and melancholy humour. It is, of course, necessary to make due allowance for the degree of education and the age of the patient. The emotional state of the patient is a part of the psychical condition; and, as an index to the mental status, the expression of the face may give valuable information. The occurrence of delirium and more or less complete unconsciousness may also be conveniently included in this part of the general examination.

The class of symptoms here considered may occur in connection with either acute or chronic disease, and the disease may be one which affects the brain and nervous system alone or may be a general morbid condition of which the symptoms referable to the nervous system are merely indications or effects, as follows:

(1) **Facial Expression.**—(*a*) An *anxious* expression is observed in diseases attended with pain, as angina pectoris, aneurism pressing on nerve branches or causing erosion of the vertebræ, simple intestinal or lead colic, pleurisy and pneumonia, intestinal obstruction from any cause, and especially in peritonitis. A peculiarly worried and anxious look is especially important if occurring in patients with the symptoms of appendicitis, as it is significant of peritoneal involvement. It also attends diseases which cause dyspnœa, as asthma, pneumothorax or large pleural effusions, and laryngeal stenosis from any cause. It is a frequent accompaniment of general septic conditions (septicæmia, pyæmia) and the beginning of many infectious fevers, as well as of pericardial and endocardial inflammations.

(*b*) An expression of fright and alarm is usual in connection with large hemorrhages, and is frequently seen in nervous and hypochondriacal individuals.

(*c*) A face devoid of expression, vacant and unmeaning in aspect, may be witnessed in varying degrees in facial paralysis, idiocy, dementia, and hydrocephalus. Its most frequent occurrence is in typhoid fever (hebetude) and the typhoid condition which attends so many low fevers and septic processes.

(*d*) There are certain contortions or spasms of the facial muscles which give a peculiar expression to the face, as in chorea, hysteria, or insanity. Here, too, may be classed the *risus sardonicus*, due to contraction of the depressors of the angles of the mouth, which may be seen in tetanus and strychnine poisoning, and which has been observed also in inflammation of the diaphragmatic pleura and general peritonitis.

For further information concerning the *facies* of disease, consult index.

2. **Emotional State.**—(*a*) *Mental Depression.*—A melancholy humour is characteristic of hypochondriasis, melancholia, jaundice and other derangements of the digestive system; and an alternation of high and low spirits not infrequently attends neurasthenia, hysteria, chloro-anæmia, embolic or thrombotic cerebral softening, and the menopause.

(*b*) *Mental Excitement.*—A condition of emotional excitement is present in certain forms of mania, delirium, and the first stage of acute alcoholism. It may be seen also during the administration of chloroform, ether, or nitrous oxide.

(*c*) *Irritability of temper*, manifested by outbursts of anger, or, in a lesser degree, by querulousness, may be present in connection with gout, rheumatism, jaundice, neurasthenia, and chronic invalidism.

(*d*) A noticeable *change of the habitual temper*, usually from placid to fretful, less frequently the opposite, is witnessed in pregnancy, melancholia, typhoid fever in its middle course, in the aura of epilepsy, and occasionally in the early stage of exophthalmic goitre.

(3) **Intellection.**—The disorders of intellection embrace dulness or confusion of the mental processes, loss of memory, delusions, illusions, hallucinations, and delirium. Disorders of consciousness will be considered under a separate heading.

(*a*) *Dulness or confusion* of mind may be observed in patients who are quite conscious, and is manifested by difficulty or slowness in apprehending the meaning of questions which may be put to them, and a corresponding difficulty in framing the proper answers. In the extreme degrees it is impossible to overcome the mental apathy sufficiently to obtain intelligible replies. The patient makes an evident effort to grasp the significance of the spoken words, but the endeavour is fruitless (see also Aphasia). The hebetude of typhoid fever has been mentioned. Mental dulness is seen in cerebral inflammations at certain stages of their progress, in some of the scleroses of the brain, in cerebral thrombosis and embolism, myxœdema, typhus and other low fevers, and in some of the psychoses or insanities. Confusion of thought may be incidental to neurasthenia, anæmia, brain tumour, and old age.

(*b*) *Loss of memory* usually accompanies dulness or confusion of mind, but is especially characteristic of neurasthenia, epilepsy, overuse of the bromides, and beginning insanities.

(*c*) *Delusions, Hallucinations, and Illusions.*—A *delusion* is an absurd and unfounded belief, and if it occurs in an insane person it is an insane delusion. The truth of the belief and the insanity of the believer are matters of judgment. A belief which is false and ridiculous to one may be an article of faith with another. In ordinary medical diagnosis it is usually easy to distinguish between true and false beliefs without splitting hairs.

An *hallucination* is a sense perception without a physical basis. If one imagines that he hears a sound or smells an odour, when the sound or odour does not exist, he is the subject of an hallucination. Any one of the senses may be the agent of an hallucination.

An *illusion* is a false interpretation of objects which really exist. If one sees a chair and imagines it to be a man, or hears a gentle rap upon the door and insists that it is an explosion, he is the subject of an illusion.

If the patient is unaware of the falsity of his hallucinations or illusions, they are said to be delusive; if he knows their falsity, they are non-delusive.

Delusions, hallucinations, and illusions are characteristic of the insanities. They are occasionally seen in hysteria, and to some degree in every case of hypochondriasis, both of which are considered on good authority to be forms of insanity. False beliefs and false or perverted sense perceptions also constitute a part of the mental condition known as delirium.

(*d*) *Delirium.*—This is a state of mental agitation characterized by restlessness, incoherence of speech, delusions, and sensory perversions. Two varieties of delirium are recognised, although transition forms are common. The first form is termed *active* or *wild delirium*, in which the patient tries to escape from bed, shouts, struggles with imaginary enemies, and requires forcible restraint. The other is a *low* or *muttering* delirium, the patient lying with comparative quietness, but incessantly engaged in incoherent and disjointed converse with imaginary personages, or communing with his own disordered brain. The onset of delirium may be sudden or slow. If coming slowly, the first indication of its presence may be a certain confusion and difficulty in realizing his surroundings after waking from sleep, the mental confusion lasting for a longer period on each occasion, and finally developing into well-marked delirium.

Active or wild delirium is typically seen in delirium tremens; low or muttering delirium in the 2d and 3d weeks of typhoid fever. A practical point to bear in mind is that a quiet delirium may shift very suddenly into the active form.

Aside from the insanities, delirium may be present in the acute infectious diseases. Any febrile disease in children may manifest it to a greater or less degree. I have seen several cases of dysmenorrhœa in young women, attended with a rather active delirium lasting from 12 to 24 hours. It attends the typhoid state without reference to the cause, and may be present toward the close of life in either acute or chronic disease. It is associated also with inflammatory cerebral disease, and may be present in connection with uræmia and with poisoning by certain drugs, such as alcohol, belladonna, or opium. Nocturnal delirium frequently persists after convalescence has been well established, especially in pneumonia and typhoid fever. Under these circumstances it is doubtless akin to the delirium of inanition. The latter, as its name indicates, is usually associated with acute wasting diseases, either febrile or anæmic. Its onset is sudden, in the early morning, and the patient is found to be extremely weak and collapsed. It may continue from a few hours to 2 days. Brief delirium may follow, and in some instances may replace, an epileptic convulsion. Delirium is also one of the manifestations of hysteria.

III. **Insomnia, and Starting during Sleep.**—Sleeplessness possesses slight diagnostic value. Disturbed sleep is present in the majority of febrile diseases. Prolonged or persistent wakefulness is a prominent symptom in delirium tremens, pneumonia, melancholia, gout, meningitis, and cerebral syphilis, and it may also be caused by the over-free use of tea, coffee, and tobacco.

Sudden twitching or starting of the body during sleep may be due to mental or physical fatigue, especially if the person be already neurasthenic. It is not at all uncommon in connection with dyspepsia or acute indigestion, as well as with delirium tremens, meningitis, and cerebral embolism. In children it may be due to fever, dentition, or worms, and may be indicative of disease of the joints. It is a very unpleasant symptom, occurring just as the patient is falling asleep, when the compensation is broken in valvular disease of the heart. In meningitis and joint diseases it is usually accompanied by a cry. The most frequent causes are dyspepsia, fatigue, fever, and dentition.

SECTION VIII

DISTURBANCES OF CONSCIOUSNESS

INABILITY to cognize impressions, from within or without, which are ordinarily capable of producing physical or mental sensations, constitutes *unconsciousness*, which as a symptom is of much diagnostic importance. It is also, as a symptom, the cause of frequent diagnostic perplexity.

Loss of consciousness may be gradual or sudden, and may present various degrees of completeness. With reference to its completeness, three degrees are clinically recognised: *somnolence*, *stupor*, and *coma*. *Coma vigil* is a fourth acknowledged variety or form of unconsciousness.

Somnolence, or *lethargy*, is the lightest degree of disordered consciousness, and is manifested by a persistent sleepiness, from which, however, the patient can be easily aroused.

Stupor is a more decided, but partial, loss of consciousness, a profound slumber from which it is possible to awaken the patient, but only with great difficulty and by importunate solicitations.

Coma is a condition of insensibility from which it is impossible to arouse the patient. The face is expressionless, the respiration may be stertorous, the mouth is usually open, and the tongue dry.

Coma Vigil.—In coma vigil the patient lies with his eyes open, but absolutely unconscious of his surroundings, and there is usually a muttering delirium, with aimless movements of the hands. It is a condition indicative of extreme peril.

The Diagnostic Significance of Coma

Coma occurring very suddenly is characteristic of apoplexy, catalepsy, and sunstroke. More or less complete, and making its appearance gradually, it may attend any of the fevers and acute infectious diseases, as typhoid, typhus, and pernicious malarial fevers. It is also a symptom of narcotic poisoning, as from alcohol, chloral, opium, ether, chloroform, nitrous-oxide gas, and coal gas. In addition to the poisons of the specific infectious diseases, coma is caused by uræmia, and, late in the disease, by diabetes. Other poisoned states of the blood which may produce it are those of septicæmia, pyæmia, carcinomatous growths, and the very rare acute yellow atrophy of the liver. Asphyxia is attended with drowsiness or perhaps actual coma. Injuries to the head which involve concussion or laceration, and pressure upon the brain, as from a depressed fracture, may produce total unconsciousness. Hysterical coma is not infrequent. The convulsions of epilepsy and infantile eclampsia are followed by a period of unconsciousness. A momentary loss of consciousness may be almost the only symptom of minor epilepsy (*petit mal*). Coma may attend any organic, hemorrhagic, or inflammatory disease of the brain, as in the various forms of cerebral meningitis, in which it makes its appearance late in the disease and is due to pressure from the exudate. It may be associated also with disease of the cranial bones, meningeal hemorrhage or tumour, abscess or tumour of the brain, acute encephalitis, cerebral syphilis, cerebral hemorrhage, embolism or thrombosis, and softening, thrombosis of the cerebral sinuses, general paresis, and multiple sclerosis.

Syncope is a sudden loss of consciousness due to acute anæmia of the brain. A rather sudden coma may occur as the result of severe muscular exertion, independent of sunstroke. Finally, coma, beginning from a few hours to several days before death, may herald the end of many acute and chronic diseases.

Coma vigil may be present in connection with typhoid and typhus fevers, and with the *typhoid state*, especially when it supervenes in septicæmia, pyæmia, and delirium tremens.

The Diagnosis of the Varieties of Coma

In General.—In all cases of more or less sudden unconsciousness the following points are of value:

1. *Skin.*—During extremely warm weather it is important to observe the temperature of the skin. If it is excessively hot and dry, one may infer the presence of sunstroke (insolation).

2. *Head.*—Examine for evidence—bruises, cuts, or depressions—which may point toward injury of the brain.

3. *Face.*—Determine the presence of unilateral facial paralysis (one side of face expressionless and smooth), which is usually associated with hemiplegia and indicates a unilateral brain lesion—e. g., hemorrhage, embolism, thrombosis, or other cause of interference with intracranial function; or hysteria.

4. *Eyes.*—If both pupils are contracted and do not dilate when covered, suspect narcotic poisoning (especially by opium) or a cerebral lesion. If the pupils are unequal (anisocoria), it is pretty certain under such circumstances to be caused by a cerebral lesion. If upon attempting to raise the eyelids there is a quivering resistance and the eyeballs are kept continuously turned upward, hysteria is the probable cause of the coma. In other forms of unconsciousness there is never, or but very rarely, the slightest difficulty in raising the lids, and the eyes are not necessarily upturned.

5. *Mouth and Tongue.*—If the lips or tongue are bitten and there is froth upon the lips, one may correctly suspect epilepsy, although the injuries may have been inflicted accidentally by the teeth when the patient fell unconscious.

6. *Extremities.*—These are to be examined for paralysis, especially hemiplegia, in which the arm and leg of the same side fall limp and helpless when raised and dropped. If the unconsciousness is absolute, all four extremities may be apparently paralyzed, but a greater degree of relaxation may be manifest on one side than on the other. At the very onset of an apoplexy there may be an "initial" rigidity of the paralyzed side. See also 3, preceding.

It should be borne in mind as a diagnostic aid that there are certain comatose conditions which may be preceded or followed by general convulsions. These are: the primary convulsion and subsequent extreme drowsiness caused by the exanthemata and other acute specific infections in children; the coma which follows convulsions due to dentition or digestive disorders in children; the coma sequent to the epileptiform seizures of cerebral syphilis, general paralysis of the insane and, more rarely, of alcoholism; the coma subsequent to the convulsions of epilepsy and hysteria; and the coma following the uræmic convulsion. The coma of sunstroke, cerebral hemorrhage, and some other ailments of less frequency and importance may be preceded by a general convulsion.

In Special.—Certain special varieties of coma (with or without convulsions) are of sufficient frequency and consequence to be grouped and described with enough detail to enable at least a presumptive differential diagnosis to be made. The discrimination is of great importance because of the liability and the danger of confounding one with another, especially the first four which will be considered—viz., coma from opium, alcohol, apoplexy, and uræmia.

1. *Opium Coma.*—The patient is deeply comatose; the face is dusky and cyanotic; the respirations are slow (12 to 4 or even less per minute); the pulse is also infrequent and full until the terminal stage; the pupils are equal, and contracted to pin points. The temperature of the body is normal, and, except toward the end, the skin is dry and moderately warm.

2. *Alcoholic Coma.*—It may or may not be possible to arouse the patient. If he can be awakened, he may protest by words or blows. The face is usually flushed, often somewhat cyanotic, more rarely pallid. The respirations are of normal frequency, deep, and sometimes stertorous. The odour of alcoholic liquor can be detected in the breath; and there is usually the peculiar sour, mawkish smell which results from the disturbing effect of alcohol upon the buccal

and gastric mucosa—the odour of "drunkard's stomach." This odour is of no value as a diagnostic symptom; its absence is of importance as a negation of alcoholism. The pulse is rather rapid, full, and strong, finally becoming small and feeble. The pupils are equal, and either of normal size or dilated. Very commonly the skin is cool and moist, and the bodily temperature below the normal point. Convulsions are infrequent, but there may be some local muscular spasm or twitching.

Two points should be remembered before making a diagnosis of alcoholic coma: one, that it may be closely simulated by apoplexy; the other, that apoplexy may occur in a drunken person and the symptoms of a cerebral lesion be added to those of acute alcoholism. Further, it should be borne in mind that the patient may have had administered by mouth a dose of liquor from the hands of some well-meaning person after the onset of the attack. Consequently, in cases simulating drunkenness, the signs of apoplexy, to be described in the next paragraph, especially paralysis, should be most carefully looked for.

3. *Coma from Apoplexy.*—The coma is usually profound, and the patient can not be aroused. The face is flushed or pale and cyanotic. The respirations are slow, stertorous, and sometimes of the Cheyne-Stokes type. The lips are blown out, and one cheek flaps more than the other during respiration. The pulse, as a rule, is full, strong, and infrequent, and the arteries are hard. The pupils do not react to light, are dilated, and often unequal. There may be conjugate deviation of the head and eyes—i. e., they are turned persistently to one side. One side of the face is usually paralyzed, as indicated by the smoothing out of its wrinkles, the flapping cheek, and the droop of one angle of the mouth. By lifting the limbs and finding those of one side to fall more flaccidly than those of the other side, the presence of hemiplegia may be demonstrated. The skin is dry and the temperature of the body above the normal.

4. *Uræmic Coma.*—Convulsions often initiate an attack of uræmic coma, but the unconsciousness may develop gradually and without spasm. The face may present the swollen pallor of renal disease. The pulse is generally infrequent and is apt to be of high tension. The pupils are equal, either dilated or of normal size. There may be muscular twitchings and rigidity affecting the hands and feet. The temperature may be normal, in severe cases subnormal, and in those attended by several convulsions it may be greatly elevated. The breath and bodily exhalations may have a urinous and ammoniacal odour. In suspected uræmic coma the urine should be promptly examined for albumin and casts, the heart and vessels for hyper-

trophy and sclerosis, the eye grounds for retinitis, and various portions of the body for œdema. It is not to be forgotten that there are occasional instances of uræmic hemiplegia, often transient and without discoverable organic lesions of the brain, which resemble apoplexy so closely that it proves impossible to make a differential diagnosis except at autopsy.

5. *Coma from Epilepsy.*—There is usually little difficulty in the diagnosis of the post-convulsive coma of epilepsy. The history of a convulsion, the bitten tongue, the foam on the lips, and, above all, the brief duration of the gradually lessening unconsciousness, are in the majority of cases sufficient to settle the question. The face is congested, the breathing stertorous, the limbs are relaxed, but there is no hemiplegia.

6. *Hysterical Coma.*—There is something characteristic, although difficult to describe, in the appearance of a patient in the coma of hysteria. The attack may be preceded by laughing, crying, delirium, or convulsive movements; or it may come on without premonition. The face is somewhat flushed, the pulse is usually of normal rapidity, the breathing may be rapid but not stertorous; the pupils are equal, of normal size, and responsive to light; the eyelids resist opening, and the eyeballs are persistently upturned. After a little experience with such cases the facial expression, attitude, and general appearance of the patient make an impression upon the observer difficult to define, but extremely characteristic—as of an unconsciousness, in part real, in part intentional. An irritant, such as ammonia, to the nostrils or continued firm pressure upon the supraorbital nerve at its point of emergence, will at least partly arouse the patient.

In view of the undoubted fact that serious organic disease of the brain may be ushered in by pseudo-hysterical symptoms, it is best to be somewhat chary in announcing a positive diagnosis of hysteria, unless there is a total absence of organic symptoms and the hysterical characteristics of the attack are so marked as to be beyond question.

7. *Syncope.*—Unconsciousness from sudden anæmia of the brain is rarely confounded with other forms of coma. The pallor of the face is absolute, the respirations are shallow and almost imperceptible, the pulse is weak, perhaps absent, and the pupils are widely dilated. The eyes may remain open. If the syncope is due, as it is in the majority of cases, to a temporary weakness of the heart action from emotional causes or the sudden assumption of the erect position by an enfeebled person, consciousness will soon return under appropriate treatment.

It is to be remembered, however, that if there is pallor *plus* cyanosis and some stertor, the attack may be due to serious cardiac disease, or may be one of the slight apoplectic seizures premonitory of a serious cerebral thrombosis.

8. *Diabetic Coma.*—This variety of coma may occur without premonition, may be preceded by syncope and drowsiness, or may begin with vomiting, headache, delirium, and dyspnœa. After coma is established the respirations are either normal or increased in frequency, the pulse normal and full, and the bodily temperature is subnormal. The distinctive symptoms which will enable a recognition of diabetic coma are the sweetish, fruity, or "overripe-apple" odour of the breath, and the discovery of a considerable amount of sugar in the urine.

9. *Coma from Gas Poisoning.*—The cause of this form of unconsciousness is almost invariably obvious from the circumstances under which the patient is found. It results from the inhalation of carbon dioxide (old wells or deep caves), carbon monoxide (illuminating gas, charcoal fire), or hydrogen sulphide (sewers).

10. *Coma from Sunstroke.*—The patient is profoundly unconscious, the face is flushed, the skin pungently hot, the respiration is laboured, deep, and often stertorous, and the pulse frequent and full. The excessively high temperature of the body, the heat of the weather, and the circumstances under which the attack has occurred (e. g., bodily exertion under exposure to the sun), render the diagnosis easy.

SECTION IX

GENERAL CONVULSIONS

A *convulsion* is a series of involuntary contractions, involving mainly the major portion of all the voluntary muscles, and occurring with varying degrees of violence. It is customary to apply the term *spasm* to convulsive contractions of the muscles belonging to particular portions of the body. It is a distinction which can not be accurately drawn, because the same factors are often causative in each, and general convulsions may precede, follow, or alternate with local convulsions or spasms.

Convulsions may be tonic or clonic. A *tonic* convulsion is characterized by a continuous contraction of the affected muscles, which may last for a few seconds, as at the onset of an epileptic paroxysm, or for days, as in tetanus. A *clonic* convulsion is distinguished by rapidly alternating contractions and relaxations of the muscles involved, as in hysteria or in the epileptic convulsion immediately following the primary tonic contraction. The body and limbs in a tonic convulsion are rigid and immovable, contrasting vividly with the agitated and violent motions of the clonic form. A clonic (or epileptiform) convulsion is usually of cerebral origin; the tonic (or tetanic) convulsion is more apt to result from the heightened excitability of the spinal cord. Consciousness may be preserved, as in tetanus, partly lost, as in hysterical convulsions, or totally absent, as in the epileptic convulsion.

The diagnostic associations of general convulsions are with uræmia, puerperal eclampsia, epilepsy, and hysteria; poisoning from alcohol, lead, aconite, prussic acid, and veratrum viride; tetanus, strychnine poisoning, and hydrophobia. In the last three, consciousness is preserved. Convulsions may occur also in connection with profuse hemorrhages and, very seldom, with sunstroke and apoplexy. They appear with much frequency in the later stages of organic cerebral disease, as in infantile hemiplegia; tumour, cyst, or sclerosis of the brain; cerebral syphilis and general paresis, as well as in meningitis, hematoma of the dura mater, and other inflammations of the membranes of the brain and spinal cord.

Convulsions occur in children, especially those under 2 years of age, upon very slight provocation. Digestive disturbances from an unsuitable or overlarge food supply are frequent causes. Irritation from dentition and from intestinal parasites may give rise to convulsions, but these causes are probably not operative as frequently as popular opinion supposes. Convulsions may attend the onset of acute poliomyelitis and the acute infectious diseases, especially scarlet fever, measles, pneumonia, and malaria, a convulsion replacing the chill of the adult. In some infants who are predisposed by a congenitally unstable nervous system, or by the existence of rachitis, eclampsia may initiate any slight febrile attack.

Spasm or localized convulsions, jerking or choreic movements, and tremor are considered in detail in connection with the examination of the nervous system (*q. v.*).

Certain ailments, of which general convulsions constitute the sole, leading, or important symptom, may be here considered as follows:

(1) **Epilepsy.**—The epileptic convulsion is typically clonic. It is in many instances preceded by an *aura*, a peculiar premonitory symptom, usually subjective and sensory, more rarely motor. The most common is epigastric or abdominal distress, with or without palpitation of the heart. Less frequent are subjective sensations of taste, odour, noises, musical sounds, voices, flashes of light or colour; or peculiar mental states of terror or perplexity; or rapid moving or

turning after the manner of a whirling dervish. Immediately succeeding the aura, and in many cases preceded by a scream or outcry, the patient falls unconscious; all the skeletal muscles become tonically contracted, with the legs extended, hands tightly closed, jaw clenched, and head turned forcibly to one side; the muscles of respiration are fixed, and the face in consequence is deeply cyanosed. In less than a minute the clonic movements supervene. The muscles of the face, eyes, eyelids, and jaw work convulsively, and the extremities jerk violently and rhythmically. Foam, blood-stained if the tongue and lips have been bitten, exudes from the mouth; the cyanosis lessens; the bladder and rectum may be emptied unconsciously. After two or three minutes the movements cease and the patient is left in deep coma.

(2) **Hysterical Convulsions.**—In young patients epilepsy and hysteria may bear so close a resemblance that great difficulty may be experienced in the discrimination. The hysterical (or hysteroid) convulsion, however, is often obviously initiated by some form of emotional excitement. It is preceded by "globus," palpitation, tingling, and numbness of both hands or both feet, or all four extremities, and the onset of the attack is frequently gradual. The single outcry at the beginning of the epileptic attack may be replaced by a series of screams during the course of the hysterical paroxysm. The movements of the hysterical convulsion are more purposive and less rhythmical; there may be rigidity, opisthotonus, and attitudinizing; the tongue is rarely bitten or the patient injured; there are no involuntary evacuations; the attack lasts longer than that of epilepsy, and the subsequent coma is not so profound. The hysterical facies is not unimportant evidence.

(3) **Uræmic Convulsions and Puerperal Eclampsia.**—The nature of the epileptoid convulsions in these cases is, as a rule, readily recognised by the urinalysis in both instances: by the history in the first ailment, and the presence of pregnancy or the puerperium in the second.

(4) **Infantile Eclampsia.**—Convulsions in children resemble those of epilepsy, except that the symptoms are usually not quite so severe and fully developed. Such attacks are usually symptomatic of some causative condition, viz., overeating, especially of indigestible food, rachitis, debility from exhausting diarrhœal diseases (hydrencephaloid state); high fever, especially at the onset of the acute specific infections; very seldom dentition, phimosis, and acute middle-ear inflammation; injuries to the brain at birth, infantile hemiplegia, meningitis, and tumour of the brain; rarely of spinal-cord disease. As some disturbance of the digestion is probably the most frequent

cause of a single convulsion, one should always inquire with reference to possible overfeeding, improper food, or constipation.

If a convulsion with high fever and perhaps vomiting occurs suddenly in a previously healthy child, it is proper to suspect and watch for acute meningitis or infantile hemiplegia, scarlet fever, malaria, or lobar pneumonia, although indigestible food may be alone responsible for the attack. It is also to be borne in mind that lobar pneumonia in children may be mistaken for meningitis, as the pulmonary disease may present convulsions, delirium, rigidity or retraction of the neck, and other signs of a meningeal character. In searching for the cause of the eclampsia the urine should be examined if practicable. Rachitis and debility from exhausting disease are not uncommon causes of spasms (usually tonic) affecting mainly the hands, feet, and larynx (carpo-pedal spasm, laryngismus stridulus). Jacksonian or localized convulsions affecting one extremity may be an early symptom of tumour of the brain or infantile hemiplegia. Convulsions recurring at irregular intervals during childhood, without obvious cause, are in all probability true epilepsy.

(5) **Tetanus and Strychnine Poisoning.**—Tetanus and overdoses of strychnine each give rise to convulsions or spasms of the tonic (tetanic or spinal) type. The convulsions due to these two different causes may bear a close resemblance. In each case the muscles are tetanically contracted, the body may be curved and twisted into various postures (emprosthotonus, opisthotonus), and the spasms occur at irregular intervals, with more or less muscular rigidity existing between the seizures. The distinction is to be made by noting that in strychnine poisoning the jaw muscles are the last to be involved (perhaps escaping entirely), there is little if any rigidity between the paroxysms, and there will probably be a history of the ingestion of the poison. On the other hand, in tetanus lockjaw is the earliest well-marked symptom, the muscles remain rigid during the interparoxysmal periods, and there is a history of an injury, especially a punctured or lacerated wound of the hand or foot.

Tetany (*q. v.*) is an entirely different disease, in which the spasm and peculiar position of the hands and feet, particularly of the hands, together with the altogether different history, enable its discrimination from tetanus.

SECTION X

CUTANEOUS SURFACE

THE cutaneous surface is studied in general with reference to its colour, heat and moisture, the presence of a rash or eruption, of cicatrices, swellings, œdema, and varicosities. Abnormal conditions of the joints are also included. It is advisable, in examining the colour of the skin, to inspect also the colour of the mucous membrane of the mouth and the conjunctiva.

I. THE COLOUR OF THE SKIN

The effects of exposure to wind and weather, as seen in labourers, drivers, railway employees, and especially in seafaring men, are sufficiently familiar. The tanned, leathery, fine-wrinkled skin will in such persons hide marked changes of colour unless other skin or mucous surfaces are examined. Habitual pallor is seen in persons living an indoor life, or who sleep in the daytime and work at night. Allowing for the foregoing physiological conditions, the colorations of the skin which occur in disease are as follows:

Pallor.—This may come suddenly, or may begin insidiously and progress so slowly that only by looking back over months or years can the date of its onset be determined. In general it must be due to one or both of two conditions: first, a lessened amount of blood in the cutaneous capillaries, caused either by spasm of the arterioles or defective action of the heart; second, by blood alterations—viz., a decreased number of red cells, an enormous leucocytosis, a deficiency in the amount of hæmoglobin, or loss in the total amount of the blood by hemorrhage.

(1) *Evanescent pallor* may be caused by a temporary weakness of the heart's action, as in syncope, rigours or chills, nausea, the slighter degrees of shock, and the arterial spasm of certain vasomotor neuroses.

(2) *Sudden* and more or less *permanent* paleness is seen for the most part in large and rapid hemorrhages, and in cases where the heart fails abruptly. Leaving traumatism out of consideration, the first diagnostic symptom of internal hemorrhage (*q. v.*) may be a sudden pallor, to be soon followed by associated symptoms and perhaps, depending on its source and within a varying period, by a discharge of blood from an orifice of the body.

(3) *Slow-coming* and *permanent* pallor arises from a gradual diminution in the number of red cells and the amount of hæmoglobin. Such alterations may, in a sense, constitute the disease, as

in maladies due to defects in the hæmatopoietic or blood-making organs, or may be secondary to other chronic diseases, as in tuberculosis. The pallor is often modified by tints which are somewhat characteristic of the causative disease, but in themselves are of little value except as corroborating a diagnosis made by means of other signs and symptoms. A list of the principal diseases attended by slow and permanent pallor, either anæmic or circulatory, is appended:

Cancer (yellow tint).
Chloro-anæmia (yellowish-green tint).
Chronic arsenical poisoning.
Chronic febrile diseases.
Chronic gastro-intestinal disease.
Chronic lead poisoning.
Chronic mercurial poisoning.
Chronic suppurations.
Heart diseases (especially fatty heart, mitral, and aortic stenosis).
Hemorrhages (slight recurring).
Leucæmia.
Malarial cachexia.
Nephritis (waxy pallor).
Pernicious anæmia (lemon-yellow tint).
Pseudo-leucæmia.
Syphilis.
Tuberculosis.

Redness.—Unusual redness of the skin depends upon the overfilling of the cutaneous capillaries—hyperæmia. It may be physiological, as in those of a fresh and florid complexion, in blushing, in the general redness consequent upon a warm bath, friction of the surface, and exercise. Long exposure to heat, cold, and moisture produces a purplish redness of the hands, which may be seen typically in common labourers and washerwomen. The hands are cold, and pressure with the point of the finger leaves a light-coloured spot to which the blood very gradually returns, showing a very sluggish capillary circulation. Nevertheless, this condition may coexist with perfect health.

Pathological redness may be either diffused or localized.

(1) *Diffused redness* is seen in many fevers, especially in children, because of their particularly free capillary circulation. Full doses of belladonna or hyoscyamus will produce general hyperæmia.

(2) *Localized redness* is sometimes characteristic, as in the bilateral flush upon the cheeks which accompanies excitement or fever in the phthisical, the redness of one cheek in acute pneumonia, and the unilateral redness of the face which attends some attacks of migraine. In certain cases of anæmia (*chlorosis rubra*) the flushing of the face is so marked that at first the existence of a deficiency of hæmoglobin may be quite unsuspected. The dusky redness of the face in chronic alcoholics is familiar, and in obstructed portal circulation there may be limited red areas on the nose and cheeks.

Cyanosis.—A blue or purple tint of the skin, dependent upon the presence of dark venous, imperfectly oxygenated blood in the capillaries, is seen in many diseases and conditions. It is best observed in the finger nails, lips, and mucous membranes, because of the thinness and translucency of their epithelial covering. If the cyanosis becomes decided, the whole cutaneous surface will assume a dusky, leaden tint. The causes of cyanosis may be enumerated under the following heads. It will be evident that more than one cause may be present in a given case.

(1) *Conditions which Hinder the Admission of Air to the Lungs.*—Angina Ludovici, phlegmonous inflammation of the floor of the mouth; glossitis; pharyngitis, if acute and severe, and retropharyngeal abscess may cause cyanosis by producing œdema of the glottis; laryngismus stridulus; spasmodic croup; laryngitis (acute or chronic); tuberculous or syphilitic inflammation of larynx; diphtheria of larynx (membranous croup), trachea, and bronchi; traumatism of larynx and pharynx, old or recent; foreign bodies in the upper air passages; paralysis of dilators of larynx; compression of trachea or bronchi from aortic aneurism, goitre, enlarged bronchial glands, or tumours of mediastinum; spasmodic asthma; fibrinous or plastic bronchitis; bulbar paralysis and peripheral neuritis, by causing paralysis of the muscles of respiration; peritonitis, by causing paralysis of the diaphragm; epilepsy, tetanus, and strychnine poisoning by causing respiratory spasm; pleurisy, pneumonia, intercostal neuralgia, diaphragmatic pleurisy, pleurodynia, and peritonitis, the pain which attends these diseases preventing the full and free action of the respiratory muscles.

(2) *Conditions which Lessen the Working Breathing Surface of the Lungs*, either by effusion or exudation into the air cells, by alterations in the form and structure of the air cells, or by direct compression from outside, as in pneumonia (all varieties), collapse of lungs, pulmonary œdema, pulmonary tuberculosis, emphysema, pleurisy with effusion, hydrothorax, pneumothorax, pericarditis (with large effusion), thoracic tumours, and large abdominal effusions and tympanites, by pressing up the diaphragm.

(3) *Conditions which Interfere with the Pulmonary or Systemic Circulation.*—The interference may be with one or both, causing a general cyanosis. There may be also a limited cyanosis due to localized obstruction of the venous trunks of an extremity or a portion of the body. The circulation through the capillaries of the lungs is necessarily obstructed by any of the diseases or conditions previously mentioned (e. g., pleurisy with effusion), which act in such a manner as to compress the lung, because the capillaries also are compressed

and their calibre lessened to a greater or less extent. In emphysema of a high grade and in pulmonary tuberculosis many capillaries are obliterated. General cyanosis is also produced by the large class of valvular and degenerative diseases of the heart when compensation fails and the heart muscle loses power, especially when the right ventricle becomes dilated and is unable to clear itself. It appears in pericarditis with large effusion, or when mediastinal tumours press upon the superior or inferior vena cavæ at their entrances to the right auricle.

Local cyanosis is caused by pressure upon large veins or venous trunks, thus damming the venous blood back upon the part or area drained by them—viz., thrombosis of the femoral or brachial, compression of the inferior vena cava by ascites, or pressure upon any vein by inflammatory swellings or neoplasms.

(4) *Cyanosis may be Produced by Certain Drugs or Poisons.*—Examples of this condition occur with overdoses of the coal-tar preparations (antipyrine, acetanilide), with drugs which depress the respiratory centres (opium and its preparations), or with those which cause chemical changes in the blood (hydrocyanic acid, chloride of calcium).

Jaundice or Icterus.—This is a yellowish coloration of the skin, mucous membranes, and fluids of the body, varying in intensity from a light lemon-yellow to a brownish-yellow or saffron tint, and caused by the presence of bile pigment in the blood. In exceptional cases it is a dark-brown or greenish-black, the so-called "black jaundice." It is observed first and best in the conjunctiva and the oral mucous membrane, and, when slight, in the eyes and mouth only. In order to demonstrate it in the mucous membranes, it may be necessary to render an area of the membrane anæmic, by pressure with the fingers or by a glass mounting slide, which allows the yellow ground tint to become visible. It may be readily seen upon the under surface of the tongue and the anterior portion of the floor of the mouth.

Sources of error are the yellowish tint of chloro-anæmia, malignant tumour, malarial cachexia, renal cirrhosis, lead poisoning, and the temporary icterus of the newborn; but mistakes may be avoided by noting that the conjunctiva retains a normal colour. In examining the eye, the presence of yellow subconjunctival fat will not deceive the careful observer.

In studying jaundice we may consider it from two aspects—first, its cause or origin; second, its severity.

A. JAUNDICE, WITH REFERENCE TO ITS ORIGIN, may be either *obstructive* or *toxæmic*.

(1) **Obstructive Jaundice.**—In this, as its name indicates, there is some hindrance to the passage of bile from the liver into the intestine, and in consequence it is absorbed into the rootlets of the hepatic vein and carried into the general circulation.

In addition to the yellowish skin and mucous membranes previously described, the *symptoms* of the obstructive form are as follows: There is a yellow coloration of the sweat (rarely of the saliva, tears, and milk) and of the sputum if pneumonia coexists. The urine is more or less deeply coloured and may resemble dark beer. As no bile enters the intestine, the stools are pasty, fetid, and of a drab or clay colour. The clay-coloured fæces constitute an important differential point between obstructive and toxæmic jaundice. Constipation is usual, but diarrhœa may be caused by excessive putrefaction in the intestine. Distressing cutaneous itching or pruritus is common in the more chronic cases. Furuncles, urticaria, xanthelasma, and other diseases of the skin may ensue. In chronic cases there may be red patches up to an inch in diameter, due to dilated vessels, on the skin and mucous membranes, and the blood coagulates very slowly, giving rise to obstinate and sometimes fatal hemorrhage following injury or operation. Large ecchymoses, purpuric spots, and, although rarely, spontaneous bleeding from the mucous membranes may be seen in chronic cases. The pulse is usually slow (40 to 50), especially in catarrhal jaundice; and the respiration slow, 10 or even less per minute. The patient is apt to be depressed and melancholy. In the chronic and fatal cases the patient may pass into the typhoid state (*q. v.*), or there may be a sudden onset of convulsions, delirium, or coma—usually, in either case, followed by death.

The *causes* of the obstructive form are: Gastro-duodenal catarrh; catarrh of the bile ducts, common, large, or small, with swelling of the lining membrane; lodgment of gallstones or roundworms in the common duct; pressure on or closure of the duct by tumour of the liver, stomach, kidney, omentum, and especially of the pancreas; and new growths or cicatricial tissue affecting the duct itself so as to produce stricture or obliteration of its lumen. In rare instances the pressure of a pregnant uterus, an abdominal aneurism, or a large fæcal accumulation may be responsible for the obstruction.

(2) **Toxæmic Jaundice.**—The non-obstructive form of jaundice depends upon the presence in the circulation of various poisons which destroy the red cells of the blood, or more rarely the hepatic cells as well.

The *symptoms* of toxic jaundice may be very slight. The yellow colour may be slight and is rarely so intense as in obstructive jaundice. As there is no hindrance to the entrance of bile into the intestines, the fæces not only retain their normal colour, but because of an increased flow of bile (polycholia, due to the destruction of the red blood cells) may be darker than usual. The urine may be deepened in colour, but bile pigment is absent or small in quantity, and the coloration is due to an increase in the amount of the normal pigments of the urine. If the toxæmia is severe, there may be grave general symptoms—coffee-ground vomiting, hemorrhages into the skin and mucous membranes, delirium, convulsions, and high temperature. Toxæmic jaundice is never chronic, death or recovery taking place within a comparatively short period.

The *causes* of toxæmic jaundice are the toxines or venoms produced by living organisms, or poisoning by certain chemical compounds. Consequently this variety of jaundice is met with as a consequence of the acute infectious diseases—viz., epidemic influenza, malaria (intermittent and remittent), pneumonia, typhoid fever, typhus fever, scarlet fever, yellow fever, relapsing fever, pyæmia, ulcerative endocarditis, acute yellow atrophy of the liver, and Weil's disease; also poisoning by snake venom, antimony, arsenic, chloral hydrate, chloroform, copper, ether, mercury, phosphorus (especially), potassium chlorate, and toluylendiamine.

B. JAUNDICE, WITH REFERENCE TO ITS SEVERITY, may be either mild (*icterus simplex*) or severe (*icterus gravis*). It is a matter of much clinical importance to determine in which of these categories an individual case belongs.

(1) **Mild Jaundice; Symptoms and Causes.**—*Icterus simplex* is frequently ushered in by nausea or vomiting, which may last only for a day or two, with malaise, slight fever, some headache, clay-coloured stools, slow pulse, and slight or no itching of the skin, although the latter may be much stained. This mild type almost always arises from a catarrhal inflammation of the common duct, the swollen mucosa occluding the duct and preventing the bile from entering the intestine. It is usually an extension of a gastro-duodenitis, and recovery is the rule within a few weeks. A mild jaundice may or may not be present in the later stages of certain diseases of the liver—viz., cirrhosis, passive congestion, carcinoma, syphilis, amyloid and fatty liver, and echinococcus.

(2) **Severe Jaundice; Symptoms and Causes.**—*Icterus gravis* may be acute, subacute, or chronic. The symptoms are of an ominous character as compared with the mild form. The jaundice is associated with delirium, vomiting, hemorrhages, and high fever; or the dry tongue, mental confusion, low continued fever, and profound adynamia of the typhoid state. This severe type of icterus may be due to

toxæmic causes (*q. v.*), or, if chronic and attended with emaciation, to some of the conditions which produce more or less permanent and perhaps irremediable obstruction of the bile ducts—e. g., an impacted gallstone. Jaundice caused by gallstones is almost invariably preceded by pain; when due to other causes (e. g., cancer of the head of pancreas), pain is absent. Jaundice with a marked cachexia may indicate carcinoma of the liver; with chills and fever, hepatic abscess; with ascites, a cirrhotic liver or a chronic peritonitis.

Bronzing.—Affecting the general cutaneous surface, this is seen in Addison's disease (*q. v.*). It consists of a brown or brownish-black discoloration, not dispelled by pressure, most marked in the face and hands and in the portions of the body which normally contain pigment. It occurs also in the mucous membrane of the mouth and vagina as discrete brown spots of varying size. The nails, cornea, and conjunctiva usually escape. The bronzing is due to the deposition of pigment in the deeper layers of the skin and mucous membranes.

Other conditions may present an abnormal amount of pigmentation, either general or local. Patchy yellow spots may appear, usually but not always, on the forehead and face, in persons who suffer from habitual constipation or chronic "biliousness"; dilatation or chronic ulcer of the stomach; pregnancy with uterine diseases; cancer or tuberculosis of the abdominal viscera or the peritoneum; diseases of the liver, especially cirrhosis and the chronic congestion due to cardiac lesions; exophthalmic goitre; severe acne, tinea versicolor, and following syphilitic eruptions. General and deep pigmentation, from dirt and lice, may be seen in tramps; also, but rarely, in scleroderma and melanotic cancer.

Gray Skin.—This has been observed after the long-continued therapeutic ingestion of silver nitrate. It is occasioned by the presence of minute deposits of the albuminate of the metal in the skin.

II. THE HEAT OF THE SKIN

This may be estimated roughly by placing the hand upon the skin, or accurately by the use of a surface thermometer. The temperature of the skin, however, is not an index of the internal temperature, because a high degree of fever may coexist with marked coldness of the surface, as in the cold stage of intermittent malarial fever.

(*a*) *General coldness* of the surface is usually associated with a poor capillary circulation, the blood returning slowly to a spot which has been rendered anæmic by pressure. It occurs in all chills and rigours, in many forms of general cyanosis, and in all afebrile diseases attended by a weak or failing heart. (*b*) *General abnormal heat* of the surface may and usually does exist in all fevers and diseases attended with decided febrile temperatures, but, as stated, this is by no means always the case.

(*c*) *Local coldness* of the surface may be due to vasomotor spasm, arterial or venous thrombosis, or other obstruction to the circulation in a localized area; (*d*) *local heat*, to inflammations or new growths under that portion of the skin which exhibits the increased temperature.

III. THE MOISTURE OF THE SKIN

The amount of moisture may be greatly increased—*hyperidrosis;* or entirely absent—*anidrosis.*

(*a*) *Hyperidrosis.*—More or less profuse sweating is not incompatible with fever, as in acute rheumatism and other diseases, but occurs more frequently when the temperature is normal or subnormal. In general, hyperidrosis is attendant upon debility (as in convalescence), great weakness (as in collapse), dyspnœa, infectious and septic conditions, tuberculosis (night sweats), severe pain, and the use of diaphoretics.

Partial or localized sweating is an occasional event in connection with certain ailments. Sweating of the hands or the feet attends some conditions of general debility, or may be a constitutional peculiarity. Sweating of the head is seen in rachitis; unilateral or one-sided sweating of the head or face, in migraine, neuralgia, and other affections of the nervous system, suppurative parotitis, and pressure on the sympathetic by a thoracic aneurism; unilateral sweating of the body (hemidrosis), exceptionally in hemiplegia. Partial sweating is usually caused by deranged innervation of the vasomotor nerves.

(*b*) *Anidrosis.*—Diminution or absence of perspiration is observed in many febrile diseases, especially if the temperature is high and prolonged. It accompanies diseases in which there is a profuse discharge of fluid from the bowels, kidneys, or stomach. If the skin is stretched or altered in structure so that the cutaneous circulation is hindered, as in general dropsy or anasarca, or in myxœdema, anidrosis exists as a direct result.

(*c*) *Alterations in Character.*—In rare instances alterations in the composition or colour of the perspiration are noted. In uridrosis, occurring with diseases in which the action of the kidneys has been impaired, the sweat has a urinous odour and deposits white scales or crystals of urinary solids upon the skin. Yellow sweat, from the biliary pigments, may be present in severe jaundice. Blue, brown, yellow, or red sweat (*chromidrosis*) has been observed in hysteria.

Very rarely bloody sweat (*hæmatidrosis*), a capillary hemorrhage into the sudoriparous glands of nervous origin, or a species of vicarious menstruation (*menidrosis*), occurs.

In the consideration of sweating as a symptom, its normal increase, especially in warm weather, after exercise, hot baths, hot drinks, and strong mental emotion, should not be forgotten.

IV. RASH OR ERUPTION

There are certain morbid appearances or lesions of the skin which are of importance, because of the fact that they accompany, or indeed may be a symptom of, some disease or condition affecting the body as a whole.

The cutaneous lesions possessing general diagnostic significance are as follows:

Cutaneous or Subcutaneous Hemorrhages.—These vary in size from a mere point to 3 or more inches in diameter, and occur most abundantly upon the lower extremities. Small hemorrhagic spots (petechiæ) are frequently found in the hair follicles. Larger hemorrhages (ecchymoses) are diffuse. If recent, their colour is dark red, but as absorption progresses this tint alters to a reddish brown or dark yellow. Petechial, ecchymotic, and pigmented points or areas may be readily distinguished from hyperæmic or inflammatory redness by pressure made with the finger, or better with a glass slide. Under pressure a hemorrhagic spot becomes more obvious because of the surrounding anæmia, while the redness of hyperæmia or inflammation will vanish.

The diagnostic value of hemorrhages into or beneath the skin depends entirely upon the symptom group in which they are found. They may be infarctions, dependent upon the lodgment of septic emboli in the smaller arteries, as in pyæmia; a consequence of alterations in the blood, as in pernicious anæmia; an accompaniment of infectious diseases, as in typhus fever; a result of the ingestion of certain drugs, as in mercurial poisoning; an effect of traumatism, as in contusions; or of mechanical obstruction to the return flow through the veins, local or general, as in cyanosis; and, finally, of neurotic or unknown origin, as in locomotor ataxia and the various forms of arthritic or hemorrhagic purpura. In all cases there must exist either alterations in the composition of the blood which will allow it to pass through the uninjured vessel walls, or histological changes in the vessel walls due to traumatic, neurotic, or other pathological causes, which render them abnormally permeable.

The most important diagnostic associations are with fever, as in the infectious or septic diseases; with fever and joint pains, as in peliosis rheumatica; and with hemorrhages from the nose, stomach, intestines, and other mucous surfaces, as in purpura hæmorrhagica.

The diseases and conditions in which petechiæ and ecchymoses may occur, and the poisons which may produce them, are as follows:

Acute exudative erythema.
Acute yellow atrophy of liver.
Anæmias (especially pernicious).
Cancer (especially later stages, of stomach and liver).
Cerebro-spinal meningitis (epidemic variety).
Convalescence from fever (in the legs).
Cyanosis (all forms).
Diphtheria.
Epilepsy (from venous stasis).
Erythema nodosum.
Flea-bites (in debilitated persons).
Hæmophilia.
Henoch's purpura.
Hepatic cirrhosis (at a late stage).
Jaundice (in severe forms).
Measles (if severe).
Myelitis (acute and transverse).
Old age (in the extremities).
Peliosis rheumatica (Schönlein's disease).
Pertussis (from venous stasis).
Purpura hæmorrhagica.
Purpura simplex.
Pyæmia.
Renal cirrhosis (at a late stage).
Sarcoma (of skin and bones).
Scarlatina.
Scurvy.
Septicæmia.
Snake venom (poisoning by).
Stigmata (bleeding points in hysteria).
Tuberculosis (with extreme debility).
Typhoid fever.
Typhus fever.
Ulcerative (malignant) endocarditis.
Variola.
Yellow fever.
Belladonna (poisoning or idiosyncrasy).
Copaiba (poisoning or idiosyncrasy).
Ergot (poisoning or idiosyncrasy).
Mercury (poisoning or idiosyncrasy).
Phosphorus (poisoning or idiosyncrasy).
Potassium iodide (poisoning or idiosyncrasy).
Quinine (poisoning or idiosyncrasy).

Herpes Facialis.—These are small vesicles ("cold sores") containing a clear fluid, grouped upon a reddened and slightly elevated base. The fluid becomes puriform, and in a few days the lesion dries and scales off. They are not attended by pain, thus differing from herpes zoster. They form most frequently upon the lips (*herpes labialis*), also upon the nose, cheeks, or ear, and may appear in the mouth. Herpes facialis is associated with acute catarrh of the respiratory passages; ephemeral fever; pneumonia, in which it is of some diagnostic value; cerebro-spinal fever, in which it may be quite extensive; and with the rapidly rising temperatures of intermittent fever and pyæmia.

Sudamina.—These consist of small, clear vesicles, appearing in great numbers upon all parts of the body, but especially upon the trunk. They give a sensation of roughness to the hand as it is passed over the skin, and with a good light the minute pearly vesicles may be seen. They are formed after a prolonged period of anidrosis when the sweat glands begin to act. The dry epidermis obstructs the flow from the sweat tubules, each vesicle corresponding to the opening of a sudoriparous gland. Their diagnostic value is nil.

Erythematous or Inflammatory Eruptions.—Erythema is an inflammatory hyperæmia of the skin, either simple or exudative; and, if associated with or replaced by more active inflammatory processes, its varieties constitute a class of cutaneous lesions which are of great value in medical diagnosis, because they are essential events or frequent accompaniments of many serious and important, usually febrile and infectious, diseases. There are certain fevers in which the skin lesions are characteristic (eruptive fevers or exanthemata). The diseases accompanied by more or less diffused and characteristic eruptions are as follows:

- Exanthemata
  - Measles.
  - Rubella.
  - Scarlatina.
  - Varicella.
  - Variola.
- Cerebro-spinal fever.
- Dengue.
- Glanders (acute).
- Erysipelas.
- Influenza (rare).
- Miliary fever (sweating sickness).
- Pyæmia.
- Relapsing fever.
- Septicæmia.
- Syphilis.
- Typhoid fever.
- Typhus fever.
- Ulcerative endocarditis.

Drug Eruptions.—The possible occurrence of erythematous or other eruptions as a result of the ingestion of overdoses of certain medicinal substances, or of individual idiosyncrasy, must be borne in mind, as otherwise a mistaken diagnosis may readily be made. The drugs which may cause puzzling rashes or eruptions are: Antipyrine, arnica, arsenic, atropine, belladonna, cannabis indica, capsicum, carbolic acid, chloral, copaiba, copper, cubebs, croton oil, digitalis, iodoform, lead, mercury, morphine, opium, potassium bromide, potassium iodide, quinine, salicylates, salicylic acid, santonin, silver, sulphur, tar, and tartar emetic.

Roseola.—Fugitive roseolous rashes not infrequently initiate the eruptive fevers, antedating the true exanthem, and may thus cause confusion. The diseases which may be preceded by evanescent rosy rashes are cholera, diphtheria, malaria, measles, scarlatina, typhoid fever, typhus fever, and variola. The roseola may be mistaken for a beginning measles, rubella, or scarlet fever. It may also follow childbed and surgical operations.

Urticaria.—Nettle rash may exist as an accompaniment or result of certain diseases, and the ingestion of certain poisons and articles of food. It is largely of neurotic pathogeny. It occurs in connection with the following diseases, drugs, and foods: Cerebro-spinal fever, dengue, gastro-intestinal disorders (especially), hydatid cysts (after tapping), malaria (especially in children), menstruation (disorders of), mental emotion, neurotic œdema, parasites (intestinal), pulmonary diseases (bronchial mucous membranes), purpura, rheumatism, typhoid fever, variola, antipyrine, quinine, buckwheat cakes, mineral waters (in excess), mushrooms, oatmeal, pastry, pork, shellfish, and strawberries (especially).

Furuncles (boils) and carbuncles are apt to occur in connection with diabetes (*q. v.*), and repeated attacks demand a careful examination of the urine.

V. CICATRICES OR SCARS

Whether old or recent, scars may possess considerable diagnostic value as indicative of previous diseases or injuries, as follows:

(*a*) Linear scars, striæ, or lineæ albicantes, occur from overstretching of the skin and consequent separation and atrophy of its fibres, in obesity, œdema, pregnancy, and large abdominal tumours; are occasionally post-febrile (typhoid, scarlet fever); or idiopathic.

(*b*) Small circular pits or depressions, especially upon the face, are significant of varicella or variola.

(*c*) Small scars upon the face may result from acne; upon any part of the body, from furuncles or carbuncles, the latter especially upon the nape of the neck.

(*d*) Scars of irregular shape, usually depressed and adherent, are due to tuberculous or scrofulous disease of the glands, and are seen significantly in the location of the cervical, axillary, and inguinal glands. They may also be a consequence of tuberculous disease of the bones. Lupus of tuberculous origin leaves large flat scars.

(*e*) The scars of syphilitic ulceration are large and nearly circular. Non-traumatic scars, if single, upon the forehead or the legs, in a person below middle age, are usually specific.

(*f*) Scars upon the head or spine or over important peripheral nerves may throw light upon the cause of cerebral symptoms, or disease of the cord and spinal nerves. The scars which may result from injuries sustained during convulsions, epileptic or other, should be borne in mind.

(*g*) Scars of little importance are the contracted large or small cicatrices from burns, the symmetrical linear scars from wet-cupping, and the multiple minute scars of pustulation from the external application of croton oil and tartar-emetic ointment.

VI. DROPSY, ŒDEMA, ANASARCA

Dropsy is the generic term indicating an accumulation of watery fluid in one or more of the serous cavities, or a diffusion of such fluid through the areolar tissue of the body or its organs, or a combination of these conditions. *Œdema* is the effusion of watery fluid into the tissue of a part. *Anasarca* is a subcutaneous œdema diffused over the body at large—general œdema.

(1) **Dropsy of Cavities.**—Dropsies involving the cavities of the body have received particular names. Dropsy of the peritoneal cavity is designated as ascites or hydroperitoneum; joints, hydrarthrosis; brain, hydrocephalus; pleural cavity, hydrothorax; Fallopian tube, hydrosalpinx; pericardium, hydropericardium; pelvis of the kidney, hydronephrosis; and adnexa of the testicle, hydrocele. Dropsies of cavities (*q. v.*) are considered elsewhere.

(2) **Recognition of Œdema.**—The existence of œdema is in most cases readily perceived. There is painless swelling, the skin is pale, smooth, and shining, and if pressure is made with the point of the finger, especially over a bony surface (tibia, malleolus), pitting will occur, and an appreciable time will elapse before the depressed skin regains its former level. The part is apt to be unduly cool, and a serous fluid will ooze out from a needle puncture. Œdema is to be discriminated from subcutaneous emphysema by the fine crackling produced by pressure in the latter condition; from the thickened skin due to infiltration of mucin in myxœdema (*q. v.*) by the fact that in the latter the swelling is firm, and does not pit; from phlegmonous inflammations, by the lack of pain and redness; from localized overgrowths of connective tissue, which are hard, and do not pit on pressure; and from scleroderma.

(3) **Pathology of Œdema.**—In general, œdema is directly due to a disturbance of the relation between the amount of fluid which transudes from the capillaries and that which is absorbed and carried away by the lymphatics. If the lymphatics are obstructed, or if from any cause the capillaries become abnormally permeable and allow more fluid to escape than can be removed, the excess of fluid will accumulate in the connective-tissue spaces and lymph radicles. The character of the minute changes in the vessel walls which allow free transudation of serum is still unsettled.

(4) **Causes of Œdema.**—The causes and varieties of dropsy may be classed as follows: (*a*) Venous obstruction; (*b*) toxæmic or hydræ-

mic conditions of the blood; (*c*) effect of inflammation upon the neighbouring circulation; (*d*) vasomotor or other causes belonging to the nervous system; (*e*) lymphatic obstruction; and (*f*) idiopathic or essential œdema, the nature of which is as yet undiscovered.

Venous Obstruction.—The diseases which may be attended by general œdema are those which tend to prevent the return flow of blood to the right side of the heart. Here belongs the rather characteristic œdema of cardiac origin occurring when the heart muscle fails from valvular defects or other causes. Cardiac œdema is at first localized and makes its appearance primarily in the feet, whence it may extend upward. It is most marked in the lower extremities after standing or walking during the day, and lessens, or in slight cases disappears, after a night's recumbency. When extreme, it may involve the entire body, the scrotum or labia majora being enormously swollen, and effusion taking place into the closed cavities of the chest and abdomen. Under such conditions cyanosis is usually present, so that the list of diseases causing cyanosis by venous obstruction (p. 85) will serve also for those which produce œdema by venous obstruction. *Local œdema* may be caused by thrombosis of or pressure upon a venous trunk.

Toxæmic œdema, due directly or indirectly to poisons circulating in the blood, has its principal exemplification in renal dropsy. In marked cases it is universal, affecting the entire body to a greater extent than in any other disease. Characteristically it begins first in the face, and is especially obvious around and under the eyes, because of the large amount of loose areolar tissue in this locality, thus giving a puffy and swollen aspect to the countenance. It is greatest in the morning, after hours of recumbency, and lessens during the day if the patient sits or stands. In aggravated cases the entire surface of the body will pit on pressure. It occurs particularly in the more acute forms of nephritis, such as those which constitute a sequel of scarlatina or a complication of pregnancy.

Hydræmic œdema, caused by impoverished blood, is observed in all the anæmias, usually a slight or moderate œdema of the feet and ankles after standing or walking. The œdema of the feet and ankles which may appear toward the end of all wasting diseases, and in cachexial conditions, as well as in the recent convalescent on beginning to sit or walk, belongs to this category.

Collateral Œdema.—This appears to a greater or less extent in the neighbourhood of localized, usually suppurative, inflammations, and is caused partly by obstruction of the lymphatics, partly by overdistention of the capillaries resulting from the afflux of blood to the point of inflammation. This form of œdema may be of considerable significance to the internalist, as in the œdema of the thorax indicative of empyema, or of the right hypochondrium in abscess of the liver.

(*d*) *Œdema of Nervous Origin.*—This is seen in a most striking form as angioneurotic œdema (*q. v.*), a singular disease in which œdematous swellings appear and disappear at brief intervals upon the face or extremities. Peripheral multiple neuritis and beri-beri are other examples of nervous disease associated with œdema, but in these the œdema is general. The "blue œdema" of Charcot is hard, bluish, the temperature of the part is lowered, and it is associated with sensory or motor disturbances of hysterical origin.

(*e*) *Lymphœdema.*—This is due to a transudation of lymph through the walls of the lymphatic vessels, or to a distention of the lymph spaces from mechanical obstruction. It is usually localized or confined to a single limb. It has for its cause laceration of a lymphatic trunk, or occlusion of such a vessel by external pressure or internal obstruction, e. g., *Filaria sanguinis hominis*, of which macromelia and elephantiasis are results. General lymphœdema sometimes occurs in lymphadenoma or Hodgkin's disease. This form of œdema differs from hæmic œdema in that the œdematous tissues are much harder, inflexible and brawny, and lymph oozes from the cut surface.

(*f*) Œdema *not due to a discoverable morbid condition* is not infrequent. The "probationer's feet" of the training school, the swelling of the feet and ankles after long marches or pedestrian trips, and the so-called essential œdemas of children belong under this heading.

(5) **Topographical Occurrence of Œdema.**—The following clinical classification of œdema is useful. Two or more diseases or conditions, each of which is attended by œdema, may coexist—e. g., cardiac disease, hepatic cirrhosis, and anæmia:

A. *General Œdema or Anasarca.*

1. Beginning at the feet and extending upward—cardiac weakness or disease.
2. Beginning in the face and extending downward—renal disease.
3. Attends beri-beri and may attend multiple peripheral neuritis.
4. Attends trichinosis, first over affected muscles, then becoming general. Differs from previous forms by the absence of swelling in scrotum and labia.
5. Lymphœdema in Hodgkin's disease.

B. *Œdema of Upper Half of Body.*
  1. In renal dropsy, early stage.
  2. Of arms, head, and neck, in thoracic aneurism, large double hydrothorax, and mediastinal tumour pressing on superior vena cava above the entrance of azygos veins.
  3. Of arms, head, neck, and thorax, when point of pressure is below azygos veins.
  4. Sudden or acute œdema, as in 3, is due only to the very rare rupture of an aneurism into the superior vena cava.
  5. Œdema of one arm is caused by the pressure of enlarged lymphatic glands or tumours upon axillary or subclavian vein, or thrombosis of the vein.

C. *Œdema of Lower Half of Body.*
  1. Cardiac dropsy, early stage.
  2. With ascites in hepatic cirrhosis.
  3. Pressure upon inferior vena cava by abdominal tumours, enlarged liver, spleen, pancreas, or mesenteric glands.
  4. Chronic malarial poisoning with enlarged liver and spleen.
  5. Œdema, usually moderate in amount, with anæmias, cachexiæ, wasting diseases, long-continued slight hemorrhages.
  6. Long standing or walking, first rising in convalescence, sometimes from no ascertainable cause.
  7. Œdema of one leg, from thrombosis of femoral vein, paralysis, pressure on vein by tumour of groin or abdomen.
  8. Lymphœdema.

D. *Circumscribed, usually Single, Œdematous Swellings* (*Collateral Œdema attending Local Inflammations*).
  1. Over præcordial space in purulent pericarditis.
  2. Over affected side in empyema.
  3. Over mastoid process in inflammation of mastoid cells.
  4. Over parotid gland in mumps or parotid suppuration.
  5. Over deep-seated muscular abscesses, especially in typhoid fever.
  6. Over right hypochondriac region in hepatic abscess.
  7. Over region of appendix in some cases of appendicitis.
  8. Over one posterior lumbar region in perinephritic abscess.
  9. Associated with subcutaneous infection in any part of the body.

E. *Circumscribed Multiple Œdematous Swellings* (*usually acute, dispersed, and more or less transient*).
  1. Angioneurotic œdema.
  2. Purpuric œdema.
  3. Giant urticaria.

VII. CONDITION OF THE VEINS

Unusual distention or overfilling of the surface veins, and perhaps of the jugular veins, may be observed. Abnormal venous distention may be general or local, and is frequently preceded, accompanied, or followed by œdema and cyanosis. Œdema, if marked, may hide the overfilling of the veins.

The diagnostic indications of these phenomena are as follows:

General Venous Distention.—Caused by all conditions which hinder the return flow of venous blood as a whole, thus embracing many of the cardiac and pulmonary lesions which are responsible for the production of cyanosis (*q. v.*) and general œdema (*q. v.*), particularly when the right ventricle is failing, or when mitral and tricuspid valvular defects are present. Asthma and emphysema lead to hyperdistention by interfering with the pulmonary circulation, and a rare cause is pressure upon both venæ cavæ by mediastinal growths. It is seen acutely in the paroxysms of pertussis, and in general convulsions.

Localized Venous Distention.—Any condition which hinders the return flow through a venous trunk of appreciable size will cause a localized overfilling of the smaller veins which drain through it, as well as of the veins which communicate with it and its branches, and constitute a collateral circulation. The significant localized distentions are:

1. Of jugular veins alone, with or without pulsation: pressure by mediastinal tumour or thoracic aneurism.
2. Of veins of one arm: thrombosis of, or pressure on, axillary vein.
3. Of veins of one leg: thrombosis of, or pressure on, femoral vein.
4. Of veins of both legs: thrombosis of, or pressure on, femoral veins or inferior vena cava by abdominal or pelvic tumours, by ascites, or other pressure-producing disease.
5. Of superficial veins of skull between ear and vertex: thrombosis of longitudinal sinus.
6. Of single small veins over sternum: mediastinal tumour.
7. Of superficial abdominal veins (collateral circulation): hepatic cirrhosis, ascites, enlarged spleen, or other abdominal tumours or thrombosis causing obstruction to the portal circulation.

Venous Pulsation (*q. v.*) is considered elsewhere.

VIII. EMPHYSEMA OF THE SKIN

This consists in the presence of air or gases in the subcutaneous cellular tissue. At first glance the resulting swelling has the appearance of œdema, but it yields lightly and readily to pressure, and does not pit. The decisive test is the fine crepitation or crackling which is perceived by the palpating finger, resembling that produced by pinching an inflated normal lung. The swelling may be very considerable, and, like œdema, it is most marked where the subcutaneous cellular tissue is most abundant and loosely attached. It is especially noticeable when occurring over the neck and upper part of the thorax, owing to the obliteration of the normal depressions. It is an infrequent finding, and when it occurs is usually confined to certain localities, but very rarely may be diffused over the greater part of the body. The source of the air or gas is either from without, through a wound; from within, by rupture, traumatic or otherwise, of an air or gas-containing organ; or from cellular tissue infected by gas-producing micro-organisms.

The topographical occurrence of subcutaneous emphysema, and the particular lesions causing it, exclusive of infections, are as follows:

Face and Neck.—Wounds, perhaps of insignificant size, of the neck, breast, and lower part of the face, especially those involving the mucous membrane of the mouth. Perforation or rupture of the œsophagus caused by traumatism, ulceration, or cancer, the air passing to the external surface by way of the mediastinum.

Neck and Thorax.—Rupture of larynx or trachea by ulceration or traumatism, allowing air to enter the tissues. Cavities in an adherent lung, rupturing into the substance of the chest walls, and subsequent violent cough driving air into the tissues. Rupture of air cells from greatly increased intrapulmonary air pressure (caused by the taking of a deep inspiration and the subsequent voluntary or involuntary closure of the glottis) which occurs in heavy lifting, the expulsive efforts of labour, playing upon wind instruments (lips taking the place of the glottis), crying, shouting, and violent cough, especially in pertussis. If pulmonary emphysema is present, it predisposes to this accident. From the ruptured air cells the air passes under the visceral pleura or through the interalveolar tissue into the mediastinum and thence to the connective tissue of the neck. Wounds of the lung tissue or axilla and supraclavicular spaces.

Abdomen.—From stomach or intestines, after adhesions to the abdominal walls have been formed, and rupture has occurred, due to ulceration or traumatism. If also septic material from the digestive tract passes into the skin, there may be diffuse inflammatory or necrotic processes in the subcutaneous tissues, associated with the presence of gas-producing organisms.

Starting from any point of entrance, emphysema may spread over wide areas. The extent of its diffusion depends either upon the aspiration or suction force of the tissues into which the air is solicited, or upon the degree of the air pressure in the air-containing viscus from which it is driven into the areolar spaces.

IX. CONDITION OF THE JOINTS

The joints, large or small, may present deviations from the normal in comparative size, shape, colour, position, or mobility.

Examination of the Joints.—One should observe if the joint is swollen, distorted, or reddened, and note also the position (extended, partly flexed) in which it is preferably kept by the patient. Try passive motion to determine its mobility, and the presence of creaking or grating on movement. Palpate the joint to ascertain whether it is tender or hot; to discover irregularities or thickening of the ends of the bones forming the joint or the edges of their articular surfaces; to determine, by trying for fluctuation, if the joint contains an excess of fluid; and, by finding bogginess or firm bulging along the line of the joint, whether the synovial membrane is thickened. Finally, if the motion of the joint is limited, make an effort to decide whether it is due to shortened and spastic muscles (contractures), to anchylosis (fibrous or bony), or to changes in the bone (exostoses and outgrowths) by which the joint is locked.

Significance of Joint Symptoms.—In a certain proportion of cases the nature and associations of the joint lesions can be readily determined; in others the differential diagnosis is extremely difficult and requires a most careful consideration of the associated signs and symptoms, as well as of the character of the local articular alterations. The latter are by no means always characteristic.

Rheumatic Fever.—First one joint (usually the larger first), then another becomes swollen, red, and tender. Suddenness of onset, fever, acid sweats, and sudamina, the rapid occurrence of anæmia, and particularly a shifting of the inflammation from one joint to another are characteristic of this disease.

Chronic Rheumatism.—The joints are painful (especially in the morning), stiff, and perhaps slightly swollen, not often deformed. Fever is rarely present and the disease is essentially chronic.

Gout.—The paroxysm begins suddenly. The proximal joint of the great toe is first and most commonly attacked, then the ankle, knee, and small joints of hand and wrist. The joint is excessively painful; the skin hot, tense, and shining.

Arthritis Deformans.—In young persons many joints are involved, first small, then large; in older patients one or two large joints only may be affected. The joints may be only slightly painful. Owing to the changes which occur in the bones and articular cartilages, the joints become extremely deformed and there is grating or creaking upon movement. This disease, above all others, locks the joints in a more or less fixed position.

Post-febrile or Secondary and Septic Arthritis.—During the period of convalescence from certain of the specific infections, one or a number of joints may become swollen, tender, and contain an effusion which may, but does not often, proceed to suppuration. These are: *Gonorrhœa*, followed by rheumatism (so called), usually monarticular, affecting only one of the larger joints and lasting for months or years; *scarlet fever*, pain and swelling of multiple joints resembling rheumatic fever; *cerebro-spinal meningitis*, which is frequently complicated by simultaneous pain, swelling, and effusion (serous or purulent) of many joints; *smallpox*, joint swellings during the period of desquamation; *dengue*, attended by red, swollen, and painful large and small joints; *pyæmia*, in which one or more joints may become inflamed, with rapid suppuration and destruction of the joint; *typhoid fever*, *dysentery*, and *glanders*. Here also may be mentioned the *acute arthritis* of infants, a sudden inflammation of a single large joint, usually the hip or knee, which becomes rapidly purulent; and acute *osteomyelitis* (a condition which is liable to be mistaken for a much less fatal ailment), in which there are boring pain, swelling, and tenderness, usually of one, possibly of more than one, of the ends or epiphyses of the bones entering into the formation of the larger joints, generally in the lower end of the femur or in the tibia, and attended with high fever and serious constitutional disturbances.

Diseases of the Blood.—In hæmophilia there may be joint symptoms resembling those of acute rheumatism, affecting mainly the larger articulations; in the arthritic forms of purpura there are multiple joint swellings; and in scurvy there may be actual or apparent arthritis.

Diseases of the Nervous System.—The association of chorea with multiple arthritis, probably indeed with rheumatic fever, is well known; in hysteria one of the joints may be tender and rendered immovable by contracture, giving rise in some instances to a difficult diagnostic problem; and there may be multiple arthritis in acute myelitis.

In locomotor ataxia and syringomyelia occur the curious joint lesions called arthropathies. One or more joints, usually the knee,

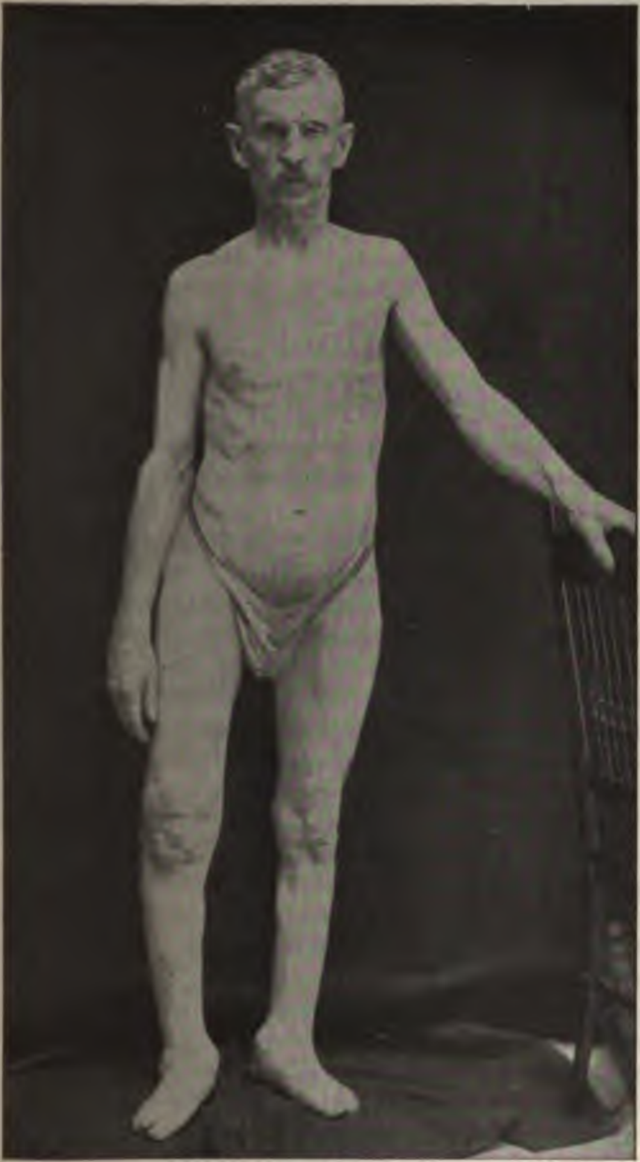


FIG. 22.—Charcot joints (right knee and both ankles) in tabes (Pearce).

but also the ankle or hip, may undergo a sudden, perhaps great swelling, frequently without pain. The cartilages and bones disintegrate and the joint may be thoroughly disorganized and dislocated, the resulting changes resembling those of arthritis deformans. The joint and bone changes in hypertrophic pulmonary osteo-arthropathy may also be classed here.

Miscellaneous affections are: simple acute synovitis with effusion (traumatic, rheumatic), most commonly involving the knee joint; tuberculosis of the joints; and the osseous changes in rachitis.

SECTION XI

THE TEMPERATURE OF THE BODY

1. Method of Taking the Temperature.—Required is a seasoned clinical thermometer, with an error of not more than $\frac{1}{5}$ of a degree. Before and after using, it should be washed with soap and cold water and an occasional cleansing given with alcohol, ether, or chloroform. In case of contagious disease the thermometer should be carefully sterilized by soap and water, alcohol, and an hour's soaking in a 1:500 sublimate solution. It is much better to have a supply on hand, so that one may be left with each contagious case, to be destroyed or sterilized at the termination of the disease. Each time before using, the mercury column should be shaken down to 95°, in order that subnormal temperatures may not escape detection. The thermometer may be placed in the *mouth*, *axilla*, *rectum*, *vagina*, or the fold of the *groin*. The temperature of the *urine* is sometimes tested in a male suspected of malingering.

(*a*) If the *mouth* is employed, neither a very hot nor a very cold drink should have been taken for half an hour before. The thermometer should then be slipped under the tongue to one side and the patient cautioned to keep the lips closed. It is allowed to remain for 3, and if accuracy is desired for 5, minutes. The special 1-minute thermometers are convenient, but very easily broken.

(*b*) If the patient is comatose, dyspnoic, can not breathe through the nose, or is too young to hold the thermometer in the mouth, the *axilla* may be employed. The armpit should be carefully dried and the bulb of the thermometer placed in its hollow. The arm should then be brought forward over the chest, the elbow touching the thorax, and held in this position for at least 5, and if accuracy is desired 8, minutes.

(*c*) If the *rectum* or *vagina* is used, the thermometer is to be oiled and introduced to the depth of 2 inches. The rectum must be empty of fæces. The temperature may be taken by the rectum or vagina in unconscious patients, in those who are being tubbed, in all cases where extreme accuracy is required, and also where there is doubt as to a recorded reading or a suspicion of malingering.

(*d*) The fold of the *groin* may be utilized in infants or plump persons, but this resort is rarely necessary. Very seldom is it requisite to take the temperature of the *urine*. If done, a rapidly acting thermometer is held so that the stream of urine impinges upon it during the act of micturition.

(*e*) The *surface temperature* is taken by a self-registering thermometer, the base of which has been flattened or coiled so as to present a relatively large surface for contact with the skin. It may be held in place by the hand, or by a perforated elastic strap encircling the part. It is better to use two instruments simultaneously, one applied to the part it is desired to test, the other to the corresponding part of the opposite side, allowing them to remain at least 5 minutes.

2. FREQUENCY OF TAKING AND MANNER OF RECORDING.—(*a*) *Frequency.*—In ordinary cases the temperature should be taken if possible morning and night, preferably at the same hour each day. In hospital practice the hours usually chosen are 8 or 9 A. M., and 5 P. M. to 8 P. M., varying somewhat in different institutions. If in private cases a professional nurse is not on duty, it is frequently possible to instruct an intelligent member of the family in the use of the thermometer, or to leave one with directions to place it in the mouth or axilla, and in due time to lay it carefully aside to be read by the physician at his next call. If not really necessary, it is advisable to take the temperature seldom or not at all, because of the exaggerated importance which the laity attach to a slight rise or fall.

In continued fevers, as in typhoid, the temperature should be taken every 4 hours. If it is a disease in which the changes of temperature are rapid and great, as in pyæmia, it may be advisable to take it hourly or every 2 or 3 hours. If a chill should occur in any disease, the temperature should be taken during its continuance, and again 1 hour after its cessation, as otherwise a high and significant temperature may be overlooked. In obscure cases (e. g., hidden tuberculosis or suppuration) the temperature should be taken sufficiently often during the 24 hours to show any possible variation from the normal.

(*b*) *Records of the Temperature.*—In all febrile cases of consequence the temperature, pulse, and respiration should be noted upon a clinical chart, because of the self-evident advantages of the graphic method of record. Personally I find the chart illustrated in Chart I

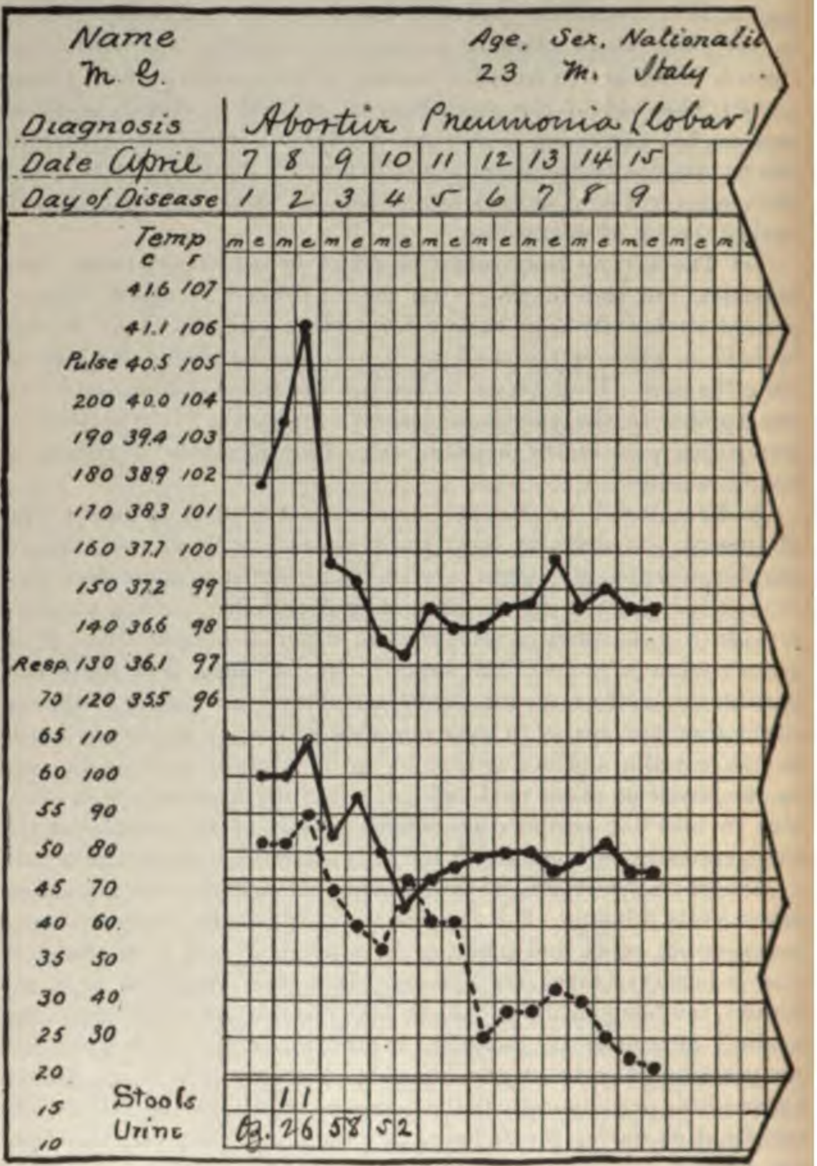


CHART I.—Abortive pneumonia. Dulness and broncho-vesicular respiration over right lower lobe on April 8th. Upper line, temperature; middle line, pulse; broken line, respiration.

suitable for private practice. It is a distinct advantage to have spaces for the date, the day of disease, the bowels, and the urine. Notes of certain events which have a direct bearing upon the pulse, respiration, and temperature—e. g., chill, hemorrhage, cold bath, or the administration of digitalis—may also be entered upon the chart in order to show their correlation in a more striking manner.

Chart I is arranged for A. M. and P. M. takings, and Chart XX, by erasing the headings and ruling vertical lines, for 4-hourly takings. Any chart thus may be adapted to any number of daily takings which may be required. It is also practicable, and will avoid the multiplication of charts, to place readings taken between the regular times as nearly as possible in their proper time spaces, and to connect *all* the readings by lines in red ink, while the regular A. M. and P. M. readings alone are connected by lines in black ink.

3. NORMAL TEMPERATURES.—The temperature of the body is registered according to the centigrade scale, or by that of Fahrenheit. The latter is used in this book.

To convert degrees Fahrenheit into centigrade, if above zero, subtract 32, multiply by 5, and divide by 9.

To convert degrees centigrade into Fahrenheit, if above zero, multiply by 9, divide by 5, and add 32.

It is seen that 1° C. = 1.8° F., and 1° F. = $\frac{5}{9}$° C.

The following table by Stuart is useful for reference:

COMPARISON OF THERMOMETRIC SCALES

	Centigrade	Fahrenheit	
	43°	109.4°	
	42°	107.6°	
	41°	105.8°	
	40°	104°	
	39°	102.2°	
	38°	100.4°	
Normal temperature,	37°	98.6°	*Normal temperature.*
	36°	96.8°	
	35°	95°	
	34°	93.2°	

The normal temperature of the body varies within narrow limits. The average is 98.6°. Any temperature from 97.2° to 99.5° may be considered to be within proper boundaries, although these may be exceeded for very short periods under certain circumstances. It is modified somewhat by *age*, the temperature of the newborn infant ranging from 99° to 99.7°; while between 60 and 70 years of age it may vary from 99.7° down to 97°. Violent exercise, especially in warm weather, will raise it to a slight extent. Mental exertion or excitement is also competent to raise the temperature to a moderate degree (100.4°). The temperature of the air exercises little influence on the temperature of the human body. In very hot weather it may be 99.5°.

The most important normal variation is that which occurs diurnally. Under ordinary circumstances the temperature is highest between 5 P. M. and 8 P. M., and lowest between 2 A. M. and 6 A. M. The difference between the highest and lowest points is about 1.8°, although exceptionally it may amount to 3.6°. The influence of this rhythmical change of temperature is seen in many fevers. In those who work by night and sleep by day this type is reversed. The temperature may rise .4° after a meal, the so-called "fever of digestion." The readings of the thermometer differ somewhat according to the cavities in which it is placed. It is to be remembered that the mean temperature of the blood in the interior of the body, as nearly as it can be ascertained, is 102.2°.

TABLE OF NORMAL TEMPERATURES

	Mean or average.		Minimum.		Maximum.	
	Fahr.	Cent.	Fahr.	Cent.	Fahr.	Cent.
Normal (thirty years of age)	98.6°	37°	97.2°	36.2°	99.5°	37.5°
Infancy to six years of age	99.4°	37.4°	99°	37.2°	99.7°	37.6°
Old age (sixty to eighty years)	98.2°	36.8°	97°	36.1°	99.7°	37.6°
Exercise (severe, in warm weather)	....		98.6°	37°	100.4°	38°
Mental activity	....		98.6°	37°	100.4°	38°
Diurnal variation	....		98°	36.7°	99.4°	37.5°

OF CAVITIES OR FLUIDS

Axilla	98.6° (37° C.).	Mouth	98.6° (37° C.).
Urine	98.6° (37° C.).	Rectum	100.4° (38° C.).
Vagina	100.9° (38.3° C.).		

The normal surface temperature of the *head* (Gray) is, for the left side, an average of 93.8°; for the right, 92.9°. The average temperature of the *thoracic walls* is 96.8°; of the *abdominal walls*, 95.9°.

4. ABNORMAL TEMPERATURES.—Heat production (*thermogenesis*) depends upon the destructive metabolisms, mainly processes of oxidation, which are constantly going on throughout the tissues of the body. The skeletal muscles and the glands, especially the liver, constitute the chief seats of heat production.

Heat dissipation (*thermolysis*) takes place mainly through the expired air, and by conduction, radiation, and evaporation from the skin. As from 77 to 85 per cent. of the total heat loss passes off from the cutaneous surface, the skin must be considered as the principal factor in heat dissipation.

As the normal temperature of the body varies within such narrow limits, there must be some means of regulating the relative amounts of heat production and heat dissipation, in order that they may balance each other with exactness, and under widely differing circumstances. It is quite certain that a heat-regulating (*thermotaxic*) mechanism exists as a part of the nervous system, although the mode of operation and location of the heat centres, and the nerve paths through which the work is accomplished, are as yet uncertain, experimental work upon this point not having given decisive results.

The abnormal temperatures are those which are *over-normal*, (*A*) fever; and *under-normal*, (*B*) subnormal temperature.

(*A*) FEVER

A condition indicated by a marked and more than transient rise of temperature implies a disturbance of the normal relation between heat production and heat dissipation, from causes acting upon one or the other, or upon the heat-regulating mechanism. Heat production in fever is largely increased, because of the more rapid destructive processes which take place in the body, as evidenced by the larger amounts of urea and carbon dioxide which are excreted, and the larger quantity of oxygen which is consumed. But increased formation of heat alone is not sufficient to account for the rise of temperature, as a corresponding increase in the heat loss can readily dispose of a large excess. It is therefore necessary to assume a disturbance of the thermotaxic mechanism, and this perturbation of heat regulation is perhaps the most characteristic element in the production of fever heat. Fever is a complex process or condition of which the presence of an elevated temperature is the most significant and practical indication.

Clinically all cases of fever are attended by certain symptoms in common, due partly to increased tissue changes, partly to the increased heat of the body, and partly to functional disturbances of certain organs. Besides the abnormal rise of temperature, these symptoms are: ill feeling, or malaise; sleeplessness; thirst; loss of appetite; mental disturbance, amounting possibly to delirium; increased frequency of pulse and respiration; lessened amount of urine, with increase in urinary solids, especially urea; usually headache and backache; and, if the fever is long-continued, general wasting of the body. There is also apt to be constipation, and a chill may initiate or accompany a sudden rise of temperature.

The symptoms accompanying fever exhibit considerable variations in severity according to the character and cause of the disease, and the duration and height of the pathological temperature. There is also a difference in the staying power of individuals, more easily recognised than explained, which with equal temperatures and apparently similar causes will allow one to continue about his daily work with slight subjective symptoms and send another to bed in a condition of extreme weakness and discomfort. The malaise may be slight or marked. Thirst and dryness of the mouth, together with anorexia, epigastric discomfort on taking food, and constipation, are due to the partial or almost entire suppression of the secretion of the salivary, gastric, pancreatic, and intestinal fluids. Indeed, the total fluids of the body are diminished in quantity, the urine is scanty and high coloured, and the solid tissues waste away because of the great activity of retrograde metamorphosis. The pulse, as a rule, is abnormally frequent, rising 10 beats for each degree of fever; but there are some exceptions. Typhoid fever, meningitis, and pneumonia may present a high temperature, and yet the pulse be but slightly accelerated; while in scarlet fever, diphtheria, and peritonitis, the pulse may be very rapid in proportion to the fever. The respiration follows a similar rule, increasing 2 per minute to each degree of fever, and also has its exceptions. The variations in pulse and respiration are doubtless due either to the effect of overheated blood or to the action of toxines upon the centres in the medulla. A third factor which may cause an unduly rapid pulse rate is the degeneration of the cardiac muscle which in some cases occurs toward the close of a long-continued fever. Backache, headache, and aching of the limbs are nearly always present, but there is considerable variation in their severity. As a rule, they are most marked at the beginning, and, as in some of the acute infectious diseases, may possess distinct diagnostic value because of their prominence as symptoms. The headache of typhoid fever and the backache of variola are examples. The mental disturbances of fever may be manifested by an increased activity of the mind, passing into active delirium; or by mental torpor, deepening into a low muttering delirium. The degree or character of the delirium or mental disturbance does not appear to depend upon the height of the temperature, but rather upon the nature and amount of the toxines circulating in the blood and their effect upon the nervous system. Personal idiosyncrasy and age modify the liability to the occurrence of delirium. Children, when suffering from fever, are especially prone to it, and some adults resemble children in this respect. Fever temperatures are classified according to (1) height and (2) type.

(1) **Terminology of Fever according to Height.**—This is shown in the following table, somewhat modified from those of Wunderlich and Finlayson. The figures of the two scales in this table do not exactly correspond, except for those of normal temperature:

		Fahrenheit.	Centigrade.
Normal temperature		97.2° 99.5°	36.2° 37.5°
Subfebrile temperature		99.5° 100.5°	37.5° 38°
Slight fever		100.5° 101.5°	38° 38.5°
Moderate fever	Morning	101.5°	38.5°
	Evening	103°	39.5°
High fever	Morning	103°	39.5°
	Evening	105°	40.5°
Hyperpyrexia, above		106°	41°

Some astonishingly high temperatures have been reported, so high that it taxes credulity to receive them as acknowledged facts. A temperature of 122° is within the bounds of belief, but temperatures of 151° and 228°, the latter 16 degrees over the boiling point of water, must, in spite of the care taken in the observations and the excellent repute of the observers, be classified with the tricks of the prestidigitateur, as impossibilities apparently accomplished by means unknown to the observer.

In almost all forms of fevers the temperature is apt to be lower in the morning, the *remission*, and higher in the evening, the *exacerbation*. In exceptional cases the morning temperature is higher than that of the evening. It is spoken of as *inversion*, or the inverse type of fever.

(2) **Terminology of Fever with Respect to its Type.**—This depends upon its duration and the amount of difference between the highest and lowest daily readings of the thermometer, as follows:

1. *Continued Fever*, in which the temperatures are usually high, and the daily differences do not exceed 2° (see Chart II, A).
2. *Remittent Fever*, in which the daily difference exceeds 2°, but the minimum temperature is above the normal limit (Chart II, B).
3. *Intermittent Fever*, in which at least once in the 24 hours the maximum is very high, and the lowest temperature is normal or subnormal (Charts II, C and III, A).

The terms intermittent and remittent have by custom acquired a special meaning, as indicating certain forms of malarial fever. In this connection reference is had to the type and not to the cause of fever, which is said to be *quotidian* when the rise and subsequent fall to normal occurs once every day; *tertian*, when it occurs every other day, having one day free from fever in between; and *quartan*, when two days of freedom from fever elapse.

If two paroxysms, or rises and falls, occur in one day, they are spoken of, according to the interval of apyrexia, or freedom from fever, as double quotidian, double tertian, or double quartan. A better knowledge of the life history (*q. v.*) of the *Hæmatozoon malariæ* (Laveran) has modified somewhat the original meaning of these terms.

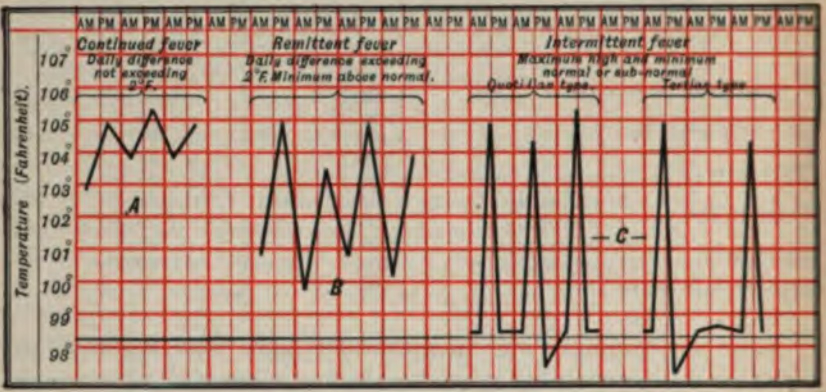


CHART II.—Types of fever. Continued, remittent, and the quotidian and tertian types of intermittent.

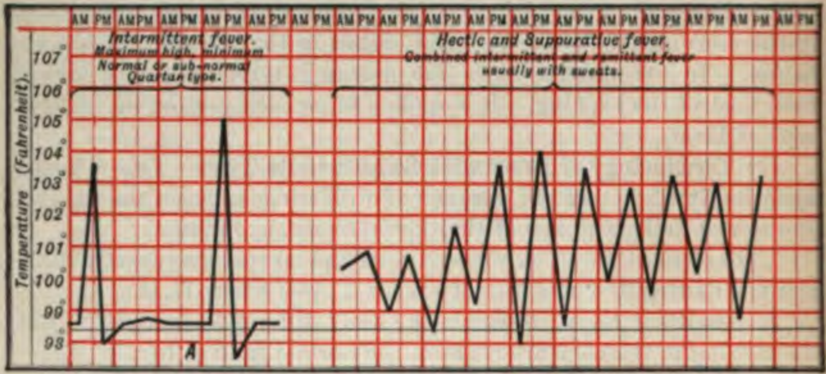


CHART III.—Types of fever. Intermittent, quartan type; also hectic and suppurative fever.

4. *Recurring fever*, a return or recrudescence of a febrile movement after several days of apyrexia.

5. *Irregular fever*, in which the temperatures are very irregular—sometimes high, sometimes low. There is no regular daily difference, and the highest or lowest point may be reached at any hour of the twenty-four. It is essentially atypical.

It is necessary to bear in mind that any one of these types may resolve itself into any other one, according to the nature, cause, or complications of the disease which it accompanies. A remittent may be merged into the continued type, and the continued type terminate as an intermittent. Nevertheless, certain diseases are characterized by certain types of fever with sufficient frequency to give the recognition of the type a distinct and sometimes great diagnostic value, and the absence of the type may be an important negative symptom.

(3) **Manner of Invasion, Course, and Termination of Fever.**—The invasion or beginning of fever may be (*a*) sudden or (*b*) gradual.

(*a*) *Sudden Invasion.*—The temperature rises to as high a point in the first 2 or 3 hours as it subsequently attains during the course of the disease. A rapid rise to a considerable height is attended by chill, coldness, chattering teeth, and a more or less violent shaking of the body. The surface is pale, the skin cool and shrunken, the lips and finger nails are blue. If the thermometer be placed in the mouth or rectum, it may indicate a high degree of fever, the coldness of the surface being due to the contraction of the peripheral arterioles, which prevents the heated blood of the interior from coming to the surface. As the contraction relaxes the chill ends, and the skin becomes abnormally hot by the restoration of the cutaneous circulation.

(*b*) *Slow Invasion.*—On the other hand, the invasion may be slow and gradual, the febrile movement requiring several days to reach its ultimate height. There may be chilliness, but the decided rigour of the rapid rise is absent.

The termination or defervescence of fever also may be (*a*) *sudden* or (*b*) *gradual.*

(*a*) If the fever ends by a sudden drop of the temperature to or below the normal, the termination is by *crisis*, and is usually accompanied by profuse sweating and an increased flow of urine.

(*b*) If the decline of the fever is slow and gradual, several days elapsing before the temperature reaches normal, the termination is by *lysis.*

Course.—Certain fevers, usually continued or remittent, possibly recurrent in type, pursue a sufficiently definite general course to permit a division of the fever into three periods. The first period, called variously the *initial stage*, *initial period*, *stadium incrementi*, is that during which the temperature is rising, rapidly or slowly. The second or middle period, the *acme*, *fastigium*, *stadium fastigii*, is characterized by a high and continuous temperature with but slight daily differences between the maximum and minimum. The third and final period, during which the temperature is descending rapidly or slowly to the normal, is termed the *defervescence* or *stadium decrementi.* It is obvious that of the terms applied to the middle period one only, acme, is appropriate to the intermittent type of fever, because of the usually brief duration of the latter.

Pathological temperatures include, besides febrile disturbances, *subnormal temperatures.* A temperature below 97.2° is subnormal, and 95° is the temperature of collapse. The lowest reported record is 71.8°.

(4) **Diagnostic Indications from the Temperature.**—Changes in the temperature are much more easily produced when it is already abnormal. Consequently, if fever exists, agencies which in health would cause little or no alteration in the temperature will give rise to marked oscillations in the fever curve. Indeed, without appreciable cause it rarely remains stationary, but varies to a greater or less extent every hour and every day. If hourly observations are made for 24 hours in almost any case of pyrexia many irregularities will be revealed which can not be explained by past events or future developments. Nevertheless, an attempt should always be made to ascertain the cause of any decided or unexpected variation, as such changes may possess an important diagnostic or prognostic value.

Putting aside for the present the variations due to disease, it is well to remember that an increase in the fever may be due to mental excitement, pain, an excessive amount of clothing, an overheated room, and the ingestion of food. On the other hand, an unusually cool room, cold sponging, and the cold tub are fever-reducing agents. If a slow loss of blood occurs, as when menstruation takes place during fever, the temperature is lowered to some extent, and a large, sudden hemorrhage may cause a drop even below the normal.

Causes of Fever.—The ascertained presence of fever is capable of a wide range of interpretation, because of the large number of conditions and diseases of which it is a symptom or concomitant. Its existence requires a careful search for its cause, and one should not rest contented until he has found a satisfactory explanation of its presence or has exhausted his diagnostic resources. In general, fever is caused by or attends:

(*a*) All *inflammations*, acute or subacute, the great majority of which are recognised as being dependent upon the presence of pyogenic micro-organisms in the inflamed area, whence toxic material enters the general circulation.

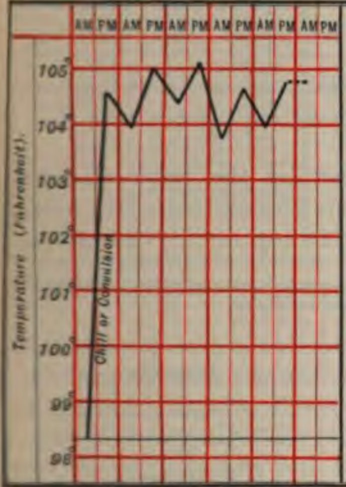
(*b*) All *infectious diseases* due to specific micro-organisms and their products, whether attended or not by local inflammatory lesions.

(*c*) Certain *toxæmias* resulting from the ingestion of poisonous material, or absorption of putrefactive products from the digestive tube, or the formation of toxic material in the glands or tissues of the body.

(*d*) Some non-toxic *diseases* or conditions of the *nervous system* in which the normal working of the heat-regulating mechanism is interfered with by direct or reflex causes.

Diagnostic Classification of Fever.—It is obvious that an attempt to catalogue the special diseases or conditions attended by pyrexia is impracticable, but clinically one may classify fever according to the diagnostic indications of its manner of invasion and termination, its course, type, and variations as follows:

(*a*) *Sudden Invasion.*—A rapid rise to a high point (Chart IV characterizes erysipelas, gastro-intestinal disease in children, middle-ear or mastoid inflammation, malaria, osteo-myelitis, pneumonia, scarlet fever, tonsilitis (lacunar), and all conditions in which large quantities of fever-producing ptomaines rapidly enter the circulation. There is apt to be a chill in the adult, and in children perhaps a convulsion. The initial outbreak of fever occurring in the diseases classified later under the intermittent type might also be included here.



SUDDEN INVASION—NO. 1

TEMPERATURE CONTINUING HIGH. RISE FREQUENTLY ACCOMPANIED BY A CHILL IN ADULTS, AND OCCASIONALLY BY A CONVULSION IN CHILDREN.

ERYSIPELAS.
GASTRO-INTESTINAL DISEASES IN CHILDREN.
MIDDLE-EAR OR MASTOID INFLAMMATION.
OSTEOMYELITIS.
PNEUMONIA (LOBAR).
SCARLET FEVER.
TONSILITIS

CHART IV.—Diagnostic indications to be derived from a sudden invasion of fever. No. 1.

(*b*) *Gradual Invasion.*—A gradual rise of temperature (Chart V) attends the great majority of febrile diseases, except as mentioned in the previous paragraph. Typhoid fever is the typical fever of slow invasion, requiring a week or more to reach the height it will ultimately attain.

(*c*) *Crisis.*—The fevers which terminate by a sudden fall of temperature (Chart VIII) to or below the normal point are erysipelas, measles, lobar pneumonia, relapsing fever, and typhus fever. The crisis or rapid defervescence is usually accompanied by sweating, increased flow of urine, and improvement in the pulse rate and respiration. Occasionally a "pseudo-crisis" occurs from 1 to 3 days before the true crisis, the temperature falling rapidly toward the normal point, but soon rising to or above the previous average. It is especially liable to be seen in pneumonia. The diagnostic import

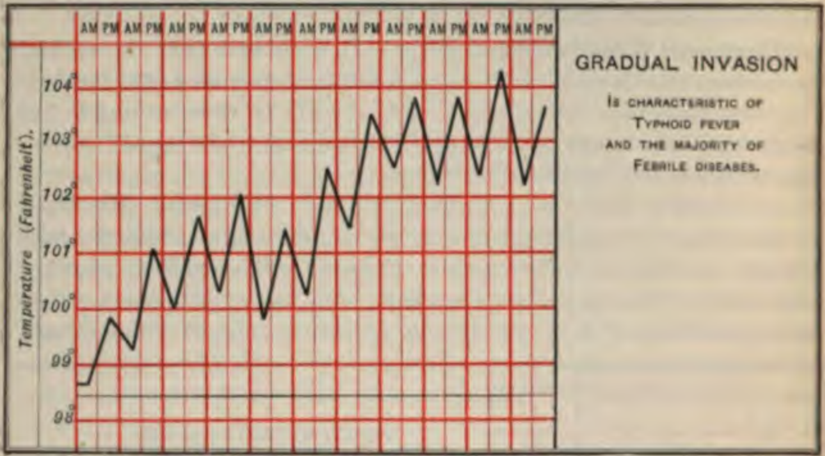


Chart V.—Diagnostic significance of a gradual rise of temperature.

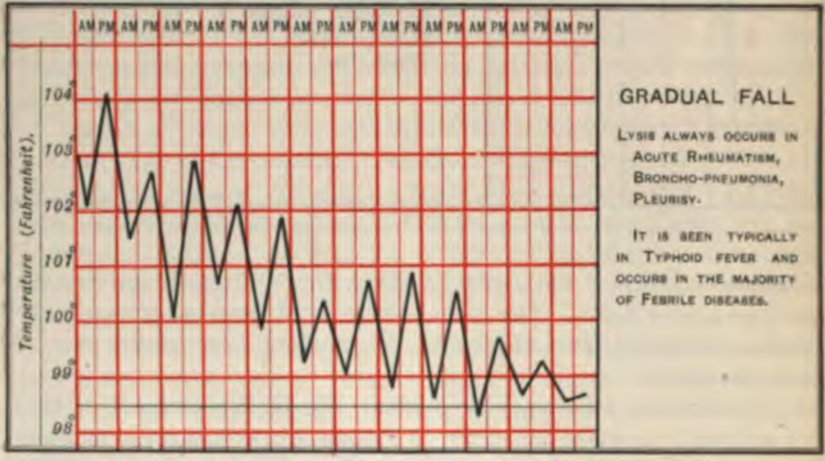
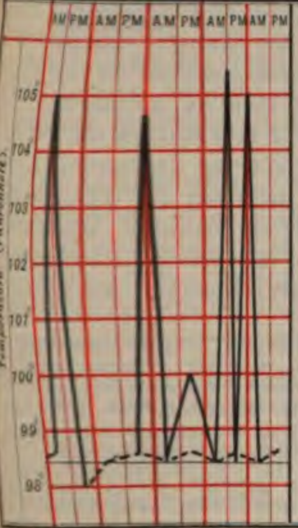


Chart VI.—Diagnostic significance of a gradual termination of fever.

of other sudden falls of temperature will be considered in the subsequent paragraph on the continued type of fever.

(*d*) *Lysis.*—A slow defervescence or subsidence of the elevated temperature is encountered in the majority of febrile diseases (Chart VI). It is not infrequently the case that pyrexial illnesses which customarily end by crisis may, by reason of complications or other disturbing causes, terminate by lysis. The diseases which invariably and characteristically end by lysis are acute rheumatism, bronchopneumonia, pleurisy, and typhoid fever.



SUDDEN INVASION—NO. 2

WITH CHILL OR CONVULSION. TEMPERATURE RAPIDLY FALLING, FREQUENTLY WITH SWEATS, AND EITHER REMAINING NORMAL (DOTTED LINE) OR RECURRING AT REGULAR OR IRREGULAR INTERVALS.

HEPATIC DISEASE (OBSTRUCTIVE, SUPPURATIVE, CIRRHOTIC, OR CARCINOMATOUS).
HODGKINS' DISEASE.
MALARIAL INTERMITTENT (QUOTIDIAN, TERTIAN).
MORPHINE HABIT (EXCEPTIONALLY).
PYÆMIA (TIMES OF RISE IRREGULAR).
SUPPURATION.
SYPHILIS (EXCEPTIONALLY).
TUBERCULOSIS.
ULCERATIVE ENDOCARDITIS.

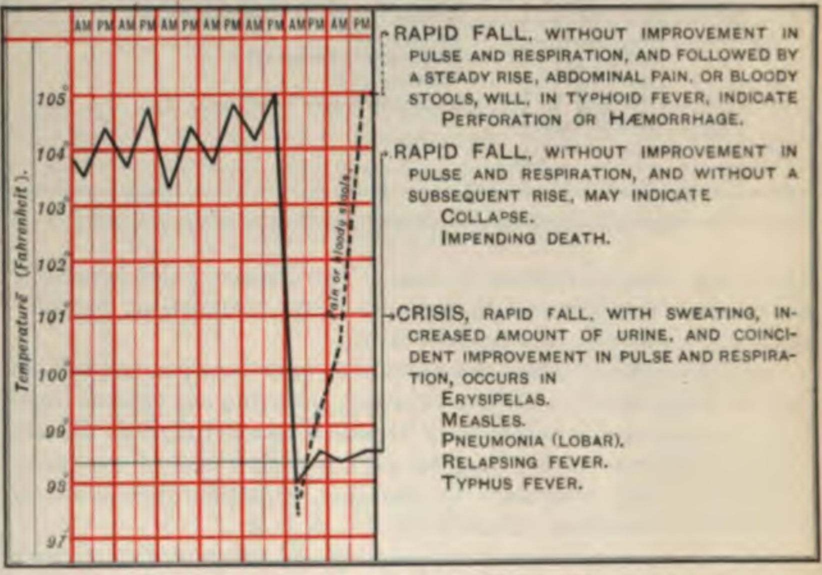
CHART VII.—Diagnostic indications to be derived from a sudden invasion of fever. No. 2.

(*e*) *Intermittent Fever.*—A sudden rise, brief duration, and sudden fall, the latter usually with free sweating, occurring one or more times in 24 hours, creates a suspicion of hepatic disease, Hodgkin's disease, malaria, morphine habit (exceptionally), pyæmia (time of maximum temperature very irregular), suppuration, syphilis, tuberculosis, or ulcerative endocarditis (Chart VII).

The intermittent fever of suppuration and tuberculosis may have regularly a normal morning temperature with an evening exacerbation. If the minimum temperature in any of these diseases remains above the normal the fever becomes of the—

(*f*) *Remittent Type.*—The remittent type may be present at some period during the course of any disease which is attended by fever. Almost all fevers exhibit morning remissions. A remittent type of fever (Chart II) is especially characteristic of tuberculosis and suppuration (*hectic fever*, *q. v.*). The remittent may become intermittent, or the intermittent change into the remittent. If the daily difference between the highest and lowest temperatures of the latter becomes less than 2° it changes its form to the—

(*g*) *Continued Type.*—Continued fever exists in erysipelas, acute pneumonia, acute tuberculosis, typhoid, and typhus fevers. It is especially characteristic of typhoid fever and pneumonia. Sudden changes of the temperature during the course of a continued fever are usually indicative of complications. If the change consists of a sudden rise, it points toward a beginning inflammation or an extension of the already present local lesions. If there is a sudden drop to or below the normal line (Chart VIII), it may betoken collapse, impending death, perforation of the bowels, or an internal hemorrhage—the last two in typhoid fever. It is necessary to discriminate the sudden drop which indicates the crisis in diseases which may terminate in this manner from the drop of collapse or hemorrhage. This may be done by noting the accompanying pulse and respiration and the general condition of the patient, all of which in crisis will show a distinct alteration for the better, thus forming a striking contrast to the change for the worse which may be seen in collapse.



RAPID FALL, WITHOUT IMPROVEMENT IN PULSE AND RESPIRATION, AND FOLLOWED BY A STEADY RISE, ABDOMINAL PAIN, OR BLOODY STOOLS, WILL, IN TYPHOID FEVER, INDICATE PERFORATION OR HÆMORRHAGE.

RAPID FALL, WITHOUT IMPROVEMENT IN PULSE AND RESPIRATION, AND WITHOUT A SUBSEQUENT RISE, MAY INDICATE
COLLAPSE.
IMPENDING DEATH.

CRISIS, RAPID FALL, WITH SWEATING, INCREASED AMOUNT OF URINE, AND COINCIDENT IMPROVEMENT IN PULSE AND RESPIRATION, OCCURS IN
ERYSIPELAS.
MEASLES.
PNEUMONIA (LOBAR).
RELAPSING FEVER.
TYPHUS FEVER.

CHART VIII.—Diagnostic indications to be derived from a sudden fall of temperature.

If the elevated temperature in continued fever is maintained beyond the usual duration of the disease which it attends, search should be made for complications or for persistent local lesions. In some cases, especially typhoid fever, an undue continuance of fever may be found to be due to inanition, the "fever of starvation."

(*h*) *Inversion of the Continued Type.*—High morning and low evening temperatures may occur, although seldom, in pneumonia, tuberculosis, typhoid fever, and dentition. This type is not necessarily of bad prognostic import.

(*i*) *Recurring Type.*—A return or recrudescence of a fever which has ceased may be caused by a relapse of the previous disease, the onset of a new malady, or the beginning of a late complication of the original disease. Relapsing fever (*q. v.*) is the typical recurring fever.

(*k*) *Irregular Fever.*—The principal diagnostic importance of an irregular fever is of a negative character, its presence militating against the existence of any febrile disease which has a characteristic temperature curve, such as typhoid fever or lobar pneumonia.

(*l*) *Solitary Observation.*—Single observations of the temperature may possess some diagnostic value—e. g., the single chill during and after which the temperature rises to about 104°. When this occurs one may apprehend in advance malaria, pneumonia, puerperal fever, pyæmia, scarlet fever, suppuration, or other febrile diseases characterized by a sudden invasion. A single high morning temperature should create a suspicion of an acute infectious disease.

(*m*) *Hyperpyrexia.*—A rise of temperature to an unusual height, 107° to 110°, may happen in connection with acute rheumatism, injury to the cervical portion of the spinal cord, malarial intermittent or remittent fever, scarlet fever, sunstroke, tetanus shortly before death, typhoid fever, and yellow fever. It occurs very rarely in erysipelas, hysteria, pneumonia, and relapsing fever. It may also result from trickery by the use of friction, pressure, hot-water bags, or poultices.

Temperatures over 106° are usually of unfavourable prognostic import, but the degree of danger varies according to the nature of the disease and the duration of the hyperpyrexia. Single brief high temperatures may be well borne if an interval of apyrexia intervenes, as in malarial intermittents; but persistent high temperatures in a long-continued disease, such as typhoid fever, are of evil omen.

(*n*) *Localized Rise of Temperature*, except in surgical diseases, is not of great diagnostic importance. The local heat of the surface is increased over a pneumonic lung, and there may be a rise in temperature in areas affected by vasomotor paralysis. Cerebral thermometry occasionally affords some assistance in obscure cases of brain tumour, but the abnormal elevation must amount to at least 2°.

(*o*) *Subnormal Temperatures.*—The diagnostic import of rapidly occurring falls of the temperature of the body as a whole to a point below the normal have been considered in connection with fever. Persistent subnormal temperature may be present in connection with convalescence from fevers, in acute alcoholism, melancholia, myxœdema, starvation (sometimes elevated), wasting diseases, and in poisoning from carbolic acid or other intoxicant. Locally, abnormal coldness of an edematous or cyanosed part may be observed.

(*p*) *Obscure Febrile Diseases.*—In searching for the cause of a persistent fever, of whatever type, it is an extremely useful rule to bear in mind the possibility of concealed suppuration, tuberculosis, irregular typhoid fever, and ulcerative endocarditis.

This clinical rule was formulated after analyzing a number of cases of persistent fever, of varying types, seen mainly with colleagues. In each instance differing diagnoses had been entertained by several consultants, some of the first rank. Some of these cases may briefly be mentioned as follows: An apparent typhoid, 4 weeks, was caused by a deep perineal abscess; an apparent typhoid, interrupted by a series of chills, with no abdominal tenderness or rigidity —autopsy showed appendicitis, pylephlebitis, multiple liver abscesses; a supposed post-operative sepsis was an irregular typhoid; a 9-week fever was finally explained by the development of tuberculosis of the ribs; a 6-week fever, due to tuberculosis of the rectum; supposed malaria, due to cholecystitis; supposed malaria or tuberculosis, caused by pyelitis; a 6-week fever, due to deep ischio-rectal abscess; a 4-week fever, due to a suppurating intestinal diverticulum; a 3-months fever, diagnosed as malaria or tuberculosis, autopsy showed to be ulcerative endocarditis without physical signs or embolic symptoms.

CHILLS

A true chill is always associated with a more or less elevated temperature, although the surface of the body is cold and pale. The bulk of the blood is retained in the interior zones, and as a result the larger viscera are more or less engorged. The teeth chatter, the lips and nails are cyanotic from the impeded circulation, and the skeletal muscles rapidly contract and relax. There are all degrees of chill, from the "creepiness," which is so often made the subject of complaint, through decided chilliness, up to a hard chill or rigour.

True chill must be discriminated from the so-called "nervous chill," which is seen upon occasions of excitement in individuals with a susceptible and easily disturbed nervous system. In the latter, while the chattering of the teeth and shaking of the body may be as marked as in the true chill, the colour and temperature of the surface remain normal, while the thermometer demonstrates the absence of fever. As a result of hysteria, neurasthenia, or depressed conditions in general, there may be sensations of chilliness or coldness, without shaking and without fever.

It is much too commonly the case, even to-day, that the occurrence of a chill or rigour, especially if recurrent, is promptly followed, *ipso facto*, by a diagnosis of malaria. It should constantly be borne in mind that the great majority of chills encountered in every day practice are due to other causes than the presence of the plasmodium in the blood; and that the finding of the characteristic organism is the only diagnostic evidence which justifies a diagnosis of malarial infection. In view of the unquestionable frequency with which an unwarranted diagnosis of malaria is made, and serious conditions, perhaps requiring surgical interference or other radical treatment, thereby overlooked until it is too late to save life, it should be a hard and fast rule not to label a case "malaria" unless the specific organism has been discovered, or the other possible causes of chill have been canvassed *seriatim*. As J. W. Walker writes me: "Let the plasmodium be no longer a scape-goat; it has sins enough of its own."

Severe Chills (rigours).—The diseases in which a rigour is most likely to occur are acute pneumonia, the onset of extensive suppuration, ulcerative endocarditis, beginning or extending sepsis (any cause), pyæmia, and peritonitis. In general all febrile diseases attended by a sudden and rapid rise of temperature are characterized by a marked chill. Other conditions are empyæma, appendicitis, phlebitis; acute pyelitis and pyelo-nephritis, renal embolism, the passage of a renal calculus, or catheterization; the passage of a gallstone, or cholecystitis; and pelvic abscess, infected abortion, or a ruptured and infected ectopic pregnancy. Certain acute specific diseases are rather frequently initiated by a chill, e. g., epidemic influenza, malaria (especially), typhus fever, variola, acute rheumatism, relapsing fever, and cerebro-spinal meningitis; less often scarlet fever, erysipelas, and diphtheria.

Recurrent Chills.—The diseases most commonly responsible for repeated chills are pyelonephritis, multiple abscesses of the liver, renal calculus, gallstones and cholecystitis, acute tuberculosis, glanders, malaria, and, less frequently, epidemic influenza.

Chilliness.—A sensation of chilliness, not a rigour, often felt up and down the back, frequently attends epidemic influenza, erysipelas, dysentery, malaria, follicular tonsilitis, epidemic parotitis, acute rheumatism, pyelitis, pleurisy, and phthisis pulmonalis.

SECTION XII

EVIDENCE OF GENERAL DIAGNOSTIC VALUE DERIVED FROM THE DIGESTIVE AND GENITO-URINARY SYSTEMS

THERE are symptoms pertaining to the digestive system and urinary apparatus which may be quite independent of local disease, or if the latter is present it may be secondary to morbid processes elsewhere. These symptoms are the condition of the appetite, the presence of unusual thirst, the occurrence of nausea and vomiting, the character and frequency of defecation, the amount and frequency of urination, and certain genital symptoms.

I. APPETITE

The appetite for food may be diminished or lost, *anorexia;* increased, *bulimia;* or depraved, *pica.*

(*a*) **Anorexia.**—Lack of appetite is most frequently caused by fever, of which it is often the first notable symptom, and is apt to persist during the course of the pyrexia. It is also found in debilitating and wasting diseases, conjoined or not with fever, as in cancer or chronic phthisis. A remarkable loss of appetite is seen in some cases of hysteria, in which the desire for food is absolutely extinguished (*anorexia nervosa*), perhaps for long periods. The loss of appetite due to worry, anxiety, grief, or suspense is familiar to all. It is a symptom common to all the dyspepsias, and such patients often fancy that they are hungry, but at the smell or sight of food, or after the first morsel is taken, the sensation of hunger is replaced by that of aversion or positive nausea. Anorexia may arise from too long continuance of an insufficient dietary, originally resorted to because of gastric distress after eating, so that the normal desire for food lessens from disuse. It is seen not infrequently in dyspeptics who practise self-prescribed abstinence, one article after another having been stricken off the diet list. The lack of appetite in the chronic alcoholic subject is familiar, as well as the acute anorexia of an impending attack of delirium tremens. Epidemic influenza is a cause of prolonged anorexia. The sense of hunger in chloro-anæmia is usually deficient. Musser emphasizes the frequent loss of appetite in diseases attended with suppuration.

(*b*) **Bulimia.**—The bulimia may be permanent (*polyphagia*) or paroxysmal. An inordinate craving for food is observed in convalescence from diseases which have been attended with fever. The voracious appetite of a typhoid-fever convalescent is proverbial. A large appetite is frequently characteristic of diabetes. A child suffering from pertussis with frequent paroxysms of cough will occasionally be greedy for food because the vomiting which accompanies the cough does not allow time for sufficient absorption to satisfy the demands of the tissues. The patient with chronic gastritis is apt to have a more than excellent appetite, except for breakfast. Bulimia may be a symptom in some of the insanities, and is a characteristic of certain idiots. Sudden and paroxysmal attacks of bulimia may occur in hysteria, epilepsy, Graves' disease, cerebral tumours, and diabetes mellitus. Acoria is an absence of the sense of satiety. This feeling of "emptiness" occurs usually in hysteria or neurasthenia.

(*c*) **Pica.**—A craving for the ingestion of unusual or injurious substances, possibly of a repulsive nature (*coprophagy*), may be significant of insanity or idiocy. Less marked perversions of normal desires may be seen in pregnancy, in chloro-anæmia, in hysteria, and in childhood, although the perversion usually amounts to no more than capriciousness.

II. THIRST

(*a*) *Absence of Thirst.*—It is important, mainly with reference to treatment, to bear in mind that the sense of thirst is blunted or annulled in typhoid fever.

(*b*) *Increased Thirst.*—This is present in many diseases. It is common to all febrile conditions. It is intense in diseases attended with profuse watery discharges from the bowels, like the various forms of cholera, and in sudden large hemorrhages. Profuse sweating (disease, hot weather, exercise) is attended with a marked thirst. Acute gastritis or persistent vomiting, especially when resulting from irritant poisoning, gives rise to great thirst, and an unusual desire for fluids is common in chronic gastritis and cancer of the stomach. Persistent thirst should always lead to an examination of the urine, especially if combined with a large appetite and progressive loss of weight, as such symptoms attend diabetes, both *mellitus* and *insipidus*. A rare cause of thirst is xerostomia, or "dry mouth," in which there is an arrest of the salivary secretion.

III. VOMITING AND THE GROSS CHARACTER OF THE VOMITUS

A sensation of nausea usually precedes the act of vomiting, although nausea alone may be present, and sudden vomiting may occur without antecedent sickness. On the other hand, one may suffer from most distressing nausea with a total inability to relieve it by vomiting, owing to a lack of completeness in the required muscular efforts. With nausea there is generally salivation.

(1) **Muscular Mechanism.**—The muscular mechanism of the act of vomiting involves a sudden deep inspiration, a subsequent immediate closure of the glottis, and a contraction of the diaphragm. At the same moment the cardiac orifice of the stomach is opened by the contraction of the longitudinal fibres. A violent expiratory contraction of the abdominal muscles immediately follows, by which the contents of the stomach are forced outward, the glottis remaining closed and the diaphragm contracted. The chief factor in the act appears to be the contraction of the abdominal walls, the muscular layer of the stomach walls playing a very small part. Persistent vomiting may cause a reverse peristalsis of the duodenum with a consequent passage of bile into the stomach and its appearance in the vomitus. If for any reason the cardiac orifice fails to relax or the abdominal muscles to contract, nausea with inability to vomit results.

(2) **Nervous Mechanism.**—The centre for the act of vomiting (Fig. 23) lies in the medulla in close proximity to the respiratory centre. It is for this reason that nausea may sometimes be relieved by rapid breathing, and that, particularly in children, a temporarily increased respiration rate is frequently significant of nausea. Impulses are sent out from this centre to the diaphragm by the phrenic nerves, to the stomach and esophagus by the pneumogastric, and to the abdominal muscles by the intercostal nerves.

(3) **Vomiting Centre.**—The vomiting centre may be stimulated to send out its impulses either directly by certain toxic substances contained in the blood which flows through it, e. g., the hypodermic use of apomorphia, or indirectly by impressions received from the periphery or from the brain above. For the most part the centre is excited to action by indirect or reflex irritation. This is true even in gastric diseases, for the irritated nerves of the gastric mucosa must send their stimulus to the centre to be reflected to the muscles concerned in the act of vomiting. Children vomit more readily than adults, partly because the nervous system is more excitable, partly because of the more vertical position and less marked cardiac curvature of the stomach.

(4) **Causes of Vomiting.**—The diseases and conditions which may cause vomiting are many. Consequently, vomiting, as an isolated symptom, possesses very slight diagnostic value and requires a careful consideration of the accompanying symptoms in order to determine its cause. In the majority of cases these are sufficiently distinctive, but there are instances in which a most painstaking investigation will leave the clinician in doubt. No case should be classed

as of purely neurotic origin until the blood and the urine have been questioned by appropriate methods.

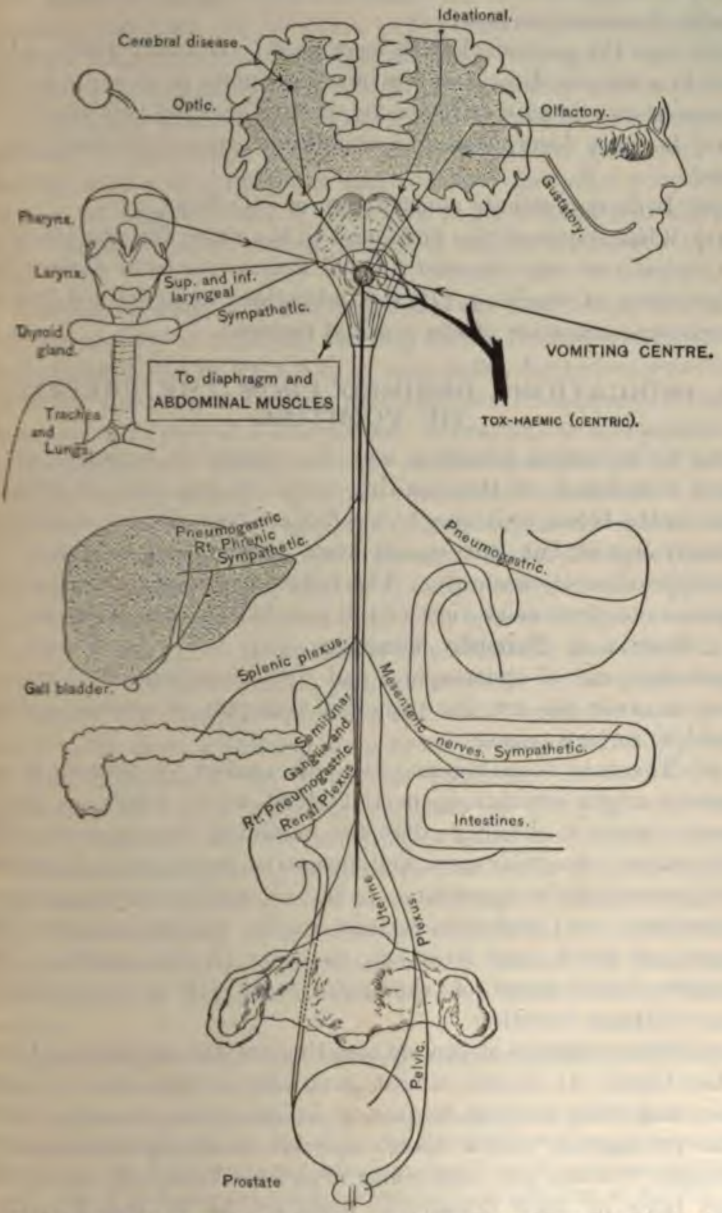


Fig. 22.—Diagram of the vomiting centre in the medulla, and the manner in which stimuli, both toxæmic and reflex, may act upon it.

In any case of vomiting the following queries should be answered:

(*a*) Has any article, medicinal or dietetic, been ingested which is capable of causing nausea?

(*b*) Has the previous health been good? In other words, is it an event in a chronic disease or a primary symptom in an acute malady? If there is gastric pain or headache, is it relieved by vomiting?

(*c*) Is there fever, headache, abdominal pain, or evidence of collapse?

(*d*) Is there obstinate constipation or jaundice?

(*e*) What abnormalities are found in the urine and the blood?

Clinically we may consider (*A*) the indications to be derived from the presence of vomiting, (*B*) the indications to be derived from the macroscopic character of the vomited material.

A. INDICATIONS DERIVED FROM THE PRESENCE OF VOMITING

As to its origin, vomiting may be centric or toxæmic, due to direct stimulation of the vomiting centre by any toxic or irritating agent in the blood, or it may be a reflex act from disease or irritation of many organs, but this classification of vomiting can at best be only approximately accurate. The following enumeration may serve to give a comprehensive view of the possibilities in a given case:

1. **Centric or Toxæmic Vomiting.**—(*a*) Acute alcoholism, the hypodermic use of apomorphia, and the inhalation of chloroform, ether, or sewer gas are the principal examples of centric vomiting caused by foreign poisons.

(*b*) Toxæmic vomiting may also be caused by poisons of non-bacterial origin circulating in the blood, as in Addison's disease, anæmia (acute or chronic); diabetes, heralding the approach of diabetic coma; irregular gout (biliousness); paroxysmal hæmoglobinuria, preceding the appearance in the urine of the colouring matter of the blood; and nephritis, perhaps as the earliest symptom of the disease, in which case it is apt to occur in the morning. The unknown toxic agent of rheumatic fever will in rare instances cause obstinate vomiting.

(*c*) Other varieties of centric vomiting are due to bacterial toxines in the blood. It occurs almost invariably at the onset of scarlet fever, and with varying frequency in erysipelas, measles, variola, acute pneumonia, yellow fever, malarial fever, epidemic parotitis (mumps), roseola, and sometimes syphilis. Vomiting which takes place later in such diseases is more apt to be due to uræmia. Vomiting may be a feature in the course of other infectious and septic diseases, as in acute miliary tuberculosis, acute yellow atrophy of the liver, ulcerative endocarditis, and gangrene of the lungs.

2. **Reflex Vomiting.** (*a*) *Cerebral.*—When vomiting is due to irritations sent from the cerebrum to the centre in the medulla, it is somewhat characteristically projectile and frequently unattended with nausea or epigastric pain. It may be caused by cerebral tumour or abscess; and intracranial hemorrhage, embolism, or thrombosis, particularly hemorrhage. Musser has seen sudden and continuous vomiting, with a slow full pulse and a flushed face, as a premonitory symptom of apoplexy, and I * can add a personal case of the same kind. Chronic and acute hydrocephalus may be attended with vomiting.

Vomiting is a common and sometimes the first symptom of meningitis, especially in children, and may continue throughout the disease. Anæmia of the brain may be responsible for nausea and vomiting, as in vomiting from loss of blood, severe anæmias, syncope, shock, and collapse; also concussion, laceration, or compression of the brain, especially during recovery in the moderately severe cases. Vomiting shortly after an epileptic convulsion is common. It may be caused by a disgusting sight, taste, or odour, or the emotion may be purely subjective. Most obstinate and intractable vomiting may be of hysterical origin, and may be of daily occurrence in children or young people who are overburdened with school work, the so-called "juvenile vomiting." It is usually attended with gastric pain, and perhaps with severe headache, pallor, dilatation of the pupils, and a slow pulse. Paroxysms of vomiting and inability to take food, lasting from 1 to 10 days, appearing in persons apparently healthy before and after the attack, the paroxysms recurring at definite intervals, constitute a very uncommon disease, the periodic vomiting of Leyden. It occurs with much more frequency in children.

Neurasthenia, especially if lithæmia coexists, is the cause of occasional attacks of vomiting, more particularly after fatigue of mind or body. The vomiting of sea-sickness is familiar. A paroxysm of Ménière's disease usually terminates with nausea and vomiting, as does migraine or hemicrania, and the so-called bilious or lithæmic headache.

Muscular asthenopia and refractive errors, particularly astigmatism, may be an occasional cause of nausea, after injudicious use of the eyes, proceeding in some cases to actual vomiting.

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* "That right line 'I' is the very shortest, simplest, straightforwardest means of communication between us, and stands for what it is worth and no more. Sometimes authors say, 'The writer has often remarked'; or 'The undersigned has observed'; but 'I' is better and straighter than all these grimaces of modesty; and I shall ask leave to maintain the upright and simple perpendicular."—THACKERAY, Roundabout Papers.

(*b*) *Pharynx, Larynx, Lungs, and Thyroid Gland.*—As an irritation of the fauces by titillation will cause reflex vomiting, so the diseases which are attended with a severe paroxysmal cough may also produce it, as in whooping cough, the irritative cough of phthisis, and the cough due to enlarged bronchial glands or mediastinal tumour. A sudden attack of vomiting and abdominal pain, in some cases accompanied with a profuse watery diarrhœa and jaundice, constitutes a not infrequent symptom during the course of exophthalmic goitre.

(*c*) *Stomach.*—Diseases affecting the stomach are responsible for the majority of cases of vomiting. Neurotic vomiting occurs in hysteria, and attacks of pain and vomiting constitute the gastric crises of locomotor ataxia. Severe vomiting is a prominent symptom of acute or subacute gastritis from putrefying, indigestible, or irritating food, or from overloading of the stomach. The vomiting of chronic gastritis is usually in the morning, so also is the "dry retching" of the laity, nausea and ineffectual attempts to vomit, in the same disease. Vomiting (often blood-streaked or coffee ground) is almost invariably present in gastric cancer and cirrhosis during the middle or later stages of the disease. Vomiting due to dilatation of the stomach may occur several hours after a meal, or at intervals of several days, in which case the amount of vomited material may be very large and contain food which has been eaten many hours previously. Vomiting due to gastric ulcer is apt to occur 2 or more hours after eating, and is preceded by the most constant symptom of this lesion, viz., pain, which usually begins ½ to 2 hours after eating and increases in intensity until vomiting takes place, after which the pain subsides. If the ulcer perforates, vomiting often begins and is attended by collapse, great and increasing pain, fever and other symptoms of acute peritonitis.

Vomiting may be due to the administration of the medicinal emetics, and to irritant poisons. With the latter there is usually pain, collapse, and subsequently more or less violent purging. Poisoning by antimony can not be distinguished by the symptoms alone from cholera, epidemic or sporadic. The history, environment, and season may arouse suspicion. The presence of bloody stools in arsenic poisoning will discriminate it from cholera. Paris green may be detected in the vomited material. Poisoning from copper colours the vomit green, turning to blue upon the addition of ammonia. In phosphorus poisoning vomiting begins several hours after taking the poison, and the vomited material smells strongly of the drug and may be phosphorescent in the dark.

(*d*) *Intestines and Peritoneum.*—Vomiting is a most common symptom at the onset of appendicitis; and in every case of acute vomiting accompanied with abdominal pain, the cæcal region should be carefully palpated to discover possible muscular rigidity or tenderness. It is early, severe, and prolonged in cholera infantum, cholera Asiatica, and sporadic cholera. In a perforating duodenal ulcer, or a perforating ulcer of other parts of the intestine, vomiting with collapse occurs in connection with the symptoms of acute peritonitis. In the acute enteritis of adults and the acute enterocolitis of children vomiting is a frequent event.

In every case of vomiting associated with abdominal pain the possible existence of hernia (femoral, inguinal, umbilical, and the rarer varieties of obturator, ventral, diaphragmatic, and retroperitoneal hernia) should not be forgotten, as a small knuckle of intestine nipped in the neck of a hernial sac may easily be overlooked without a systematic and careful examination. Often the hernial protrusion is so slight as to cause no palpable swelling or tumour, the diagnosis resting upon the localized tenderness and the exclusion of other conditions.

The acute varieties of peritonitis are almost invariably attended with vomiting, which in acute general peritonitis is an early and persistent symptom.

Vomiting may be caused by obstruction of the intestine, and is one of the cardinal symptoms of this condition. The development of this symptom depends much upon the location of the obstruction. A lesion in the upper part of the small intestine is characterized by the rapid appearance of vomiting, often of a violent and expulsive nature; whereas, in obstruction in the large intestine, vomiting comes on tardily, following general tympanites, or, indeed, there may be only a belching of gas without vomiting.

(*e*) *Liver and Gall Bladder.*—Vomiting may be a symptom of diseases of the liver, especially those which cause jaundice, such as catarrh of the bile ducts with its attendant gastro-duodenal catarrh; hepatic colic; and of obstruction of the common duct by gallstones or new growths. The vomiting of "biliousness" has been referred to.

(*f*) *Kidney.*—Uræmic vomiting has been mentioned. Attacks of renal colic, if severe, are attended with nausea and vomiting. Suppurative pyelitis has, in my experience, been characterized by persistent nausea and occasional vomiting. An abrupt onset of vomiting, usually with localized pain and diminution of the flow of urine, may be indicative of a movable kidney which has become twisted upon its pedicle.

(*g*) *Pancreas.*—Vomiting preceded by sudden pain in the epigastrium, followed by collapse, may be indicative of a possible acute pancreatitis; and when attended with paroxysmal attacks of colicky pain and persistent jaundice may be a symptom of cancer of the pancreas.

(*h*) *Uterus, Ovaries, Tubes.*—The reflex nausea and vomiting of pregnancy is familiar. Marked displacement of the uterus, pregnant or non-pregnant, may also be a causal factor. Vomiting may be a symptom of septic endometritis, salpingitis, and ovaritis. The atrocious dysmenorrhœa which characterizes certain cases of anteflexion of the cervix in neurotic young women is often attended with vomiting and partial syncope.

(*i*) *Bladder and Prostate Gland.*—Chronic cystitis may occasionally cause vomiting, absorption of some elements of the decomposing urine having taken place. In acute cystitis and prostatitis the reflex irritation from inflammatory disease of these sensitive organs may in itself be sufficient to produce nausea.

(*j*) *Other Associations of Vomiting.*—Some persons who appear to have a peculiar susceptibility of the nervous system will vomit after severe exertion, although the general health may be above suspicion. Vomiting, with abdominal pain, occasionally with diarrhœa, is observed in the early stage of trichinosis before general infection has occurred.

In this connection ***rumination, regurgitation, eructations, hiccough***, and ***pyrosis*** may be considered.

3. **Rumination or Merycism.**—This is the act of returning swallowed solid food to the mouth, when it is again chewed and swallowed. It is a neurosis, involuntary at first, but becoming voluntary by custom in some cases.

4. **Regurgitation.**—Frequent regurgitation of the liquid portions of food may occur, and is due either to relaxation of the cardiac orifice of the stomach, or to the existence of a sac or diverticulum of the esophagus, into which fluid enters and from which it is expelled at irregular intervals.

5. **Eructations.**—The frequent, spasmodic expression of gas from the stomach by way of the mouth (eructation, belching) is a common symptom. The gas expelled may be offensive (sulphuretted hydrogen) or odourless, consisting mainly of air. The gas may come from the esophagus, and in this case consists of air which has been swallowed. The belchings may take place singly and at infrequent intervals, or in paroxysms, 2 or 3 times a minute, the attack lasting for hours.

In the majority of cases eructations of gas, particularly if offensive, are significant of acute indigestion, overfeeding, acute or chronic gastritis, and other organic affections of the stomach and of the pancreas. It may be a pure neurosis, occurring in neurasthenia or hysteria, as well as in patients who are otherwise healthy or who have been worried and excited. In these instances the gas is odourless. The terminal stage of an attack of angina pectoris may be attended by explosive eructations of gas; and, rarely, the belching may signify the presence of an aneurism of the thoracic aorta.

6. **Pyrosis.**—A burning sensation in the epigastrium, frequently extending up behind the sternum to the pharynx, and sometimes accompanied by the regurgitation of a watery acrid or acid fluid, is termed pyrosis. In common parlance the sensation is called "heartburn"; the regurgitation, "water brash."

Pyrosis is often accompanied by eructations, and, like the latter, may be symptomatic of gastric or pancreatic disease, or may be a neurosis. While it is most frequently associated with a too acid gastric juice (hyperacidity), it may exist with a neutral reaction of the stomach contents.

7. **Hiccough.**—This symptom results from a sudden contraction of the diaphragm which may be repeated at more or less regular intervals. The attack may last for a minute or for several hours, and may recur during days and months.

The *causes of hiccough* are as follows:

It occurs in diseases of the abdominal viscera—e. g., gastritis, gastrectasia, cancer of the stomach; enteritis, internal and external strangulation or other cause of intestinal obstruction, appendicitis, cholera; suppurative pancreatitis; disease of liver; peritonitis, especially if it involves the diaphragmatic peritoneum; and tympanites.

Diseases of the nervous system are not infrequent causes—viz., epilepsy, tumour of the brain, meningitis, hydrocephalus, shock, mental emotions, and hysteria. To the latter many excessively obstinate cases are due.

Certain constitutional conditions appear to be responsible for otherwise inexplicable paroxysms of hiccough, such as diabetes, gout, and chronic nephritis. Other causes are gangrene of the lung, diaphragmatic pleurisy, dysmenorrhœa and pregnancy, alcoholism, Addison's disease, large hemorrhages, and, last but not least, severe typhoid fever and the typhoid state.

The milder forms of hiccough are ordinarily of little consequence, but its onset in conditions which are known to be serious adds notably to the gravity of the prognosis.

B. INDICATIONS DERIVED FROM THE MACROSCOPIC CHARACTER AND AMOUNT OF THE VOMITUS

The first act of vomiting expels the food contents of the stomach, if such be present, the character of which may furnish a clew to the cause of the vomiting. If the vomiting continues, or if the stomach is empty, the vomitus will consist of a watery fluid (partly swallowed saliva), mucus, and finally bile. In certain cases the vomitus may contain streaks of blood, or consist almost entirely of blood. Still more rarely there may be a fæcal odour or visible fæcal matter in the vomit.

(*a*) **Watery Fluid and Mucus.**—If the vomit comes from an empty stomach, is watery, and contains considerable mucus, it indicates gastric catarrh, or a neurosis, or irritation from persistent retching. Succeeding a long nausea, the mucus may have been swallowed. It may be acid or alkaline. If the latter, the watery part usually consists of swallowed saliva. If it is clear, containing little or no mucus, and has a very sour smell, and on examination responds to the tests for hydrochloric acid, it is gastric juice, constituting hypersecretion. If on quantitative examination the proportion of hydrochloric acid exceeds the normal 0.3 per cent, there is hyperchlorhydria. Vomiting of overacid gastric juice occurs persistently or periodically in the gastric neuroses; in the terminal vomiting of migraine; in hysteria; in the gastric crises of locomotor ataxia, exophthalmic goitre, and movable kidney; and in gastric ulcer before and after healing.

(*b*) **Bilious Vomiting.**—Vomit containing bile, green or greenish-yellow, makes its appearance, as a rule, only after frequent and violent vomiting. Vomiting of grass-green bile, occurring early and perhaps with slight effort, in connection with each act of vomiting, is a symptom of some diagnostic value in peritonitis, and commonly precedes fæcal vomiting in intestinal obstruction. The presence of moulds may, at times, colour the vomitus green, or even red. This may be mistaken for bile.

(*c*) **Hæmatemesis.**—The vomited blood may be bright red and fluid, in which case it has remained but a short time in the stomach. If it has been in the stomach for a sufficient length of time to be partially digested by the gastric juice, it has the appearance of coffee grounds. It may be in the form of clots, reddish or brown, indicating a stay of medium duration.

It is necessary to discriminate between hæmatemesis and hæmoptysis, or hemorrhage from the mucous membrane of the bronchi. In the latter the blood is bright red, frothy, and alkaline. It is raised by an act of coughing, not vomiting, and physical examination of the lungs usually reveals signs of its pulmonary origin. If it is from the stomach, tarry stools may be passed, and there will usually be evidences of disease of some of the abdominal organs.

It is to be remembered that the presence of certain substances in the vomited material may give the appearance of fresh blood, such as red wine, grape juice from black grapes, red jellies, preserves made from strawberries, raspberries, huckleberries, currants, cranberries, mulberries, and red cherries, or the fruits themselves. Coffee-ground vomit may be simulated by coffee or cocoa, strong broths or soups, bile, bismuth salts, and ferruginous preparations, all of which may produce a brownish or blackish coloration of the stomach contents.

If there is doubt as to the presence of blood in the vomit, a drop should be placed under the microscope to determine the presence of red corpuscles. If not found, apply *C. Strzyzowski's* hæmin test, as follows: Glacial acetic acid, water and alcohol, each 1 c.c.; hydriodic acid, 3 to 5 drops. The mixture must be freshly prepared for each test. Place the suspected substance on a slide, cover with the reagent and boil for ten seconds. Fluids should be evaporated to dryness before testing. Large and perfect hæmin crystals are thus obtained.

Boas's modification of Weber's test—used especially to determine the presence of blood in the stools—is probably the best of these methods. The patient, for several days, is not allowed to eat meat or fish. The stools are made soft by Carlsbad salts or other laxative. Mix 2 or 3 grammes of the stool with 20 c.c. of water; extract fat from stool by shaking with 20 c.c. of ether; add to the mixture one-third its volume of pure acetic acid and shake; then add 10 c.c. of ether and shake again; let stand until the ether rises to the top. To 2 c.c. of this ethereal extract add first 10 drops of freshly made tincture of guaiac (resin of guaiac 1, abs. alcohol 25), then drop by drop, 10 to 20 drops of old ozonized oil of turpentine (pure oil of turpentine, not that used in the arts, exposed to air for eight weeks). If blood is present an intense blue, or a green or greenish-blue, colour appears within a few seconds, slowly changing to reddish-violet.

Large and suddenly fatal hemorrhages from the stomach may be due to the rupture of an aneurism into the esophagus or stomach, to rupture of varicose esophageal veins, to gastric ulcer, to a large spleen, and to cirrhosis of the liver. Death has taken place before the occurrence of vomiting, the stomach containing 3 or 4 pounds of blood. In the majority of cases gastric hemorrhage is not immediately fatal and the bleedings recur for a considerable length of time. This is particularly the case in ulcer of the stomach, cirrhosis of the liver, and carcinoma of the stomach—the diseases which are responsible for the greater part of the gastric hemorrhages which are met with in practice. The large bleedings are accompanied by the customary signs of internal hemorrhage (*q. v.*). The vomiting of small amounts of blood, sometimes only a few bright-red streaks in mucus, or a moderate quantity of coffee-ground material, occurs in a number of diseases and conditions. Causes of hæmatemesis are:

1. *Swallowed Blood.*—Vomiting of blood is not synonymous with gastric hemorrhage. In epistaxis and hæmoptysis the blood may be swallowed unconsciously. It is also swallowed for purposes of deception by malingerers and hysterical persons, the blood having been obtained from an extraneous source or from self-made wounds of the buccal mucous membrane. Blood from a bitten tongue may be vomited after an epileptic seizure, and an infant may vomit blood coming from a fissured nipple while nursing.

2. *Injury.*—Blows, kicks, wounds, or other injuries in the epigastric region may be followed by hæmatemesis. Streaks of blood are not infrequently seen as a result of the violent straining which attends severe and persistent vomiting.

3. *Stomach.*—Hemorrhage may result from gastric ulcer, and gastric cancer (occasional and moderate coffee-ground). The vomit of chronic gastritis and gastrectasia may present streaks of blood. Arteriosclerosis is an important factor in gastric hemorrhage.

4. *Portal Obstruction.*—Cirrhosis of the liver frequently, cancer of the liver seldom, and an enlarged spleen at times, by causing extreme passive congestion of the gastric mucosa, will produce hæmatemesis; so also congestion of the liver and the portal system secondary to cardiac valvular disease, especially mitral stenosis.

5. *Poisons.*—The corrosive poisons—arsenic, strong acids and alkalies—may be responsible for streaks of blood in the vomitus.

6. *Hæmic Conditions.*—Bleeding from the stomach, more or less profuse, may result from severe anæmias, of whatever origin; it is an occasional symptom of cholæmia, and may be due to hæmophilia. It is sometimes an early event in leucæmia, occurs infrequently in Hodgkin's disease, and may be present in scurvy, purpura hæmorrhagica, and chronic nephritis. The poisons of the acute infectious diseases may so disorganize the walls of the vessels of the gastric mucous membrane that blood escapes from thém into the stomach. It has been observed in severe malarial fevers, typhus fever, epidemic influenza, relapsing fever, yellow fever (black vomit), malignant smallpox, and dengue. It has also occurred in phosphorus poisoning and acute yellow atrophy of the liver. It is a rare symptom in pyæmia, sapræmia, and the toxæmia of appendicitis.

(*d*) **Fæcal Vomiting.**—The presence in the vomit of material coming from the intestine is indicated by a distinctly fæcal odour. Very rarely masses or particles of a clearly feculent character and consistence may be found. Fæcal vomiting is indicative of intestinal obstruction from any of the recognised causes, or of an intense general peritonitis, or of an abnormal communication between the stomach and intestine.

(*e*) **Pus in the Vomit.**—An amount of pus in the vomit sufficient to be seen by the naked eye is of very infrequent occurrence. If present, it is usually indicative of the rupture of an abscess of a near-by organ into the stomach, as in hepatic or pancreatic suppuration, although it may result from phlegmonous or diphtheritic inflammation of the gastric walls.

(*f*) **Parasites in the Vomit.**—The parasites which have been vomited are segments of tæniæ, ascarides or roundworms, *Oxyuris vermicularis* or threadworms, trichinæ, fragments of echinococcus cysts if rupturing into the stomach from the liver or spleen, and the anchylostomum duodenale. The appearance of any one of these, except tænia and ascaris, is extremely rare.

(*g*) **Odour of the Vomit.**—The significance of a fæcal odour has been mentioned. The garlicky odour of phosphorus and the characteristic odour of carbolic acid may reveal the nature of the toxic agent in cases of poisoning. An ammoniacal odour is indicative of uræmia.

IV. DEFECATION AND THE GROSS CHARACTER OF THE STOOLS

In every case of illness it is customary and desirable to inquire as to the character, regularity, and frequency of the bowel movements and the character of the stools.

The bowels normally move once daily without pain. The colour of the stool varies from a bright to a blackish brown, and is due for the most part to the presence of reduced bilirubin (*hydrobilirubin*). The average weight of the stool is about 5 oz. (160 grammes). It is firm in consistence, sausage shaped, about 6 inches long, and is composed of insoluble or unabsorbed portions of the food, intestinal mucus and other secretions of the digestive tube, epithelial cells, and bacteria.

The rhythmical, wormlike contractions of the intestine, by which its contents are passed toward the rectum, are dependent upon the presence of automatic motor centres in its muscular wall. These centres are to a certain extent under the control of the splanchnic, which sends to them both inhibitory and motor fibres, and which affords also the vasomotor and sensory nerve supply of the intestine. Normal peristalsis, therefore, depends upon the functional integrity of the nervous mechanism, the healthy condition of the intestinal musculature, and the presence of adequate and not excessive stimulation, the last of which is ordinarily found in the contact of the chyme with the intestinal walls and in the passage of blood containing the normal proportion of oxygen and carbon dioxide through the intestinal blood vessels.

The deviations of more or less diagnostic importance which may be encountered relate to constipation, diarrhœa, incontinence of fæces, painful defecation, tenesmus, and the colour, shape, consistence, and abnormal contents of the stools.

A. CONSTIPATION

There may be considerable variations in the frequency of defecation which are quite consistent with good health. Certain individuals habitually evacuate the bowel twice or thrice daily, having otherwise a feeling of discomfort, while a movement every 2, 3, or 4 days may be the rule in persons who present no evidence of ill health.

Constipation, which may be roughly defined as a lack of intestinal peristalsis, is therefore a relative term, and its existence is to be determined by inquiry as to the habits of the individual in this respect for a series of years. It is commonly easy in practice to decide between normal and pathological infrequency. Constipation due to a mechanical cause, as in intussusception, constitutes intestinal obstruction.

If constipation becomes habitual, it may cause, in varying degrees, a feeling of lassitude or debility, headache, mild vertigo, mental torpidity, and low spirits—symptoms commonly attributed to the absorption of toxic material from the intestinal canal. These symptoms are manifested most frequently in neurasthenic and hypochondriacal individuals, less often in those of a non-nervous temperament.

As a symptom, constipation is common to many diseases and conditions. In the following paragraphs an attempt is made to state the most important facts which should be borne in mind when seeking to determine the diagnostic indications of constipation in a given case.

1. Constipation may be a *constitutional trait* and is not infrequently hereditary.

2. *Dietetic causes* are not uncommonly responsible for the existence of constipation. If the food is of such a nature as to be almost completely absorbed (milk, meat, meat extracts, eggs), there is an insufficient amount of waste material to give a normal stimulus to peristaltic action. For obvious mechanical reasons, a certain bulk or volume of fæcal matter is required, in order that the intestine, the colon in particular, may easily grasp and propel it to its proper destination. For similar reasons, a too small amount of food, even if of proper composition, will have the same effect.

Constipation may also arise from a too scanty taking of fluids, whereby the fæces become dry and hard, thus lacking the smoothness which normally enables them to slide without friction along the mucous lining of the intestine. Overloading the intestines with food of a coarse and bulky nature may give rise to constipation by overdistending and weakening the intestinal tube. Beverages which contain tannin, like tea or red wines, may lessen intestinal secretion and so cause constipation.

3. *Negligence in the regularity* of defecation and of eating, imperfect mastication, sudden changes in the quantity and quality of food, sedentary habits and consequent insufficient exercise, are more or less potent factors in causing intestinal torpidity.

4. *Alterations* in the quantity or quality of the *digestive fluids* may induce constipation; so also may a deficiency of bile, by depriving intestinal peristalsis of a powerful stimulus.

5. *Profuse* and long-continued *sweating* from heat, exercise, disease, or drugs, or *polyuria* of diabetic or other origin, may produce constipation by lessening the fluidity of the intestinal contents.

6. *Fever*, unless the disease has diarrhœa as a symptom, is usually attended by constipation, because of the diminished secretion of the digestive and other fluids which is the common concomitant of the febrile process.

7. Constipation may be due to a *weakened condition of the abdominal muscles*, on account of which the voluntary expulsive effort of defecation can not be made, as in the distention resulting from pregnancy, abdominal tumour, and obesity.

8. There are certain *local conditions* which may inhibit the emptying of the rectum by causing pain, with consequent reflex spasm of the anal sphincter. These are fissure of the anus, inflamed hemorrhoids, ulcer of the rectum in the neighbourhood of the anus, irritable or inflamed prostate gland, and a tender and prolapsed uterus or ovary.

9. A frequent cause of constipation is a *deficient excitability* of the motor apparatus of the intestine. This may be due to deranged innervation, or to an atonic or degenerated condition of the muscular layer of the intestine, or both. As a manifestation of disordered nervous action it attends myelitis, tetanus, and meningitis, and occurs as a symptom in hysteria, neurasthenia, and anæmia, or conditions of malnutrition. Acute inflammations involving the peritoneum, as in peritonitis and some cases of appendicitis, may cause a very obstinate constipation by producing paresis of the intestinal musculature; so also with acute hemorrhagic pancreatitis. Chronic portal congestion from hepatic or cardiac disease, or chronic inflammation of the intestinal mucosa may lead to degeneration of the muscular fibres, and consequent loss of propulsive power.

10. The custom of *taking purgatives* is in some cases responsible

for confirmed constipation, because of overstimulation and consequent loss of response to normal excitation.

11. A special *loss of power in the colon*, mainly at the sigmoid flexure, is not infrequently encountered, leading to a large accumulation of hard rounded masses in this locality. This may result from long-continued inflammation of the mucous lining, dilatation of the colon, or chronic dysentery or ulceration; and occurs also in connection with uterine disease, hysteria, and neurasthenia. It may be found in very old people, demonstrable lesions being absent.

12. Constipation may be due to a *contracted state* of the small and large *intestine* (spasmodic constipation), the inhibitory fibres of the splanchnic being irritated, as in hysteria, ovarian or uterine disease, and chronic plumbism; and requiring sedatives and antispasmodics rather than laxatives.

13. The presence of *hypertrophied* and *fibrotic rectal* (Houston's) *valves*, whereby the descent of the fæces is obstructed, may be responsible for obstinate constipation. This, perhaps, does not occur as frequently as some writers would have us believe.

14. Acute intractable constipation should always suggest an examination of the various sites of *hernia* before concluding that an intra-abdominal obstructive cause, instead of hernial strangulation, is responsible for the failure of laxatives.

15. The most important and serious condition which may be indicated by constipation is some form of *intestinal obstruction*. Obstinate constipation not yielding to appropriate remedies should always arouse suspicion, especially if associated with vomiting, distention, and abdominal pain. Such symptoms coming on abruptly are very suggestive of acute obstruction. In the chronic varieties the constipation is gradual in its coming, in some cases extremely variable, and may be interspersed with acute exacerbations. It should be remembered that the fæcal accumulation, especially in obstruction from impaction, may become tunnelled, and a deceptive regularity of the bowels, or even a diarrhœa, be present, requiring careful palpation of the abdomen and a digital examination of the rectum.

B. DIARRHŒA

Diarrhœa may be acute or chronic. The stools, more or less fluid in consistence, vary in number from 3 or 4 in 24 hours to the almost continuous purging of cholera. According to its cause it may or may not be attended by fever, abdominal pain, rumbling, and distention.

Excessive peristalsis with abnormal fluidity of the stools may result from one or all of three things: (*a*) Increased irritability of the nervous mechanism, so that it responds with unusual vigour to stimulation. (*b*) The presence of abnormal or irritating substances in the intestine or the action of other unaccustomed stimuli upon the nerves. (*c*) A hyperæmic condition of the intestinal mucosa with hypersecretion or transudation.

Without attempting to recapitulate in detail all of the etiological possibilities of the symptom, diarrhœa, the following paragraphs present those which are of diagnostic importance:

1. An acute diarrhœa with 2 to 8 watery stools, without pain or nausea, may be caused by *psychic influences*, as in the examination diarrhœa of students. It is commonly spoken of as nervous diarrhœa.

2. An acute, watery diarrhœa of brief duration, with or without pain, may be an "*intestinal crisis*," of locomotor ataxia, exophthalmic goitre, or movable kidney.

3. A persistent diarrhœa occurring in the early morning, 2 or 3 stools being passed at short intervals, with freedom for the rest of the day, and attended with neurasthenic symptoms, is the "*morning diarrhœa*" of Delafield and others.

4. Paroxysmal diarrhœa, preceded and accompanied by much pain, usually in the left iliac fossa, terminating with the discharge of strings or membranes composed of mucus, and occurring in an individual, usually a woman, who presents neurasthenic or hysterical symptoms, constitutes the disease known as *mucous colic* or membranous enteritis.

5. Sudden diarrhœal attacks occurring at intervals of days or weeks, as well as a chronic diarrhœa, may be due to *hysteria*, but caution should be exercised in making this diagnosis before excluding other possible causes.

6. It is a good rule to examine the urine in all cases of chronic, or repeated acute, diarrhœas, as they may be vicarious efforts to lessen the uræmia of nephritis, or be due to the secondary intestinal catarrh of the same disease.

7. By far the most common cause of diarrhœa is some form of *enteritis*, catarrhal, croupous, or ulcerative.

The etiological factors of catarrhal enteritis (*q. v.*) are various—e. g., sudden changes in the temperature, hot summer weather, too much food or indigestible food, the presence of ptomaines or toxines (resulting from the activity of micro-organisms), or inorganic toxic substances (arsenic, antimony), the profound exhaustion resulting from Addison's disease, pernicious anæmia, syphilis, and cancer, or chronic portal congestion.

8. An acute diarrhœa with frequent large serous stools, usually

with severe vomiting, is indicative of cholera infantum, cholera morbus, cholera Asiatica, ingestion of poisonous mushrooms, or acute antimonial or arsenical poisoning. In poisoning by arsenic the stools are generally bloody. More rarely there may be serous, scalding stools in cancer of the rectum, and very watery, almost serous, evacuations may be indicative of an unusually severe catarrhal enteritis or the gastro-intestinal form of epidemic influenza.

9. An acute diarrhœa, the stools at first fluid-fæcal, becoming small, bloody, and containing large amounts of mucus, or composed entirely of gelatinous mucus, and voided with great tenesmus, is characteristic of acute dysentery. Tenesmus, with frequent passing of a small amount of mucus, may be due to acute inflammation of the rectum (proctitis), syphilitic ulceration of the rectum, and cancer of the rectum or sigmoid flexure. Tenesmus and bloody stools in children should invariably cause a suspicion of intussusception.

10. Fissure of the anus and stricture of the rectum may produce a diarrhœa from irritation, either direct or reflex, due to accumulated fæces.

11. It should be remembered that while constipation is usually present in *appendicitis*, yet sometimes in adults, and not infrequently in children, a diarrhœa may be one of the initial symptoms.

12. In considering diarrhœa as a symptom, the possibility of its occurrence as an indication of *constipation* should always be borne in mind. An accumulated mass of fæces may become channelled, or the presence of separate hard rounded masses in the colon or its sigmoid flexure may cause colitis with frequent evacuations of scybala coated with mucus and sometimes bloody. The diagnosis is to be made by palpation of the abdomen and digital examination of the rectum. This condition may be present in the latter stages of typhoid fever, and the febrile temperature thus be unduly prolonged.

13. It is evident that diarrhœa is a symptom of wide affiliations. As a rule, the associated symptoms are such as to explain its occurrence. The cases in which the clinician is particularly liable to fall into the error of supposing an acute or chronic catarrhal enteritis to be the only existing condition are the diarrhœa of constipation, cancer of the rectum, and intussusception or appendicitis in children.

C. INCONTINENCE OF FÆCES

The discharge of fæces is regulated by a centre in the lumbar enlargement of the cord with afferent fibres from the rectum and efferent fibres to the sphincter ani. The lumbar centre may be inhibited or helped to a considerable extent by voluntary impulses from the brain. So long as the rectum is empty the sphincter is closed, largely by its own elasticity and tonus. When fæces enter the rectum, sensory impulses pass to the lumbar centre and the brain, and, according to circumstances, the reflex act of defecation is either resisted or facilitated by the will.

Incontinence of fæces, perhaps only for fluid or semifluid stools, may be present as a result of impaired intelligence in the insane, in idiots, and delirious persons, and in all diseases attended with coma or profound prostration. It is sometimes a symptom of paralysis of the sphincter ani, or an injury of the pelvic floor, or of disease of the lower portion of the rectum which interferes with sphincteric contraction. Spasmodic contractions of the rectum and the abdominal muscles may forcibly expel the fæces without volition on the part of the patient.

Incontinence of fæces, therefore, may occur in the following conditions: Apoplexy, uræmia, epileptic coma, meningitis, insolation, hydrocyanic-acid poisoning, shock, typhoid fever, the typhoid state, cholera morbus, cholera infantum, cholera Asiatica, dysentery, myelitis and some other diseases of the spinal cord, the grave form of chorea; tetanus and strychnine poisoning, or other diseases of which convulsions are a manifestation; laceration of the perinæum involving the sphincter ani, or surgical overstretching of the latter, and cancerous or syphilitic infiltration of the rectum. In medical practice incontinence of fæces is most frequently encountered in the typhoid state.

D. PAINFUL DEFECATION

Pain during defecation may or may not be conjoined with rectal tenesmus. Very severe pain after going to stool, which may last for several hours and then gradually subside, only to be renewed at the next evacuation of the rectum, the stools being at times streaked with blood, is very significant of an anal fissure. Pain may be due to the passage of a large, hard mass of fæcal matter or to the presence of hemorrhoids, prolapsus ani, or an inflamed and swollen prostate gland. Great rectal discomfort is not infrequently explained by the presence of salpingitis and pelvic peritonitis, and may result from cancer of the cervix, a retroflexed tender uterus, coccygodynia, or cancer of the rectum.

Obviously, the principal diagnostic requirement of pain occurring with defecation is a digital and, if a sufficient cause does not appear without it, an instrumental examination of the pelvic contents.

E. RECTAL TENESMUS

A persistent inclination to defecate, accompanied by painful and largely ineffectual efforts so to do, constitutes rectal tenesmus, a symptom of much diagnostic importance. In conjunction with colicky pain and stools of bloody mucus, it is characteristic of the various forms of dysentery, and is found to a much milder degree in severe catarrhal enteritis. It may be present in the diarrhœa caused by irritant poisons, especially cantharides. Associated with vesical tenesmus it is indicative of a calculus in the urinary bladder or a symptom of mucous colic (membranous enteritis). Rectal tenesmus may also be caused by impacted fæces, worms, or a foreign body in the rectum, hemorrhoids, and proctitis. It is very commonly present in cases of rectal polypus, adenoma, and cancer of the rectum. In infants or children tenesmic stools, consisting of bloody mucus without fæcal matter, are significant of intussusception. Rarely rectal tenesmus may indicate an enlarged and retroflexed uterus.

F. THE CHARACTER AND ABNORMAL CONTENTS OF THE STOOLS

Valuable information may be obtained by a naked-eye inspection of the stools, this inspection relating to their odour, reaction, shape, consistence, colour, and contents. The microscopical examination of the fæces (*q. v.*) is considered elsewhere.

1. The Shape of the Stools.—(*a*) A stool of normal consistence, cylindrical in shape but of unusually small calibre, is most commonly significant of the spastic form of constipation, or prolapsus ani, or an annular stricture of the rectum. More rarely a stool of this shape is indicative of the early stage of an intussusception.

(*b*) Ribbon-shaped or flattened stools of normal consistence arouse a suspicion of the existence of stricture or cancer of the rectum. More rarely this finding may indicate ischio-rectal or prostatic abscess or great enlargement of the prostate, large hemorrhoids, prolapse of the uterus, or perhaps spasm of the anus and lower bowel. The thick scar of an old anal fissure is a not infrequent cause of a partially flattened stool.

(*c*) Scybala—roundish masses of hardened fæces—are found in many cases of habitual constipation, especially when atony and sacculation of the colon coexist. They are of common occurrence in gastric ulcer, gastric dilatation, and cancer of the rectum. They are apt to be present in the constipation resulting from the use of opium, and small round balls may be shot out of the anus during the straining efforts attendant upon dysentery.

2. **The Odour and Reaction of the Stools.**—A sour odour, as compared to the fæcal odour of the healthy adult stool, is normal for the nursing infant. An unusually offensive but not putrid odour is found as a symptom of jaundice or other conditions in which there is a deficiency of bile in the intestine; in acute indigestion, acute enteritis, typhoid fever, erysipelas, rachitis, and occasionally in ordinary constipation. A musty, mousy odour is associated with cholera infantum. Ill-smelling stools are caused by the taking of sulphur or the eating of eggs, with the consequent formation of sulphuretted hydrogen. Stools with a foul and putrid odour are suggestive of syphilitic or carcinomatous ulceration of the rectum or gangrenous dysentery.

There is at present little of clinical value to be learned from the *reaction* of the stools. It is normally faintly acid in the infant, and an alkaline reaction in this case is of some significance with reference to the nature of the fermentative processes which are under way in the intestine. It may be either acid or alkaline, according to the variety of micro-organisms which are present, in the diarrhœas of infants and children.

3. **The Consistence of the Stools.**—(*a*) Semifluid or fluid fæcal stools are found in the great majority of cases in which diarrhœa is a symptom, embracing the ordinary diarrhœas and the various forms of catarrhal enteritis. At the onset of the attack the solid or semisolid contents of the rectum are discharged; but very shortly, owing to the increased amount of fluid poured out by the intestinal mucous membrane, the stools become fluid or semifluid in consistence, still, however, retaining a fæcal character. Pasty stools are usually occasioned by considerable admixtures of mucus.

(*b*) Serous stools, composed of fluid without fæcal matter, are of considerable diagnostic importance. Such stools are significant of cholera Asiatica, cholera morbus, cholera infantum, and poisoning by antimony. Arsenical poisoning also may be attended by a serous diarrhœa, and an unusually severe acute catarrhal enteritis may present this variety of stool. In cancer of the rectum the evacuations may be small, serous, and frequent. Poisoning by the non-edible mushrooms is another possible but infrequent cause of serous stools.

4. **The Colour of the Stools.**—The colour of the stool may be altered by the kind of food which has been eaten. In an exclusive milk diet the stools are light yellow, as indeed they may be if starchy foods form a large proportion of the ingesta. Fruits with deep-red juice and red wines darken the stools.

(*a*) Clay-coloured stools are usually significant of a deficient amount of biliary colouring matter in the intestine, due either to an obstruction to the flow of bile from the common duct or to an impaired bile formation. Excess of fat is a common cause of whitish stools. Bile may be present and yet be in the form of the colourless urobilin. Obstruction of the common duct by a calculus, or of the large and small ducts by inflammation and consequent swelling of their lining membrane, are the most frequent causes of clay-coloured stools. More rarely a tumour or a movable kidney, by pressing upon or causing kinking of the common duct, may be responsible for the lack of colouring matter. Light-coloured stools may be significant of impaired bile formation resulting from anemia, rachitis, or chronic lead poisoning, as well as cancer, cirrhosis, or amyloid disease of the liver. Such stools occur also in connection with acute yellow atrophy of the liver and phosphorus poisoning.

(*b*) Green stools, when occurring in infancy, are sometimes caused by the growth of a colour-forming bacterium in the intestine. The greenish "calomel," or "bilious," stools are sufficiently familiar. Most commonly this colouration is due to the hurried passage of bile through the intestines and the appearance of unchanged biliverdin in the discharges. After infancy this is a more significant finding. (See Microscopic Examination of Stools.) An unusual amount of bile may be present in any case of chronic diarrhœa or acute enteritis.

(*c*) Black stools may be due to the administration of iron, manganese, or bismuth. "Tarry" and red stools are considered in connection with the abnormal constituents of the fæces.

5. **Abnormal Contents of the Stools.**—The stools may contain undigested food, blood, mucus, pus, membranes, fat; calculi from the gall bladder, intestines, stomach, salivary glands, or tonsils; foreign bodies, intestinal parasites, exfoliated polypi, and segments of exfoliated intussusceptions and necrotic sloughs.

In searching for solid bodies in the stools the following method is recommended by Lilienthal: "Take a loop of telegraph wire a few inches greater in diameter than the entire top of the closet seat, and fasten to this a bag of at least two thicknesses of dressing gauze or mosquito netting. The bag may be sewn to the wire or simply held by safety pins, but it should be made very full, so that when the hoop is in place the wire shall be well below the level of the seat and out of the way, while the bag shall hang down into the water at the bottom of the bowl. If the patient is not confined to bed he defecates into the closet, and then simply opens the water valve often enough to wash away all soluble and amorphous matter, while solid bodies will be left in the bag.

Dr. Dudley B. Roberts has devised a useful stool sieve which he describes as follows: "The lower part of the apparatus is a band of tinned copper, to the bottom of which copper sieve is fastened in the form of a bowl. To catch complete portions of thin stools, a bowled copper plate may be placed over the sieve. The lid has a perforated plate on its under side which distributes the water in fine streams over the specimen. A rubber pipe is fastened to the pipe seen projecting through the lid, and water is thus conveyed from a nearby faucet, or from a faucet introduced in the water pipe leading to the water-closet tank.

"When the apparatus is used the stools leave no objectionable odour about the office laboratory. It is also an advantage to have the dejections received directly, thus saving transportation and transference from one vessel to another. The sieve is cleansed by detaching the supporting arms and placing it inverted in the water-closet bowl so that a stream of water may be directly applied."

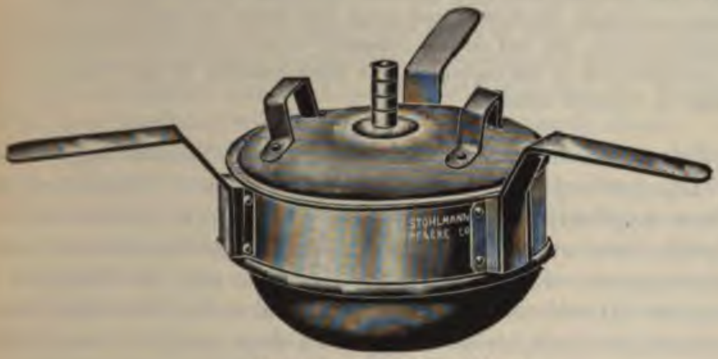


FIG. 24.—Roberts's stool sieve.

(*a*) *Undigested portions of food* (lienteric diarrhœa) may be indicative of an acute dyspeptic diarrhœa, or of imperfect mastication or digestion. In fevers the appearance of milk curds is an important sign of overfeeding, and a similar discovery in the stools of an infant is a reliable sign of an improper quantity or quality of food, and not infrequently heralds an acute attack of gastro-intestinal disturbance.

(*b*) *Blood in the stools* may show itself in its characteristic red colour, or as "tarry" stools (*melæna*), according to the source of the bleeding and the length of time it has remained in the intestine. If it has its origin in the upper portion of the digestive tract (stomach or small intestine), it is altered by the action of the digestive fluids so as to assume a black or brownish-black appearance, or may resemble coffee grounds. If there is active peristalsis, or if the blood is in large amount, it may, even if coming from the upper part of the small intestine, pass so rapidly along that it issues almost unchanged with the stool. As a general rule, however, bright blood in the defecation comes from the lower part of the bowel, particularly from the rectum. The more thoroughly the blood is mixed with the fæces, and the more marked the tarry appearance, the greater is the probability of its origin from the upper portion of the alimentary canal.

If there is any question as to the presence of blood in the stools, it may be identified either by the microscope or by chemical methods, or both. Under the microscope the red cells may be distinguished, or if these cells are disintegrated, brownish-red masses may be seen which are composed of amorphous hæmatoidin. In a certain proportion of cases the hæmatoidin may be found in crystals of the characteristic rhombic shape. In the absence of well-preserved corpuscles or crystals, the hæmin test (page 133) must be applied. As a possible source of error, the red colour imparted to the stools by the administration of hæmatoxylon or the ingestion of a large quantity of red fruits or fruit juices may be borne in mind.

1. *Tarry stools* may be indicative of a gastric hemorrhage due to ulcer, cancer, or ruptured varicose esophageal veins. The blood may have been originally derived from lungs or nose, a certain amount having been swallowed, so that any case of epistaxis, adenoid operation, hæmoptysis, or (especially) hæmatemesis may be followed by tarry stools. Duodenal ulcer and multiple ulcers of the small intestine (syphilitic, dysenteric, or typhoid) may also be responsible. Portal obstruction from any cause, particularly hepatic cirrhosis or cancer, may be attended by melæna; so also may acute yellow atrophy of the liver, purpura hæmorrhagica, hæmophilia, and leucæmia.

2. *Bloody stools* in which the red colour of the blood has not been destroyed by the action of the digestive fluids may occur in any of the conditions enumerated above as causing tarry stools, provided the blood is in sufficient amount; and, similarly, some of the diseases and conditions about to be mentioned as possible causes of bloody stools may give rise to coffee-ground or tarry defecations.

A frequent source of blood in the stools is a bleeding hemorrhoid. Cancer of the rectum, fissures, and ulcers (especially syphilitic) may give rise to blood-covered stools, particularly if the fæces are dry, hard, and lumpy. Indeed, the latter condition alone, in the absence of rectal disease, may abrade the mucous membrane of the rectum so as to cause bloody fæces. In children and feeble-minded persons the possible presence of foreign bodies should be borne in mind. An acute proctitis is an additional rectal source of blood-stained stools. A polypus of the rectum, if it becomes eroded, may bleed profusely.

Strangulated hernia and, in a child, intussusception should be suspected, the latter especially, in the presence of blood-stained stools composed principally of mucus and attended with tenesmus. An acute and unusually severe colitis in children may manifest itself by blood-streaked passages. The various forms of dysentery may present stools which have the appearance of rusty-red fleshy lumps. Cancer or ulceration of the large or small intestine, from whatever cause, as well as the perforation resulting from such ulceration, may explain the appearance of blood in the fæces. According to Grainger Stewart, amyloid disease of the intestine may cause hemorrhage. Corrosive poisons, especially arsenic, may determine the presence of streaks of blood in the passages.

The rupture of an aneurism of the abdominal aorta into the alimentary canal serves to explain some cases of large hemorrhage from the bowel. Aneurism or thrombosis of the superior mesenteric artery, to which attention has been particularly directed by Watson and Elliot, is a condition which produces tarry or bloody stools.

Engorgement of the portal circulation from cancer or cirrhosis of the liver, or as the result of valvular disease of the heart, pulmonary emphysema, or portal thrombosis, demands consideration as a cause of intestinal hemorrhage. In jaundice, whatever its origin, blood may be contained in the stools, as well as in phosphorus poisoning. In various infectious diseases, among them yellow fever, pernicious malarial fever, dengue, acute yellow atrophy of the liver, septicæmia, pyæmia, and typhoid fever (usually from an intestinal ulcer), blood in varying quantity and colour may be a constituent of the stools.

Intestinal hemorrhage may be a symptom of leucæmia, hæmophilia, purpura hæmorrhagica, and scurvy. In the occasional sudden diarrhœal attacks of exophthalmic goitre, bloody mucus is sometimes present. Intestinal hemorrhage not due to tuberculous ulceration may appear as an intercurrent event in pulmonary phthisis. Finally, injuries of the abdomen and intestinal parasites may give rise to bleeding from the bowel.

3. *Most Frequent Causes of Blood in the Stools.*—It is evident that a decision as to the origin of the blood in a given case can be reached only by a careful consideration of the history and symptoms, as well as a painstaking physical examination. It may be of service to bear in mind that the most frequent causes of hemorrhage from the bowel are hemorrhoids, typhoid fever, colitis, and cancer of the colon; that the most frequent causes of *large* intestinal hemorrhages are typhoid fever, portal engorgement from disease of the heart or liver, hæmophilia, purpura hæmorrhagica, and rupture of an aneurism; that the most frequent causes of *small* hemorrhages from the bowel are hemorrhoids, injury to the rectum or a rectal fissure by the passage of fæces, ulceration (syphilitic or cancerous) of the rectum, and intussusception in infants and children.

"Occult" blood is a term applied to blood in the fæces (or stomach contents) which is in such small quantity that it can not be recognised by the eye, or is so altered as not to be identified by the microscope. Its presence (to be determined by the Boas-Weber test, p. 133) possesses the same significance as macroscopic gastric or intestinal hemorrhage. It is said (BOAS) to be found *constantly* in carcinoma of the gastro-intestinal tract; *intermittently* in gastric or duodenal ulcers; *occasionally* in stenosis (organic or spasmodic) of the pylorus; and is *absent* in gastritis, hyperchlorhydria, hypersecretion, and neuroses. In Boas' hands the presence or absence of occult bleeding has been most valuable in differentiating between painful gastric neuroses and ulcers of the stomach or duodenum.

The extent of an intestinal hemorrhage, and on occasion its occurrence before appearing in the stools, may be judged by the constitutional symptoms, which are those of internal hemorrhage (*q. v.*).

(*c*) ***Mucus*** in the fæces, aside from a thin coating on hard lumps, is an evidence of pathological irritation or inflammation. An increased amount is ordinarily significant of a catarrhal process in some portion of the intestine. If in considerable quantity, forming a thick coating upon the fæces, or if the entire defecation is composed of mucus, it indicates inflammation of the mucous membrane of the *rectum* or *large intestine*. If in smaller quantity, and contained in thin stools, or mixed and thoroughly diffused through fæcal matter, it indicates a catarrhal process in the *small intestine*. The more finely divided the mucus and the more thoroughly intermixed it is with the fæces, the higher is its origin, and conversely. Mucus-containing stools constitute a symptom of the various forms of dysentery, entero-colitis, proctitis, and impaction of fæces. The characteristic stool of intussusception is composed of bloody mucus. Membranes, fragments, or tubular casts composed of altered mucus are voided in the disease known as membranous enteritis (*mucous colic*). Allied to this disease are the attacks of mucous diarrhœa which may occur during the course of exophthalmic goitre, and in connection with movable kidney and enteroptosis.

(*d*) ***Pus*** in the fæces, if in large quantity, is indicative of the rupture of an abscess (pelvic, periproctitic, or perinephritic) into the alimentary canal. In smaller quantities it is significant of dysentery, enteritis, proctitis, and syphilitic or cancerous ulceration of the rectum or colon. It may be derived from the urethra or vagina as a result of a severe inflammation of their lining membrane.

(*e*) ***Membranous shreds,*** not composed of mucus, are found in rare cases as the result of a superficial necrosis and sloughing of the intestinal mucous membrane which may occur in the course of acute proctitis, cancer of the colon, dysentery, and relapsing fever.

(*f*) Fatty stools, which may be recognized by their oily, greasy appearance, are found in association with obstructive jaundice because of the deficient absorption of hydrocarbons resulting from the absence of bile. Indigestion or overfeeding in infants may give rise to fat-containing stools. In the absence of jaundice or (in infants) impaired digestive capacity, the occurrence of a fatty diarrhœa should suggest cancer of the pancreas, or pancreatic calculi impacted in the pancreatic duct.

(*g*) *Gallstones*, if suspected, must be searched for by breaking up the fæces in a sieve while pouring water over the mass. Multiple stones formed in the gall bladder are faceted, especially if ejected soon after their emergence from the common duct and before any solvent action is exerted upon them by the intestinal fluids. Very rarely a gallstone is found which, being very small, non-faceted, and crumbling easily, may be conjectured to have been formed in the intra-hepatic ducts. Gallstones are most commonly composed of cholesterin, in which case they float in water; less frequently of inspissated bile or calcareous salts, and are heavier than water. The other calculi found in the stools are very rare.

(*h*) *Foreign bodies*, when found, usually indicate childhood, mental infirmity, a depraved taste, or a professional freak. Conversely, suspect foreign bodies with such patients.

(*i*) *Intestinal Parasites* (see also Index).—*Ascaris lumbricoides.*—If a cylindrical worm, pointed at both ends, of a yellowish or slightly reddish colour, and varying from 10 to 30 centimetres (4 to 12 inches) in length, is found in the stools, it is the *Ascaris lumbricoides*, or roundworm (Fig. 25). The patient is usually a child and the worm is the most common human parasite. On inspection the worm is seen to be transversely striated and to possess four longitudinal bands. It exhibits considerable motility, and this power of migration gives rise, fortunately in rare instances, to serious consequences. Thus this parasite has crawled from the upper part of the small intestine, which is its ordinary habitat, into the common bile duct and the intra-hepatic ducts; perforated the wall of the intestine and caused peritonitis; crawled into the stomach, esophagus, pharynx, and thence into the Eustachian tube, emerging from the external ear. Passing into the larynx, the worm has caused death by asphyxia; into the trachea, pulmonary gangrene. As a general rule, only one or two are present, but a considerable number may exist in the same

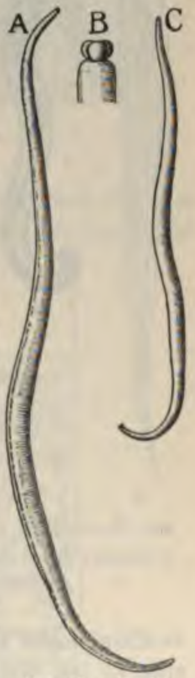


FIG. 25.—Ascaris lumbricoides.

A, female; *B*, head; *C*, male.

patient. Great masses of these worms have caused intestinal obstruction. They may or may not give rise to symptoms.

Oxyuris vermicularis.—White, threadlike worms, 4 to 10 millimetres ($\frac{1}{6}$ to $\frac{3}{8}$ inch) in length, found in the fæces, or at the anus, or in the vagina, are the *Oxyuris vermicularis*, the thread or seat worm (Fig. 26). They occur in patients of any age, most commonly in children. Their ordinary habitat is the rectum and colon.

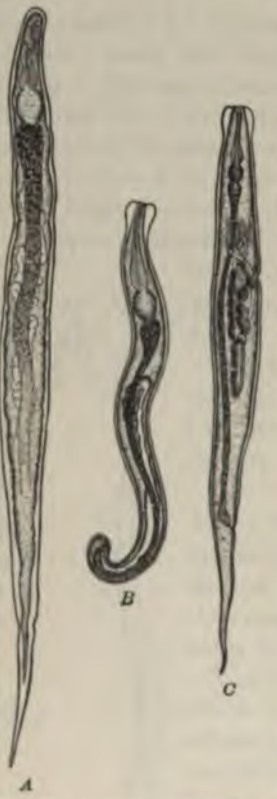


Fig. 26.—Oxyuris vermicularis. *A*, female; *B*, immature female; *C*, male.

Tapeworms.—1. *Tænia saginata.*—White or yellowish-white, flattened, oblong bodies, 10 millimetres ($\frac{3}{8}$ inch) wide, 18 millimetres ($\frac{3}{4}$ inch) in length, are the segments or proglottides of a ribbon-shaped, jointed parasite of the class of intestinal cestodes, tapeworms (Fig. 27). In this country there is a very great probability that it is the beef tapeworm, *Tænia saginata* or *mediocanellata*.

The *T. saginata* varies from 12 to 20 feet in length. The head is about 2 millimetres ($\frac{1}{12}$ inch) in diameter, square in shape, and possesses four large pigmented sucking disks, *without hooklets*. The neck is long and threadlike, gradually enlarging and becoming segmented, the segments reaching their fullest development at or about the 450th, and then containing ripe ova. Each segment contains both male and female sexual organs. If the mature segment is pressed between glass slides, the uterus is seen as a median line with 15 to 35 lateral branches. The period from the time at which the worm is swallowed and attaches itself to the mucous membrane of the intestine to the time at which ripe segments begin to pass from the rectum is from 3 to 3½ months. The segments of this worm possess motility, and are not infrequently found in the clothing, having extruded themselves from the rectum between the acts of defecation. The mature proglottides are, under favourable circumstances, ingested by beef cattle, the ova are set free, and the embryos, passing from the stomach into the muscles, liver, brain, or eye, become encysted larvæ or cysticerci.

2. *Tænia solium.*—If the segments are somewhat shorter and narrower, 8 millimetres (1/3 inch) wide and 1 centimetre (2/5 inch) in length, and the head of the worm is but the size of the head of a pin, and on examination with a power of 40 diameters is found to be

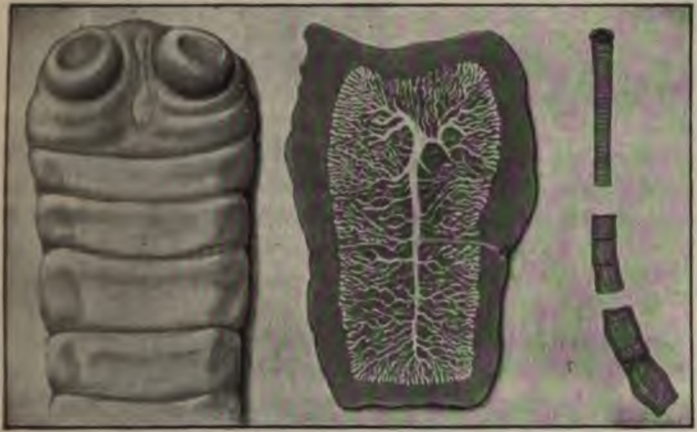


FIG. 27.—Scolex and segments of *Tænia saginata.*

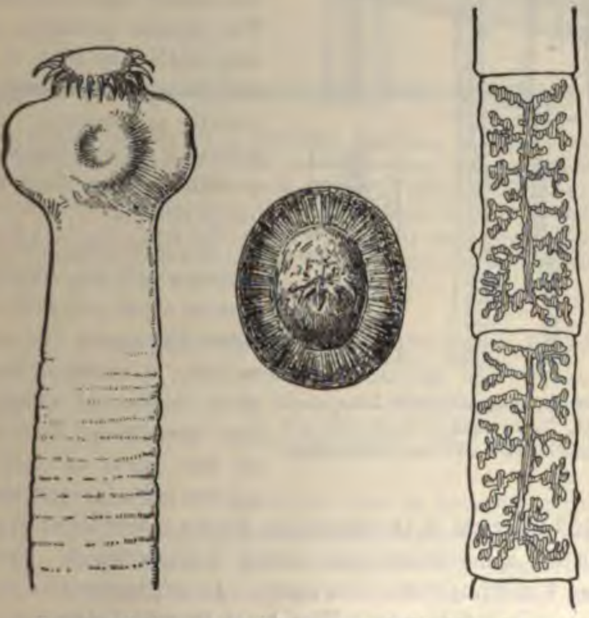


FIG. 28.—Scolex, egg, and ripe segments of *Tænia solium.*

provided *with hooklets* as well as suckers, it is the pork tapeworm, the *Tænia solium* (Fig. 28). This worm varies from 6 to 12 feet in length, and, with the exceptions noted, is similar in appearance to *T. saginata.* The uterus possesses fewer (8 to 14) lateral branches than the previous variety. It is eaten by and becomes encysted in the hog, which is then referred to as "measled." It is the common tapeworm of Europe.

If raw or imperfectly cooked beef or pork is eaten by men, the still living larvæ are liberated, and, attaching themselves in the intestine, begin the adult stage of existence.

T. saginata and *T. solium* are the only tapeworms of clinical and pathological interest which are ordinarily found in this country.

Bothriocephalus latus.—This worm (Fig. 29), most common in the lake regions of Europe, varies from 15 to 30 feet in length. The head, which measures 2 millimetres long by 1 millimetre broad, has two grooves, probably suckers, upon each of the broader surfaces. A short neck passes at once into the body segments. The full-grown proglottides are nearly square and show the sexual organs in the centre. The uterus presents as a median dark line with 4 to 6 lateral branches, causing a star, or rosette appearance. Infection probably occurs through eating insufficiently cooked fish, especially the pike.

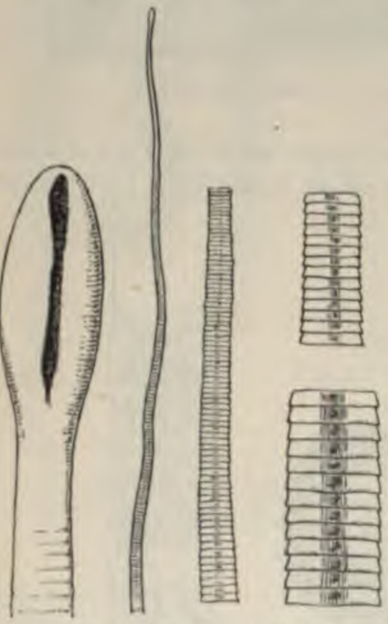


FIG. 29.—Head of *Bothriocephalus latus*, much magnified. To the right the head, neck, and proglottides of the same worm about natural size.

Ankylostoma duodenale, a member of the *Strongyloides*, is most often met with in Egypt, some European states, and Jamaica. A variety, the *Uncinaria americana* (Figs. 30–34), has been identified by Stiles as the cause of many of the severe anemias frequent in the "sand belt" districts of the Southern States. With the exception of the mouth the same description covers both varieties. In length it varies from 6 millimetres in the male to 18 millimetres in the female, both figures being extremes. The head is dorsal, the mouth being armed with one prominent dorsal conical tooth projecting into the buccal cavity. This serves to distinguish it from *Ankylostoma*

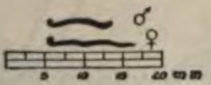


FIG. 30.—Showing actual size of male and female *uncinariæ* (A. J. Smith).

duodenale, which has four teeth. In the female the tail tapers bluntly, while in the male it spreads out into a three-lobed bursa. The ova, in which form the parasite is commonly found in the fæces, are oval, and average .03 × .05 millimetres (Fig. 34).

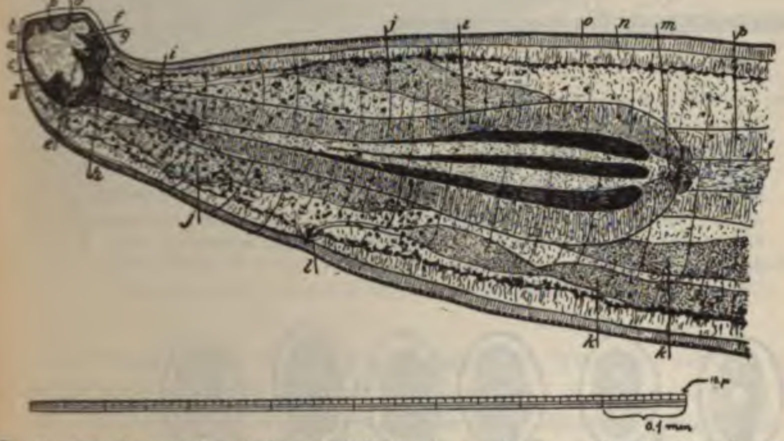


FIG. 31.—Lateral view of cephalic end of *Uncinaria americana*. *a*, Buccal cavity; *b*, oral papillæ; *c*, chitinous capsule; *d*, ventral lancets; *e*, lateral lancets; *f*, dorsal conical tooth, with (*g*) its posterior extension reenforcing part of capsule; *h*, interior of esophagus; its chitinous wall continuous with the pharyngeal teeth above; *i*, muscular wall of esophagus; *j*, esophageal glands opening into the pharynx; *k*, cervical glands opening externally at cervical papillæ; *l*, *m*, trilobed valvular opening of esophagus into intestine; *n*, muscular layer of body wall; *o*, cuticle (A. J. Smith).

A variety of other parasites, including *Trichina Spiralis* and *Anguilulla Intestinalis*, occasionally appear in the fæces. The reader is referred to special works for their identification. Trichina has been shown by Brown to be associated with marked eosinophilia (*q. v.*).

(*j*) *Vegetable parasites.*—A generous flora is present in the fæces at all times, but the forms of principal pathological interest include these bacteria: *Spirillum* (of Asiatic cholera); *Bacillus typhosus; Bacillus tuberculosis; Bacillus dysenteriæ* (Shiga's); and *Bacillus aerogenes capsulatus. Bacillus coli communis*, *proteus vulgaris*, and

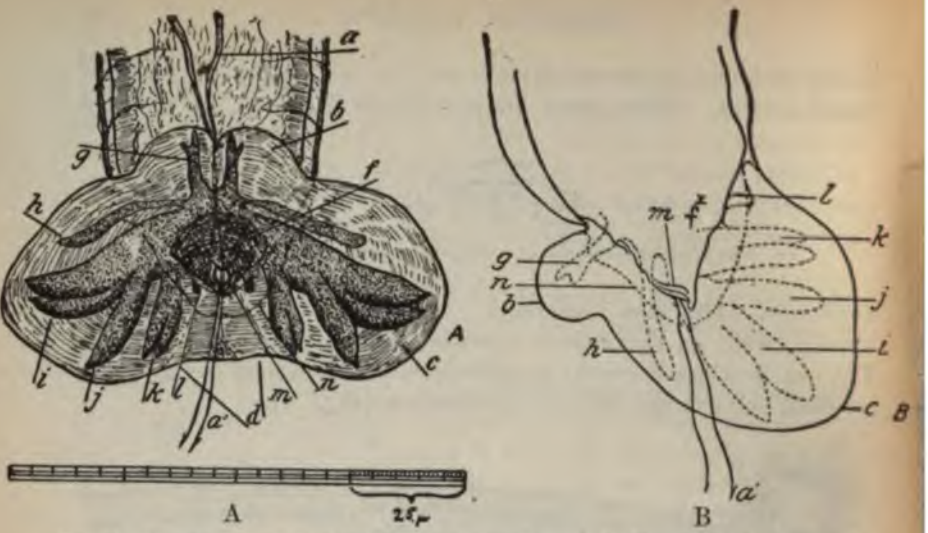


FIG. 32.—Posterior extremity of male *Uncinaria americana*. A. Dorsal view, bursa spread open and dorsal lobe thrown upward over body of worm: *a*, inner end of genital spicules; *a'*, external barbed extremity of spicules; *b*, subdivided dorsal lobe of bursa; *c*, large lateral lobe of bursa; *d*, limits of inconspicuous ventral lobe of bursa; *f*, dorsal aspect of tip of tail within bursa; *g*, bipartite-tipped dorsal ray; *h*, dorso-lateral ray; *i*, divided lateral ray; *j*, ventro-lateral ray; *k*, divided ventral ray; *l*, inconspicuous subventral ray; *m*, opening of cloaca, with chitinous furcula; *n*, cuticle of tail. B. Lateral outline of same; letters as above (A. J. Smith).

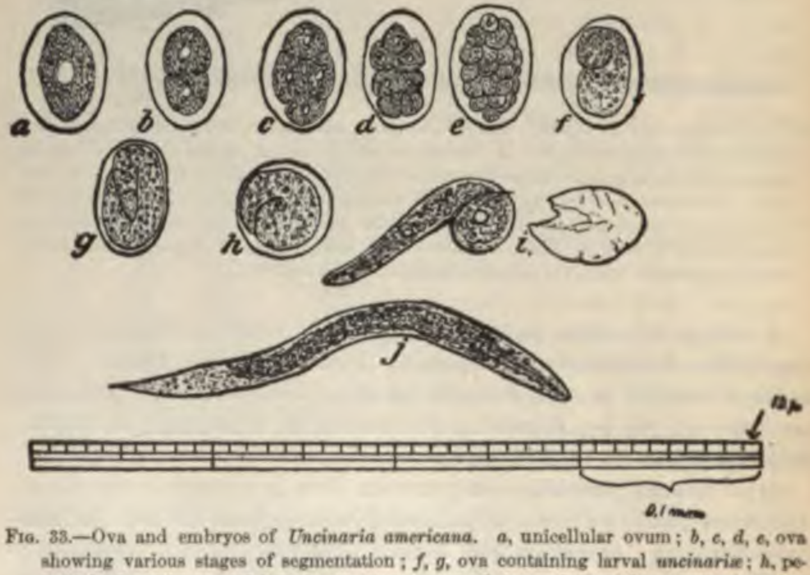


FIG. 33.—Ova and embryos of *Uncinaria americana*. *a*, unicellular ovum; *b*, *c*, *d*, *e*, ova showing various stages of segmentation; *f*, *g*, ova containing larval *uncinariæ*; *h*, peculiarly shaped ovum; *i*, larval worm just emerged from shell; *j*, larva extended after emergence (A. J. Smith).

a few others, occasionally take on pathogenic activity. Their identification belongs to the realm of bacteriology, and the reader is referred to works on that subject for the appropriate methods.

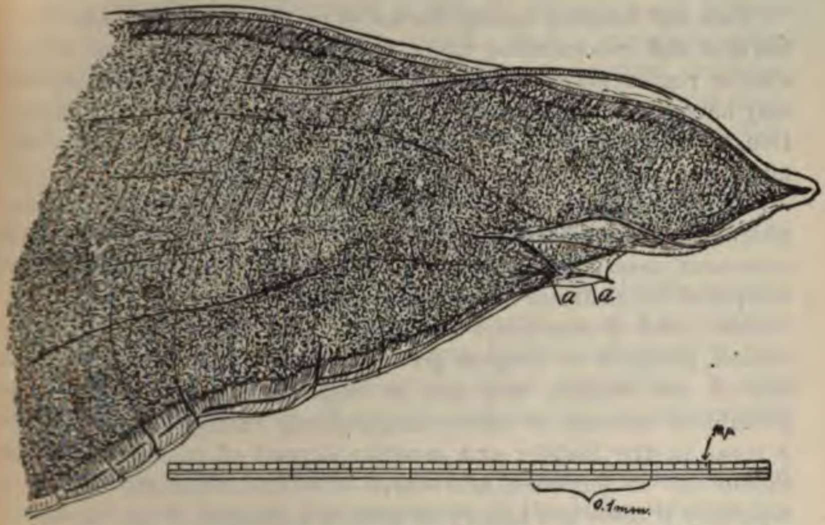


FIG. 34.—Posterior extremity of female *Uncinaria americana*, viewed ventro-laterally, showing anal opening expanded; *a*, anal papillæ, showing small chitinous tips (A. J. Smith).

(*k*) *Polypi* which have become detached from the rectum or intestine may be passed in the fæces.

(*l*) The invaginated *segment of bowel* in intussusception sometimes sloughs away and is passed per rectum.

(*m*) *Necrotic sloughs* from tumours or inflammatory processes involving the intestinal wall sometimes appear and must be differentiated from masses of undigested meat.

V. URINATION

The diagnostic information to be derived from the chemical and microscopical examination of the urine (*q. v.*) is discussed elsewhere. We consider here the significance of abnormalities of urination—i. e., dysuria (painful urination), difficult, slow, or frequent urination, incontinence, retention, and suppression of urine.

Dysuria.—Under painful micturition or dysuria, one includes *vesical tenesmus*, a persistent inclination to urinate, accompanied by painful straining; and *strangury*, the performance of urination by a spasmodic and painful effort—the former term laying a greater stress upon the presence of a continuous desire to empty the bladder.

The diagnostic indications of dysuria relate, with some exceptions, to local disease, as follows:

Pain and burning during the act of urination may be caused by a too acid and concentrated urine, as in some lithæmic cases, or for a similar reason may be symptomatic of acute nephritis. The urine may have become irritant in consequence of the ingestion, or absorption through the skin, of cantharides or turpentine, or such condiments as mustard, pepper, and horseradish.

Cystitis is the most frequent of all the morbid vesical conditions producing severe dysuria. Discomfort in voiding urine, as one of numerous associated symptoms, is not an uncommon subjective complaint in neurotic women (neuralgia of the bladder, irritable bladder), and is sometimes accompanied by vesical pain and spasm. Cancer, polypoid or fungoid growths, tuberculous disease or ulceration of the bladder, may also be suspected, but with much less probability because of their comparatively infrequent occurrence. A stone in the bladder or a sandlike deposit of calculous material should not be forgotten as a source of vesical irritation, the former especially if there is a history of sudden stoppages of the stream of urine, with, in a male, considerable pain in the head of the penis.

Certain urethral conditions also are suggested by dysuria, such as urethritis, simple or gonorrhœal; chancre or old stricture of the urethra; and, in association with variola or varicella, the presence of vesicles in the urethra. The prostate gland, if inflamed, hypertrophied, or cancerous, may be the cause of painful micturition.

In women, dysuria may be indicative of a prolapsed uterus, cancer of the cervix, acute metritis, pelvic peritonitis and abscess, particularly if there are adhesions preventing the complete collapse of the bladder, in which case the greatest pain is felt toward the end of micturition. It is sometimes associated with the various forms of dysmenorrhœa; and vesical tenesmus with a discharge of mucus may occur in connection with paroxysms of mucous colic (membranous enteritis). In a recent case of the latter disease in my wards at the Methodist Episcopal Hospital there was so much vesical tenesmus with a discharge of bloody mucus that suspicions of malignant disease were entertained, but a cystoscopic examination by Pilcher, of the surgical staff, showed nothing but a coating of mucus on the inner lining of the organ. A sudden attack of painful and spasmodic urination may be a vesical crisis of locomotor ataxia.

Inflamed hemorrhoids, perineal abscess, acute proctitis, and acute dysentery are not infrequently accompanied by reflex dysuria and vesical tenesmus.

Difficult or Slow Urination.—Any condition or lesion which narrows the lumen of the urethra or impairs the muscular power of the bladder walls will give rise to slow or difficult urination, and perhaps to a diminution in the size of the stream. Such a symptom, consequently, leads to a suspicion of a tight prepuce, a urethral stricture, an enlarged or inflamed prostate gland, or a loss of tone of the bladder, the latter from overdistention or disease of the nervous mechanism concerned in the act of micturition.

Frequent Urination.—Many of the conditions which are responsible for dysuria (*q. v.*) are also attended by an abnormal frequency of urination, particularly diseases of the bladder.

The cause of a too frequent desire to empty the bladder is occasionally found in some abnormality of the urine—e. g., a too concentrated urine, azoturia (an excess of urea), lithuria (an excess of uric acid and urates), and the presence of irritant substances. So also with the opposite condition of an abnormal increase in the amount of urine (*q. v.*).

A frequent desire to urinate is a reflex symptom of the passage of a renal calculus through the ureter, and if the stone becomes impacted in the ureter this annoying symptom continues. It is also associated with some cases of pyelitis. Finally, frequent urination may precede an ague fit, accompany an attack of angina pectoris, and constitute a symptom of sunstroke.

The more or less constant dribbling of urine in some cases of retention should not be mistaken for simple frequency of urination. Palpation, percussion, and, if necessary, the catheter should be employed in order to determine that the bladder is empty.

Incontinence of Urine.—An inability to control the escape of urine from the bladder, or the passing of it unconsciously, is due either to contraction of the detrusor (longitudinal) muscular layer of the bladder, or to relaxation or paralysis of the sphincters. If both are paralyzed, it leads to retention *plus* incontinence, manifested by distention of the bladder with constant dribbling. If the compressor muscle is paralyzed, and the sphincter is contracted or the urethra obstructed, retention alone ensues.

Involuntary or unconscious passing of urine may be the direct result of all conditions which annul or interrupt the ordinary and normal voluntary control exercised by the brain over the act of micturition. These conditions may pertain (1) to the brain itself, or (2) to that portion of the cord which puts the brain in relation with the vesical centres.

(1) The conditions or lesions which annul conscious cerebral activity are: all forms of coma (apoplectic, alcoholic, epileptic, especially the nocturnal form of the latter); idiocy and some varieties of insanity; sunstroke; shock; and the poisons of certain infectious diseases, such as severe diphtheria, typhus and typhoid fevers, and the typhoid state from whatever cause.

(2) The lesions which interfere with conduction to and from the vesical centres in the sacral segments are: injuries and tumours of the cord, intraspinal hemorrhage, transverse myelitis, spinal meningitis, and locomotor ataxia, provided that the lesion does not destroy the vesical centres. If the reflex arc is abolished by the same lesions affecting the centres, total paralysis of the bladder, with retention and dribbling, will result. If the paralysis be partial there is partial retention, with occasional voiding or accidental escape of the urine with any sudden muscular effort. From a diagnostic standpoint, the presence of vesical disturbance militates against amyotrophic lateral sclerosis, poliomyelitis, and multiple neuritis, as distinguished from myelitis and locomotor ataxia, in which bladder symptoms are more or less prominent.

Incontinence of urine may be due to an increased reflex excitability of the nervous mechanism, of which nocturnal enuresis is an example. Under such circumstances, and especially if an unusual irritation is present, the urine is passed involuntarily, usually during sleep, when the normal cerebral control is in abeyance; sometimes during waking hours when the mind is profoundly engrossed. The sources of local irritation which should be sought for are ascarides (seat worms), cystitis, vesical calculus, phimosis, contracted meatus, balanitis, and concentrated or diabetic urine. It may also be caused by general weakness of the nervous system, or the reflex irritation due to dentition. In women a relaxation of the pelvic floor, associated with cystocele, is a common cause of incontinence.

Increase of the intrapelvic pressure, combined with lack of tone of the vesical sphincters, may produce incontinence, as in the leakage of urine during a paroxysm of pertussis or other cough, sneezing, laughing, or muscular effort. Hydrocyanic-acid poisoning is attended with the involuntary discharge of urine.

As with frequent urination, so with incontinence, the possible presence of an overdistended bladder should not be overlooked.

Retention of Urine.—As previously stated, retention may alternate or coexist with incontinence of urine or frequent urination. The same injuries and diseases of the cord which cause the latter two will also produce retention. It is met with in all forms of coma, in typhoid fever, and other febrile or non-febrile diseases in which the typhoid state occurs, in peritonitis (pelvic or general), and very seldom it arises from diphtheritic paralysis. In women, aside from parturition, which is not inconsistent with the regular voiding of a portion of the retained urine, the most common cause of retention is hysteria.

Atony of the bladder consequent upon an unduly long postponement of micturition leads to brief retention upon attempting to urinate, usually overcome without catheterization, unless the prostate gland is enlarged. In the infant, irritating urine may cause so much pain during urination that after one or two trials the child will refrain from so doing as long as possible. In elderly men prostatic enlargement is the most frequent cause of retention. Stricture of the urethra, urethritis, impaction of a calculus in the urethra, usually near the meatus, cystitis, or tumours of the bladder produce it, and the lodgment of a calculus in the ureter may by reflected irritation cause a spasm of the vesical sphincters.

(*f*) **Suppression of Urine.**—When for any reason urine is not secreted by the kidney, or, if secreted, does not reach the bladder, it constitutes *anuria*, or suppression of urine. Anuria must be separated from retention by using the catheter and finding the bladder empty.

Anuria not infrequently shows a striking absence of symptoms. In other cases uræmia (*q. v.*) supervenes. In persistent total anuria death usually occurs within 12 days. Total suppression of urine is rather rare. Ordinarily a small quantity is secreted, but so small that it practically constitutes anuria.

The various conditions and diseases which may be attended by suppression of urine are as follows:

In acute congestion, acute nephritis, the terminal stage of chronic nephritis, renal abscess, hydronephrosis and pyonephrosis anuria may exist because of interference with the secreting tissue of the kidney. Poisoning by lead, turpentine, cantharides or phosphorus and, very rarely, the inhalation of ether, may cause it. It may be due to the prolonged watery drain from the blood in Asiatic cholera and cholera infantum, and is a possibility in cholera morbus; so also with collapse or shock from injuries, operations (especially those involving the urinary tract in the aged), or gastro-intestinal perforations.

Yellow fever, typhoid fever and the typhoid state, the terminal stage of acute yellow atrophy of the liver, pernicious malarial fevers, the terminal stage of sunstroke, and, infrequently, peritonitis, may conduce to a cessation of the urinary function, presumably by causing hyperæmia or disturbed innervation of the kidneys.

A curious condition, due to disturbed innervation, is the anuria of hysteria, which may be so prolonged as to evoke uræmic symptoms. More frequently the condition is that of retention rather than suppression. This diagnosis should be made with great caution, and is to be based on well-marked associated symptoms of hysteria. While not intending to classify hysteria with malingering, it is here convenient to speak of those cases of feigned anuria which every hospital physician sees. A successful device for detecting this trick is to catheterize at a certain hour, and 2 or 3 hours after, when the patient least expects it and has made no preparation for it, to use the catheter again.

Rare causes of anuria are thrombosis of the inferior vena cava and of the renal vein. This condition is remotely conjecturable if there has been an injury of the kidneys, with blood, albumin, and casts in the urine, followed by diminution and suppression.

Obstructive suppression embraces all conditions in which one or both ureters are occluded by outside pressure or obstacles within their lumen. Pressure from the outside may result from an intra-abdominal tumour, or a large aneurism of the abdominal aorta, or cancer of the bladder involving the ureters at their entrance. Obstructions within the ureter when present are usually impacted calculi. Both ureters may be obstructed at the same time. If only one ureter is occluded, anuria may still follow because the opposite kidney is diseased or absent. Such cases are not extremely rare, two instances having come to my knowledge within the past year.

In distinguishing between obstructive and non-obstructive suppression, if any urine at all is passed some aid may be derived from the urinary examination; a high specific gravity, blood, albumin, and casts, favouring the non-obstructive form, while a normal specific gravity and the absence of abnormal constituents argues for ureteral obstruction. The recent advances in cystoscopy and ureteral catheterization are of much service in the diagnosis and treatment of some forms of renal disease, provided the requisite skill can be secured.

VI. SYMPTOMS BELONGING TO THE GENITALIA

1. **Males.**—The genital symptoms of somewhat general diagnostic value which occur in the male are urethral discharges, priapism, pendulous testicles, varicocele, spermatorrhœa, impotence, and masturbation.

(*a*) *Urethral Discharges.*—Discharges from the urethra may be due to a concealed initial lesion of syphilis, simple or gonorrhœal urethritis, or gleet, and inflammation of the prostate gland. A thin mucous discharge may be caused by excessive or ungratified sexual desire. From the medical standpoint, urethral discharges may have a bearing upon the diagnosis of the manifold lesions of syphilis; gonorrhœal rheumatism or conjunctivitis; syphilis, or gonorrhœal pelvic inflammations in the wife of the infected male; and sexual neurasthenia or hypochondriasis.

(*b*) *Priapism.*—Prolonged or abnormally frequent erection of the penis, with or without sexual desire, constitutes priapism.

Priapism may be indicative of vesical calculus, a distended bladder, hypertrophy of the prostate gland, gonorrhœal urethritis, or adherent prepuce. It may be a symptom of poisoning by cantharides. A loaded rectum, inflamed hemorrhoids, a blow upon the perinæum, or the irritation from seat worms (ascarides) may be responsible for it. It is an occasional symptom of injuries (cervical and lumbar) and diseases of the spinal cord, as in myelitis and spinal meningitis, and has been observed in lesions of the pons and in hemorrhage into the cerebellum. It is at times a premonitory or immediate symptom of an epileptic seizure. Priapism is a not infrequent event in leucæmia, of 6 weeks' duration in one case. It may also be present in hydrophobia, tetanus, rarely in diabetes mellitus, and is sometimes caused by alcoholic or sexual excesses.

(*c*) *Pendulous Testicles.*—A loss of tone in the muscular and other components of the spermatic cord and scrotum, thus allowing the testicles to droop unduly, may be a symptom of old age, self-abuse (especially if attended with spermatorrhœa), excessive sexual indulgence, and conditions of debility in general. It is also present in connection with the impotence resulting from locomotor ataxia and diabetes mellitus.

(*d*) *Varicocele.*—This is a condition in which the spermatic veins are enlarged and varicose. It may occur under similar circumstances to (*c*) and is sometimes associated with neurasthenia and hypochondriasis.

(*e*) *Spermatorrhœa.*—In a continent individual involuntary emissions during sleep, if occurring at irregular intervals of 2 to 6 weeks, are quite normal. If these discharges are habitually more frequent, 2 or 3 times a week or perhaps every night, a pathological condition exists. If the emissions occur without erection, unconsciously to the patient, in the daytime or while straining at stool, their pathological character is very marked.

Before accepting the statement of a patient that he has spermatorrhœa, especially if he has been terrorized by the perusal of quack literature, the history should be gone over carefully. He not infrequently believes that a heavy phosphatic sediment in the urine or a thin mucous discharge due to prostatic or other irritation is escaping semen. Occasionally there is a fixed delusion that clear and normal urine is the constant carrier of the testicular fluid. A chemical examination of the urine will prove the nature of the phosphatic deposit, and a microscopical examination the presence or absence of spermatozoa.

Spermatorrhœa is variously indicative of self-abuse, excessive coitus, sexual neurasthenia as cause or consequence, and the early stage of locomotor ataxia. Sedentary habits, an habitually loaded rectum, ascarides, and the too free use of condiments and alcoholic liquors may be responsible for the slighter degrees of this symptom.

Very often in these cases there is mental depression, debility, lack of energy, and a persistent brooding over a condition which is supposed to be, and sometimes from this very fact becomes, incurable. The urine is apt to be of high specific gravity, acid and containing oxalates, or alkaline with a deposit of phosphates.

(*f*) *Impotence.*—An inability to perform the act of coitus is a common and sometimes an early sign of diabetes, and is usually present in the later stages of locomotor ataxia or other diseases of the spinal cord in which there is involvement of the bladder and rectum or anæsthesia of the glans penis.

There is an impotence which is purely psychical, emotions of fear, shame, or lack of confidence interfering with the normal action of the nervous mechanism, erection failing to occur, or ejaculation taking place prematurely. Impotence may come on at an unusually early age, although there is no strict dividing line of years, in those who have practised excessive venery or self-abuse.

(*g*) *Masturbation.*—Modern writers attribute much less pathogenic power to this habit than do those of 20 or 30 years ago. Its effects are practically those of excessive coitus. It may be a symptom and not a cause of insanity and sexual perversion. On the other hand, excessive masturbation, like excessive venery, may cause sexual neurasthenia, and hypochondria, spermatorrhœa, pendulous testicles, "nervousness," debility, and palpitation of the heart. Little importance is attached to onanism and excessive coitus as causes of locomotor ataxia and other organic diseases of the spinal cord.

As the majority of boys practise this habit until enlightened concerning its evils, it should be suspected in cases exhibiting anomalous nervous symptoms without other appreciable cause. There is no characteristic expression of the face which will betray the onanist. If he is aware that he is doing wrong, the facial expression will simply correspond to that of a conscious wrongdoer.

2. **Females.**—The symptoms pertaining to the female genitalia which possess a general medical interest are vaginal discharges, amenorrhœa, dysmenorrhœa, menorrhagia, and metrorrhagia.

(*a*) *Vaginal Discharges.*—Aside from its constant presence as a symptom of various pelvic disorders, leucorrhœa, the most common of vaginal discharges, may be a symptom of anæmia and debility from sundry causes, as fatigue, deficient food supply, phthisis pulmonalis, and overlong lactation. An offensive serous or purulent discharge, tinged or not with blood, is a frequent symptom of carcinoma uteri, and in the absence of pelvic pain may give the clew to an obscure anæmia and debility. In children, a discharge from the genitals may be due to a vulvitis or vaginitis, usually the former, caused by the migration of ascarides from the rectum and the irritation consequent upon their presence.

(*b*) *Amenorrhœa.*—Omitting the menopause, pregnancy, imperforate hymen, and congenital absence or imperfect development of essential organs, cessation of the menses is almost invariably dependent upon some abnormal condition of the blood or nervous system, or both.

It may occur as a symptom of grave hysteria and melancholia or other insanities, or be due to strong emotions (fear, grief, worry) which interrupt the normal nervous balance, or to a change of surroundings and occupation, of which the amenorrhœa sequent to a sea voyage is an example. Mental overwork in schoolgirls is sometimes responsible for the absence of the menstrual flow, so much nerve force being expended in one direction that other functions must suffer.

Anæmia, associated with a weakened nervous system, is the condition most commonly productive of amenorrhœa. Chloro-anæmia and phthisis pulmonalis are the underlying causes most frequently encountered, the anæmia and malnutrition attending these maladies being of such a grade that the additional loss due to menstruation can not be afforded. Indeed, the amenorrhœa of these and other diseases is a conservative consequence, and not, in opposition to a natural false logic on the part of patients, a causal factor. For a similar reason, amenorrhœa may be indicative of chronic nephritis, diabetes, tuberculosis of the kidney, the cancerous, malarial, or saturnine cachexiæ, chronic mercurial poisoning, morphine addiction, and leucæmia. It is not infrequently due to the anæmia which attends gastric ulcer and convalescence from typhoid fever and the exanthemata. Scanty menstruation with recurring amenorrhœa is rather common in obese, anæmic patients—the "fat anæmics."

(*c*) *Dysmenorrhœa.*—Of the recognised varieties of dysmenorrhœa, the neuralgic form only is of direct medical interest, as it is not associated with pelvic lesions but is part and parcel of a general neurotic condition or a manifestation of some constitutional or blood disorder.

The pain of neuralgic dysmenorrhœa is somewhat characteristic. It is paroxysmal and radiating like the neuralgias, with its greatest intensity in the hypogastrium, passing to one or the other iliac region, whence it shoots down the corresponding thigh. It begins before the flow, and may stop or continue after the flow is established. It bears no relation to the amount or character of the menstrual loss, and there are none of the usual symptoms or physical signs of pelvic lesions. In cases of sufficient severity to call for treatment, a more or less neuropathic diathesis is almost invariably found. There is apt to be a hyperæsthetic condition of the skin of the lower abdomen, with painful points at the emergence of nerve branches. The character and severity of the pain may be such as to cause delirium or partial coma, and in some intractable cases, fortunately few in number, the steadily recurring seizures may give rise to grave hysteria, and perhaps to epilepsy or mania. The face in marked cases is pale, and the patient may present the symptoms of collapse (*q. v.*) with lowered temperature. In conjunction with neurotic tendencies, certain general disorders may initiate or intensify the pain. In all cases of neuralgic dysmenorrhœa search should be made for malaria, syphilis, gout or lithæmia, rheumatism, and anæmia.

A peculiar form of dysmenorrhœa is that which is termed "membranous," in which there is expelled with miniature labour pains a hollow, membranous cast of the uterine cavity. The membrane, when examined microscopically, appears to be the abnormally thickened menstrual decidua. While this condition is attributed to an endometrial inflammation, yet there appears to be a relation between the nervous system and the painful passing of membrane. Certainly several cases seen have exhibited nervous idiosyncrasies.

A word may be said here regarding *intermenstrual* pain, which has been described by Palmer and others. It is apt to begin about two weeks after menstruation has ceased. The pain, beginning gradually, increases day by day until it reaches an unbearable intensity and finally passes away with the advent of the next menstruation. It is rather a rare condition, depending upon disease of the ovaries and requiring their removal for its relief.

(*d*) *Menorrhagia and Metrorrhagia.* — An excessive menstrual flow (menorrhagia) and an intermenstrual flow (metrorrhagia) may be caused by the vast majority of diseases of the uterus and its appendages. But either or both may be, and not infrequently are, due to general diseases and conditions.

The various infectious diseases—influenza, dengue, malarial fever, scarlet fever, typhoid fever, variola, and cholera—may be attended by excessive menstruation or a bloody uterine flow. Menorrhagia is not uncommon in the early stage of phthisis pulmonalis, although the reverse is usually the case.

Certain blood conditions may manifest one or the other abnormality. Among these are the grave anæmias (rarely), more often hæmophilia, purpura, scurvy, leucæmia, the uræmia of nephritis, and severe cholæmia or jaundice. Plethora has been assigned as a doubtful cause. Certain intoxications may originate or increase uterine bleeding, as acute or chronic alcoholism, emmenagogues, chronic lead poisoning, and phosphorus poisoning. Among miscellaneous factors, valvular or other organic disease of the heart, cirrhosis of the liver, and acute articular rheumatism should not be overlooked, nor, if the age of the patient be suggestive, the possible beginning of the menopause.

In searching for the explanation of a menorrhagia or metrorrhagia, first eliminate local pelvic causes, and then examine particularly for chronic cardiac, renal, or hepatic disease, syphilis, and malaria.

SECTION XIII

CERTAIN SYMPTOM GROUPS OF CLINICAL SIGNIFICANCE

THERE are symptom groups or sets of correlated symptoms which it is of service to present here as distinct clinical pictures. These groups comprise coma, dyspnœa, fever, hyperpyrexia, internal hemorrhage, shock or collapse, syncope, weakness or debility, irritant poisoning, jaundice, obstruction to the portal circulation, hectic fever, pyæmia, tympanites, and the typhoid state.

I. **Coma.**—There is loss of consciousness, with stertorous respiration and expiratory puffing of the cheeks and lips. The mouth is partly open and the tongue is dry. The cornea is insensitive, and one or both pupils are either dilated, contracted, or with defective response to light. There may be an unusually slow pulse. Involuntary dejection and urination (*q. v.*) may occur.

II. **Dyspnœa.**—There is either rapid or laboured respiration. If the dyspnœa is severe, the face is cyanosed and wears an anxious expression. The skin is covered with cold perspiration, the patient speaks in broken sentences, and may be unable to lie down (orthopnœa, *q. v.*).

III. **Fever.**—There are slight chilly sensations, the pulse is accelerated, there are thirst, loss of appetite, headache, backache, slight or severe aching of the body and limbs, and a feeling of weakness. The tongue is coated, and the urine is usually high-coloured and decreased in amount, with an abundant deposit. Finally, the temperature is found to be elevated.

IV. **Hyperpyrexia.**—If the temperature is found to be 106° or over, there may be added to the symptoms just enumerated in III dicrotism of the pulse, delirium, and marked restlessness.

V. **Internal Hemorrhage.**—The symptoms of internal or concealed hemorrhage are not sufficiently distinctive to enable a positive diagnosis to be made, without taking into account the presence of diseases or conditions in which such an event is liable to take place. These symptoms are practically identical with those of shock or collapse, except that air hunger and restlessness are especially prominent features in the clinical portrait of hemorrhage. In the majority of cases of internal hemorrhage the blood makes its appearance externally, as in hæmatemesis, hæmoptysis, epistaxis, metrorrhagia, hæmaturia, and bloody stools, thus making evident the nature of the symptoms. But with a knowledge of the diseases and conditions in which concealed hemorrhage may occur, and by instituting a careful search for the existence of such lesions, the diagnostician is at times enabled to predict the later appearance of blood at some of the natural orifices of the body or its finding at operation, as in bleeding from a typhoid ulcer or the rupture of an ectopic gestation. In order to produce recognisable symptoms the hemorrhage must be large. Slight hemorrhages do not, of course, give rise to appreciable symptoms, nor do the perturbations caused by visible and external bleeding differ from those due to unseen bleeding.

The symptoms indicative of concealed hemorrhage are as follows:

Pain may or may not be present, depending on the nature of the lesion which causes the hemorrhage. The face becomes pallid, pinched, and anxious, and the extremities are cold. The surface of the body is covered with perspiration. The respirations are shallow and sighing, the patient gapes repeatedly, and urgently desires more air. There is great restlessness with turning of the body from side to side. The radial pulse is rapid, weak, and thready, and may become imperceptible. If the hemorrhage is large and rapid, syncope and unconsciousness may ensue. The mind may be clear, but more commonly there is delirium. Nausea is usually present, and vomiting may take place. Jactitations and convulsions may be added to the scene. At the onset of the attack the heart beats violently, due partly to excitement and partly to the fact that the ventricular blood content is rapidly growing smaller, but very soon the apex beat disappears as the systole becomes weaker and weaker. If the heart and the large blood-vessels are auscultated, loud hæmic murmurs are heard. When hemorrhage takes place during the presence of fever the temperature falls rapidly to or below the normal point.

The diseases and pathological conditions in which concealed hemorrhage, sufficient to cause symptoms, may supervene are:

(1) *In the Thorax.*—Bleeding into a large pulmonary cavity or the rupture of a thoracic aneurism into the mediastinum or pleural cavities.

(2) *In the Abdomen.*—Gastric ulcer, duodenal ulcer, typhoid or other ulcers of the intestine, or the rupture of an aneurism of the abdominal aorta into the stomach, intestine, or peritoneal cavity.

(3) *In the Pelvis.*—Concealed uterine hemorrhage before or after labour or rupture of an extra-uterine fœtation. Although in the majority of cases pelvic hæmatoma (effusion of blood between the folds of the broad ligament) and pelvic hematocele (effusion of blood into the peritoneal cavity) are due to ectopic gestation, yet, as these conditions do occur from other causes, they are mentioned here separately.

(4) *Hæmophilia* and purpura hæmorrhagica may be responsible for a variety of internal hemorrhages, sometimes of considerable extent.

(5) *Trauma.*—Crushing injuries, leading to rupture of organs and penetrating wounds, are especially liable to concealed or internal hemorrhage.

VI. **Shock or Collapse.**—Although these terms are ordinarily considered to be synonymous, the useful clinical distinction drawn by Shrady should prevail, whereby "collapse" shall imply sudden prostration occurring in cases not strictly surgical, as in irritant poisoning or intestinal perforation; while "shock" is limited to a similar condition resulting from accidental or surgical traumatism.

The symptoms of collapse are pallor, anxious expression, lowered temperature, cold perspiring skin, thready or imperceptible pulse, intense weakness, and intact or impaired intellection. This condition may develop rapidly or slowly, usually the former. Depending on the resisting power of the individual as well as of the tissues involved, it varies in severity. Cases occur in which collapse symptoms are almost entirely absent and yet the autopsy reveals lesions which ordinarily give rise to profound prostration at the time of their occurrence, as in some instances of intestinal perforation. On the other hand, certain ailments, commonly unattended with danger, may exhibit an apparently alarming degree of weakness, as in a form of dysmenorrhœa frequently associated with an anteflexed cervix, which presents a typical collapse invariably followed by a favourable termination.

The diseases and conditions causing collapse are:

(*a*) Those already mentioned as attended with *internal hemorrhage* (*q. v.*).

(*b*) The various *infectious diseases*, sometimes at their onset, more commonly at their terminal stages, as variola, pernicious malarial fevers, typhus fever, typhoid fever, glanders, acute tuberculosis, erysipelas, dysentery, cholera (stage of collapse), diphtheria, and acute yellow atrophy of the liver.

(*c*) *Lesions of the Heart and Lungs.*—A severe attack of pneumothorax shows a marked collapse. So also is it with embolism of the pulmonary artery, pulmonary abscess, and pulmonary gangrene. Pneumo-pericardium, chronic valvular disease of the heart, and ulcerative endocarditis develop collapse, the latter two usually toward the termination of the illness.

(*d*) *Disease within the Abdomen.*—Severe acute enteritis or diarrhœa, intestinal obstruction, gastric, duodenal, or intestinal perforation, acute septic peritonitis, intense hepatic colic, perforation of the diaphragm by hepatic abscess or subphrenic abscess, and acute pancreatitis exhibit collapse or prostration in varying degrees.

(*e*) *Narcotic and irritant poisons*, as in acute alcoholism, acute poisoning from arsenic, antimony, tartar emetic, and chronic poisoning from ergot. In irritant poisoning the collapse symptoms usually appear during the later stages.

(*f*) *Traumatism*, particularly if there is a rupture of one of the viscera or concussion of the brain or cord.

(*g*) *Other Causes.*—Collapse also occurs with inversion of the uterus, trichinosis, and occasionally hyperpyrexia, cancrum oris, and suppurative tonsilitis.

VII. **Syncope.**—Fainting or syncope is a sudden and usually temporary pallor and loss of consciousness from a weakening of the heart's action. The face is calm, the respirations are quiet, the pulse is weak or imperceptible, the pupils are dilated but responsive to light. An attack of syncope may sometimes be anticipated by the occurrence of yawning, nausea, and sighing respiration. Fainting much resembles collapse, except that in the former loss of consciousness takes place, the failure of power is confined to the heart, and the attack is ordinarily evanescent.

VIII. **Weakness or Debility.**—The patient has a feeling of languor and weakness and tires easily. There is usually some shortness of breath on exertion and the movements are slow and made with evident effort. The heart sounds are weak, especially the first, and the pulse quickens unduly after gentle exercise.

IX. **Poisoning by Irritants.**—In general, the symptoms of irritant poisoning (suggestive but not pathognomonic) are nausea and violent vomiting, with much tenderness and severe pain in the epigastrium, followed by diarrhœa and the evidence of marked collapse. The fæces may show traces of blood.

X. **Jaundice.**—A yellow or saffron-coloured skin and conjunctiva, general itching of the body, depression of spirits, slow pulse, dark-brown urine, and pale or clay-coloured stools, constitute the symptom group of obstructive jaundice (p. 87).

XI. **Obstructed Portal Circulation.**—Hydroperitoneum or ascites, with enlarged veins around the umbilicus, slight jaundice or sallow skin, hemorrhoids, gastric disturbances, and, when of some standing, great œdema (*q. v.*) of the genitals and lower extremities.

XII. **Suppurative or Hectic Fever.**—A patient with bright eyes and a clear mind, who is running a temperature which rises in the evening to various heights and subsides in the early morning, usually with sweating, with a continuously rapid pulse, a pale face with a circumscribed flush on the cheek, and persistent anorexia, is probably the subject of suppurative or tubercular disease of considerable duration. It should not be mistaken for typhoid fever.

XIII. **Pyæmia.**—If alone, or in addition to the symptoms of suppurative fever, there are irregular, recurring chills, with nausea and vomiting, the temperature running suddenly to a high point and falling rapidly with profuse sweating, the cause may be found in the formation of metastatic or embolic abscesses, derived from a primary suppurative focus and constituting the condition called pyæmia. It is not a very uncommon occurrence for pyæmic chills and fever to be considered as of malarial origin.

XIV. **Tympanites.**—Meteorism or tympanites consists of a marked distention of the stomach and intestines with gas. As a consequence of the upward pressure against the diaphragm, there is dyspnœa, a rapid, feeble, or irregular pulse, and an upward displacement of the apex beat (see also Abdomen, General Distention of).

XV. **Typhoid Status.**—The symptoms of this condition are muttering delirium or coma vigil, dry brown tongue, teeth covered with sordes, twitching of the tendons (*subsultus tendinum*), and possibly picking at the bedclothes or grasping at imaginary things in the air (*carphologia*). The bases of the lungs may be passively congested (hypostatic pneumonia), and ordinarily the temperature is considerably elevated, although the typhoid state may exist with the mercury at the normal point.

The typhoid state is common to a number of different diseases and lesions, as follows:

(*a*) *Infectious Diseases.*—The typhoid state occurs with the greatest frequency in infectious diseases, especially lobar pneumonia, erysipelas, pyæmia, and the terminal stage of acute dysentery. It is also found, as indicated by the name, in typhoid fever (less common in cases treated by cold tubbing), typhus fever, pernicious malarial fevers, malignant variola, malignant scarlet fever, epidemic influenza, acute yellow atrophy of the liver, acute miliary tuberculosis, ulcerative endocarditis, puerperal septicæmia, anthrax, and glanders.

(*b*) *Certain Abdominal Diseases.*—Of these the typhoid state may develop, toward the close of the disease, in jaundice of the obstructive form in which the obstruction is insuperable. It may supervene also in the course of hepatic abscess, appendicitis (especially if unrecognised), acute enteritis and peritonitis (infrequently), chronic nephritis, and pyelitis.

(*c*) *Intracranial Causes.*—It is sometimes found in meningitis, toward the end of cerebral embolism and thrombosis with much softening, and general paresis.

(*d*) *Other Causes.*—The typhoid state may be due to purpura hæmorrhagica and acute phosphorus poisoning. It may also be produced by any suppurative inflammation, particularly if pyæmia arises therefrom. One of the most typical examples of the typhoid status that I have seen was the result of a previously unrecognised deep-seated perineal abscess, the incision and drainage of which led to recovery.

SECTION XIV

HEAD AND FACE

I. Size and Contour.—There are certain changes in the size and contour of the head and face which are of diagnostic importance in the following diseases:

(*a*) *Hydrocephalus.*—The hydrocephalic head is abnormally large and usually globular, sometimes pyramidal, in shape (Fig. 35); the anterior fontanel is large and bulging, the sutures vary in width from ½ to 2 inches or more, and the veins of the scalp are visibly distended. The face is relatively small and the eyelids are raised with difficulty. The occipito-frontal circumference of the head is normally, at birth, a little more than 14 inches, and at 1 year old about 18 inches. An abnormal increase in this measurement is the principal symptom by which chronic hydrocephalus (due to ventricular distention) may be recognised. It has been known to reach

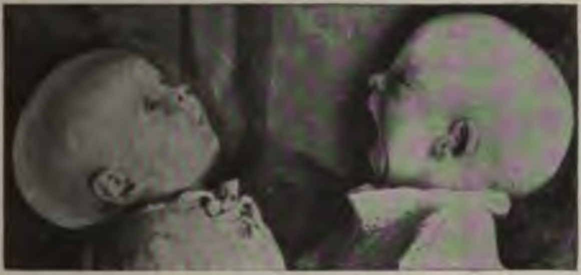


FIG. 35.—Twins 10 months old; both normal at birth. The infant to the right shows a beginning hydrocephalus. M. E. H. case. Photograph by Dr. Raymond Clark.

32 inches at 8 months of age. Aside from the enlargement the most characteristic feature is the prominence of the forehead at the root of the nose. The most common error is to mistake the rachitic head for hydrocephalus.

(*b*) *Rachitis.* — If the cranium, especially when viewed from above, is elongated, large, and square, the vertex flattened, the frontal eminences protuberant, the face small and delicate, the anterior fontanel open when it should be closed, and alterations in the other skeletal bones be present, these changes are due to rachitis (Fig. 36).

(*c*) *Sporadic Cretinism.* —If the head is large and flat-topped, the anterior fontanel open (even as late as the tenth year), the face broad and flat, with wide, negroid nose, the forehead low, the eyes wide apart, the mouth partly open and the tongue slightly protruding (Fig. 37), it is a case of sporadic cretinism.

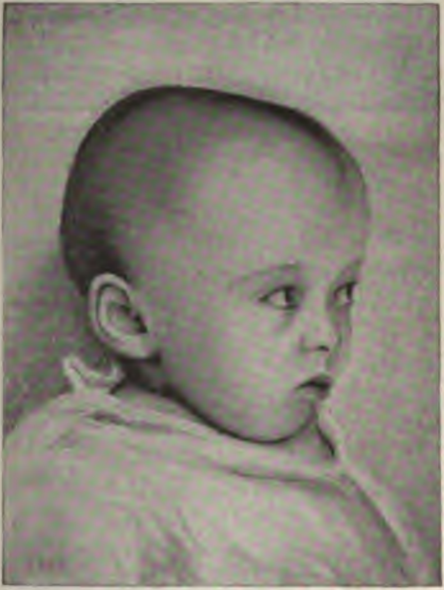
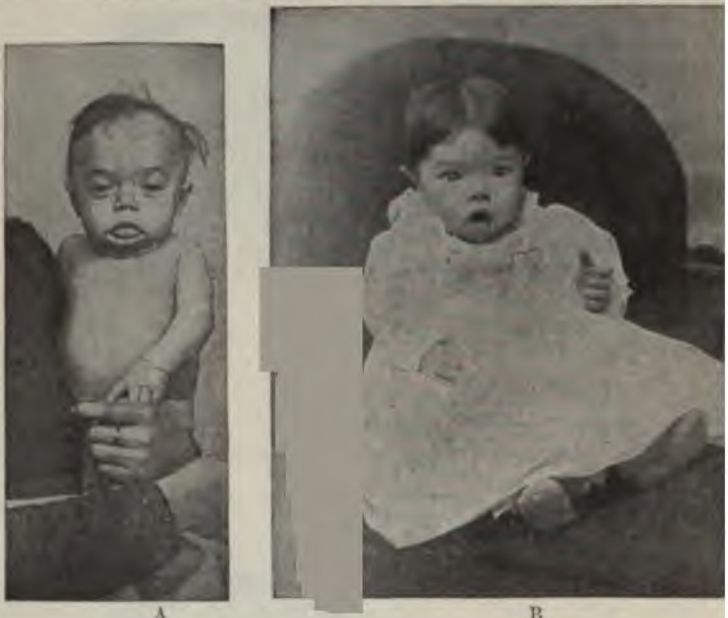


FIG. 36.—Rachitic head; Italian child two years old; square, prominent forehead and flat vertex (Holt).

(*d*) *Idiocy.*—In some cases the shape and size of the may suggest the presence of imbecility or idiocy, but, as are no physical changes which can be considered charact



A B

FIG. 37.—Sporadic cretinism. A from Koplik, B from Pearce.

of the various forms of mental or moral weakness con under these terms, the diagnosis must be made principally physical symptoms. Hydrocephalus may be the cause of " cephalic" idiocy.

(*e*) *Acromegaly.*—If the head is somewhat enlarged, with greater increase in the size of the face, which becomes broaden elongated, assuming an ovoid shape with the large end down consequence of the great increase in size of the maxillæ (es the lower), and, furthermore, if the teeth are widely separat tongue, nostrils, eyelids, and ears thickened and hypertrophie 38), it is a case of acromegaly (*q. v.*).

(*f*) *Myxœdema.*—If the patient has a coarse-featured, "full-moon" face, broad, thick nostrils, a thick-lipped, large (Fig. 39), and a rough, dry skin, the existence of myxœdema is almost certain, but further confirmatory evidence sho obtained.

(*g*) *Osteitis Deformans.*—If the face is triangular in sha the base upward, and the head is lowered and carried forward

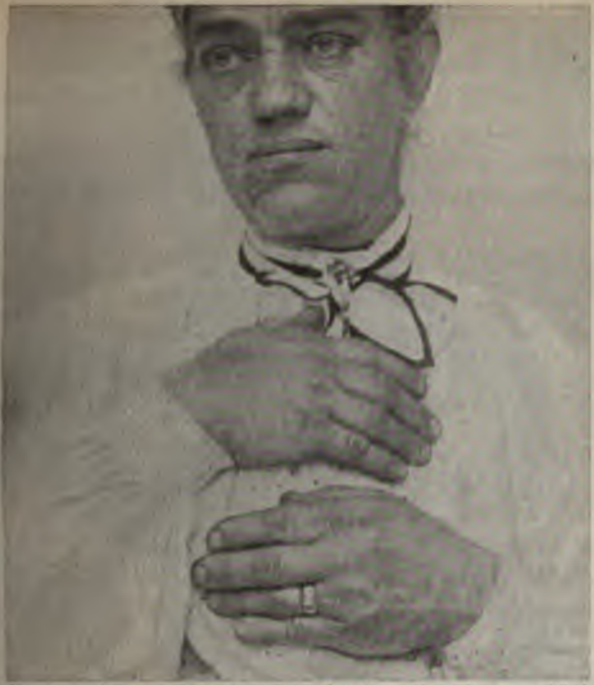


FIG. 38.—Acromegaly in a woman, showing enlarged supra-orbital ridges, inferior maxilla and hands (Pearce).

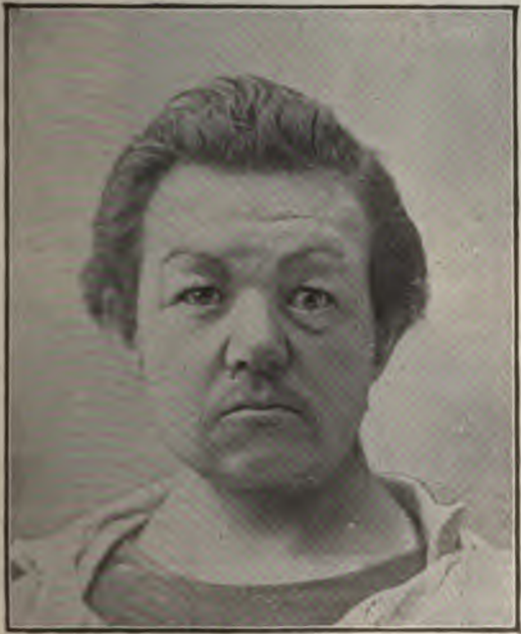


FIG. 39.—Face of myxœdema (Gordinier).

the chin is below the episternal notch, a search for other bone alterations is required, as it may be an example of "Paget's disease" or osteitis deformans.

(*h*) *Facial Hemiatrophy.*—If the face presents an appearance as if it was composed of two lateral halves from different individuals, with the vertical line of junction sharply defined, and the hair of the smaller side is thin or absent, the eye sunken, and the skin altered in appearance (Fig. 40), it is an instance of facial hemiatrophy. Facial asymmetry may exist also with congenital torticollis or wryneck (*q. v.*).

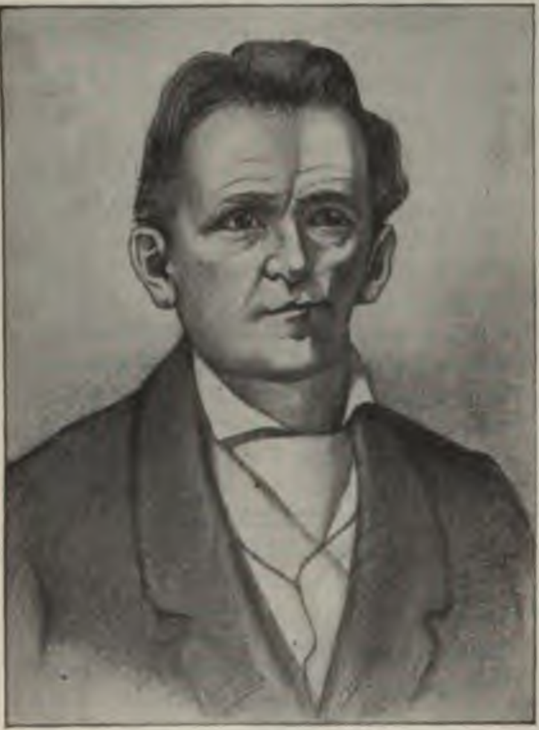


Fig. 40.—Facial hemiatrophy. Redrawn from Strümpell.

(*i*) *Leontiasis Ossea.*—If the cranium is enlarged and globular, the malar prominences are marked, and the orbital rims massive (Figs. 41, 42), the presence of hyperostosis cranii or leontiasis ossea, may be suspected.

(*k*) *Leprosy.*—The tuberous growths of leprosy (*q. v.*) occurring upon the face, together with the resulting ulceration and cicatrization, may slowly change the shape and contour of the countenance, which assumes a leonine aspect, the *facies leontina.*

II. **Fontanels and Sutures.**—(*a*) Prominent or bulging fontanels (in infants or children) are significant of chronic hydrocephalus, acute and tuberculous meningitis, and meningeal hemorrhage. The anterior fontanel is frequently prominent and pulsates strongly in the acute febrile affections of infants, probably in consequence of cerebral hyperæmia.

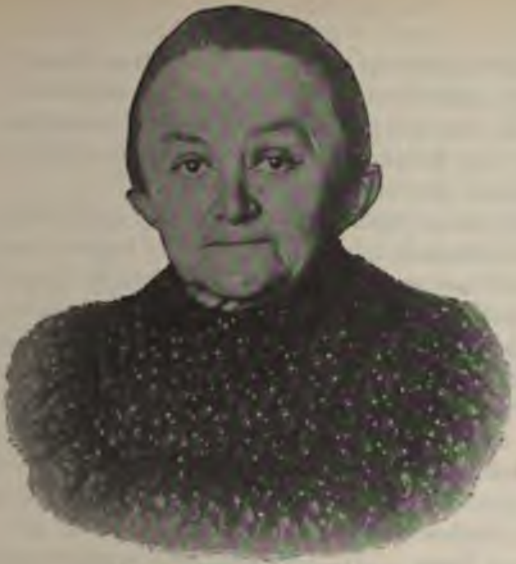


FIG. 41.—Leontiasis ossea (Edes).

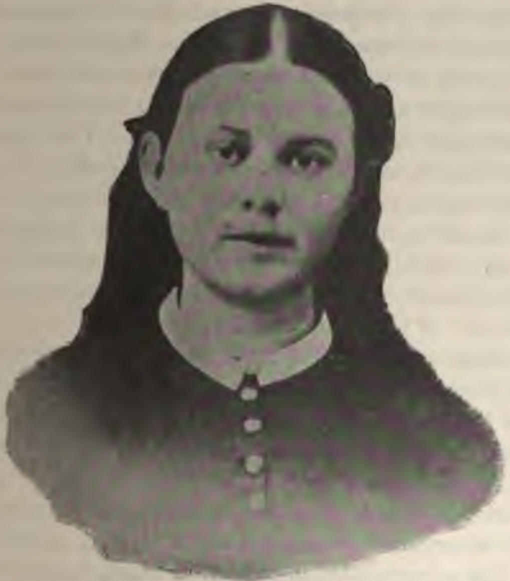


FIG. 42.—Same patient as in Fig. 41, previous to the development of the disease (Edes).

(*b*) A *sunken fontanel* is found in wasting diseases and severe diarrhœal or choleriform affections, such as marasmus and cholera infantum. In the latter disease a depressed fontanel is always present as one of the symptoms of the hydrencephaloid state (spurious hydrocephalus). The sutures may overlap.

(*c*) A *very large fontanel* is particularly symptomatic of hydrocephalus, and if smaller, but still unusually large, of rachitis, cretinism, and hereditary syphilis. When the infant is 1 year old the anterior fontanel should not exceed 1 inch in diameter.

(*d*) *Delayed closure* of the fontanels, the posterior normally disappearing at the end of the 2d month, the anterior from the 14th to the 22d month, is due ordinarily either to rachitis or to hydrocephalus, particularly the former.

(*e*) *Abnormally wide* cranial sutures, which physiologically are fused by the end of the 6th, certainly by the 9th, month, may indicate rachitis, hydrocephalus, or cretinism.

III. **Cranial Bones.**—(*a*) *Craniotabes.*—If in infants under 6 months of age several spots, ½ to 1 inch in diameter, are found on the occipital bone or the posterior portion of the parietal bones, which upon pressure by the finger give a soft crackling sensation, it is termed craniotabes and signifies rachitis or syphilis, or both conjoined.

(*b*) *Meningocele, Encephalocele, and Hydrencephalocele.*—A congenital pulsating tumour, varying in size from a walnut to a fœtal head, bulging out between the cranial bones either in the occipital or the frontal regions, and if large, probably translucent, is a meningocele, encephalocele, or hydrencephalocele. The tumour is ordinarily pedunculated, and pear-shaped or rounded.

(*c*) *Nodes.*—Soft, painful, and doughy swellings of the skull, the pain being worse at night, and the swellings becoming harder as time passes, are syphilitic nodes (gummatous periostitis). If cerebral symptoms coexist, it is probable that similar nodes are forming on the inner surface of the cranial bones.

IV. **The Face as Indicative of Certain Diseases.**—In addition to the value of the facial expression in affording a clew to the psychical condition of the patient (*q. v.*), the face may present a more or less suggestive physiognomy in certain diseases or conditions. An appreciation of the facial traits of disease, like the power of making a rapid diagnosis, is based upon the capability of comparison derived from many observations and, when analyzed, doubtless involves more than the recognition merely of a characteristic countenance. Nevertheless, as a help to an oftentimes difficult problem, the facies of disease is not infrequently of great service.

The diseases and conditions which exhibit, with more or less frequency and distinctness, a characteristic *facies* are: acute diffuse peritonitis, dyspnœa, exophthalmic goitre, paralysis agitans, phthisis, pneumonia, renal disease, typhoid fever, impending death, and, in children, mouth-breathing and hereditary syphilis.

(*a*) *Acute Diffuse Peritonitis.*—In this disease the expression of extreme anxiety may be very striking. The teeth are uncovered by the raising of the upper lip, and the whole expression is significant of pain and mental disquiet. The respiration is somewhat quickened because of the fixation of the abdominal muscles. In a sharp and well-developed attack, particularly if the vomiting has been severe and continuous, the Hippocratic countenance is seen more frequently, perhaps, than in any other disease except cholera. Localized, slight peritonitis has no characteristic physiognomy, although the faintly anxious expression is present and significant.

(*b*) *Dyspnœa.*—In conditions which are causative of difficult, laborious, or painful breathing, the mouth is open, the lips and tongue may be dry, the nostrils dilate with each inspiration, and the face presents a bluish pallor (cyanosis).

(*c*) *Exophthalmic Goitre.*—The most obvious facial trait of this disease is the excessive protrusion of the eyeballs (exophthalmos, proptosis), which may be so great as to prevent the complete closure of the eyes. In a well-marked case the singular staring expression is unmistakable.

(*d*) *Hysteria.*—The physician of experience will sometimes make a tentative and frequently confirmed diagnosis of hysteria from the silly and vacuous but very amiable smile which greets his introduction to the patient, or which accompanies the answer to every question which is put. On the other hand, a peculiarly intense frowning expression may appear in response to every remark directly involving the patient's symptoms, the face clearing and smoothing when the subject is changed to one of outside interest. The immovable face of hysterical coma, with its natural colour and the quivering resistance of the upper eyelid when the examiner attempts to raise it, is also of value in diagnosis.

(*e*) *Paralysis Agitans.*—In this disease the face presents a sphinx-like immobility and lack of expression, conjoined usually with a colour which is apparently too healthy to correspond with the general condition. This stony visage (Parkinson's mask) is of considerable value in the diagnosis of a doubtful case.

(*f*) *Phthisis Pulmonalis.*—In advanced phthisis the countenance is very expressive. The wide-open, appealing eye, with its usually white sclerotic, the emaciated face, the pallor of which is in strong contrast to the spots of red over the malar bones, the dilating alæ and panting respiration, constitute a significant and graphic portrait.

(*g*) *Pneumonia.*—In an ordinary frank pneumonia the face as a whole is flushed, with a deeper tint of red upon one cheek, and the alæ work strongly in consonance with the rapid respiration. As the disease approaches the septicæmic type, a general pallor brings the malar flush into stronger relief, and in the markedly typhoid variety the flush may entirely disappear.

(*h*) *Renal Disease.*—A pale, puffy face, with baglike swellings beneath the eyes, is seen in those diseases of the kidney which are attended with edema, particularly the diffuse nephritides, but not infrequently a suspicion is aroused by a glance at the patient which subsequently fails of confirmation by the urinalysis.

(*i*) *Typhoid Fever and the Typhoid Status.*—At the height of a well-marked case of typhoid fever the facies is very characteristic. The expression is dull and apathetic. The mentality is impaired and the patient is quite indifferent to his surroundings. He may be in a quiet muttering delirium. The tongue is apt to be dry and the teeth covered with brownish sordes. This facies is witnessed not only in typhoid fever but in all diseases which exhibit the typhoid status (*q. v.*).

(*j*) *Impending Death.*—"A sharp nose, hollow eyes, collapsed temples; the ears cold, contracted, and their lobes turned out; the skin about the forehead being rough, distended, and parched; the colour of the whole face being brown, black, livid, or lead-coloured." As a rule, such a countenance is a sure precursor of death, except in acute diffuse peritonitis, cholera, or starvation.

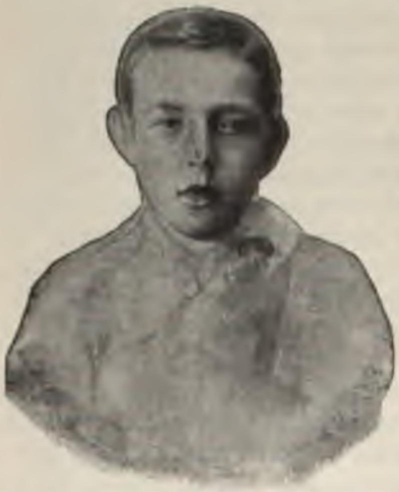


FIG. 43.—Composite photograph of 8 mouth-breathing boys. By Dr. Thomas R. French.

(*k*) *Mouth-breathing.*—A familiarity with the appearance of a mouth-breather is of great importance because of the serious results (anæmia, gastric disorders, phthisis, *et al.*) which may follow in neglected cases if the causative factors are allowed to exist during the developmental period. If the condition has lasted for a considerable time—years, perhaps—the nostrils are small and the nose itself relatively insignificant in size, the mouth is large and constantly open, the lips are thick and dry, the eyelids droop, and the expression is stupid and vacuous (Fig. 43). Further examination will reveal an insufficiently developed thorax.

(*l*) *Hereditary Syphilis.*—A child suffering from a well-developed hereditary syphilis has a weazened and pitifully old-looking face, very much like that of some species of the quadrumana. The skin is yellow and wrinkled, and it is likely that labial fissures, mucous patches, alterations in the shape of the nose, and other evidences of syphilis (*q. v.*) will be found.

V. **Colour of the Face.**—The skin of the face, as a part of the general cutaneous surface, shares in diffused pallor or cyanosis. But there are certain special alterations in the colour of the face of some diagnostic value—viz., sallowness, brownish discoloration, and flushing of the face.

(*a*) *Sallowness.*—This is a combination of pallor with a yellow or brownish-yellow tint. It is normally present in brunettes or natives of hot climates. But the presence of sallowness should always suggest its possible pathological character.

A sallow face may be indicative of one of the cachexiæ due to cancer, lead, syphilis, or malaria. It is also seen in the anæmias of brunettes. Addison's disease need only be mentioned as a possible cause. Arthritis deformans usually gives rise to a notable sallowness. Very many, indeed, the great majority, of the sallow faces seen in the consulting room are due to some disturbance or disease of the digestive system and resulting anæmia. Under this head come those who are subject to habitual constipation, chronic gastric disorders, or chronic enteritis. Hepatic congestion, cirrhosis and abscess of the liver are usually accompanied by a sallow complexion. The yellowness of the sallow face is to be discriminated from the yellow tint of slight jaundice by the absence of colour in the sclerotic and of bile pigments in the urine.

(*b*) *Brown or brownish-yellow spots* upon the face, the so-called "liver spots" of the laity, are in the greater number of instances examples of the chloasma (localized deposit of pigment) found in connection with pregnancy, chronic affections of the uterus or liver, and exophthalmic goitre. The possibility of the presence of Addison's disease should be considered. Localized deposits of pigment may be caused by continued scratching or by the use of counter-irritants or vesicants. There appears to be in some individuals an unusual predisposition to the deposit of colouring matter in the skin after the local use of mustard, turpentine, or cantharides, a fact not to be forgotten for cosmetic reasons in connection with women patients. The internal use of arsenic or the external application of the oil of cade may cause permanent discoloration of the skin.

(*c*) *Flushing of the face* may be of considerable duration, lasting for hours or days, or it may be sudden and evanescent, passing as quickly as it arrives. A permanently flushed and ruddy face may on closer inspection be seen to be caused by dilated arterial twigs or venous radicles, in which case it should arouse suspicion of atheromatous arteries or chronic nephritis. A flushed face is characteristic of the early stage or onset of the majority of febrile temperatures. It is particularly noticeable in malarial fevers and acute articular rheumatism, and may persist for days in typhoid fever. The unilateral flush of pneumonia is frequently seen, and the malar flush of pulmonary phthisis is sadly familiar. Ordinarily there is a red face in the first stage of acute alcoholism and the apoplectic form of intracranial hemorrhage. The face is usually flushed in hysterical convulsions and frequently in the comatose form of the same disease. Large fibroid tumours and ovarian cystomata are not uncommonly associated with a florid face. A heart which has hypertrophied to a greater extent than is demanded by its work at a given time may produce more or less permanent flushing of the face.

Evanescent or transient flushing of the face is a manifestation of irregular vasomotor action, which may be due to a variety of perturbing influences. In many cases such flushings are simply evidences of a more or less marked congenital instability of the nervous system, and when exhibited under mental excitement by children and young adults, particularly young girls, are not of diagnostic value. Care should be taken in such cases that a face flushed by the slight agitation of an interview with the physician does not disguise the presence of a decided anæmia (*chlorosis rubra*). It is not unusual, after the first excitement has subsided, to see the familiar pallor replace the red upon the cheeks and lips. In addition to anæmia, transient flushings are associated with conditions of fatigue, especially neurasthenia and exophthalmic goitre, sometimes with constipation, gastric catarrh, and gastric neuroses. Alternate redness and pallor of the face is quite common in cerebral meningitis, and is occasionally witnessed in typhoid fever. The vasomotor condition which above all others is made the subject of bitter complaint is the flushing, accompanied by a sensation of heat and by sweating, which attends the menopause.

VI. **Skin of Face.**—This shares in the eruptions of the exanthemata and generalized diseases of the skin. It is especially the seat of milium and acne. Ecchymoses of the face, if not due to traumatism, may be the result of the rupture of small subcutaneous vessels from violent coughing, as in pertussis, or are manifestations of purpura.

VII. **The Hair.**—This is to a very slight extent an index to the general robustness and vigour of the individual. Thick, coarse hair is at times associated with rude strength of constitution, and scanty, fine-textured hair with a delicate habit of body; but there are many instances in which this is not the case. There is some diagnostic value in an observation of the colour and scantiness of the hair.

(*a*) *Colour of the Hair.*—The physiological loss of the pigment of the hair (grayness) begins in the average individual at forty to forty-five years of age, and slowly increases as the years pass. Early grayness is frequently a sign of premature old age, and is associated with degenerative arterial changes, but is compatible with good health and otherwise normal tissues. It is sometimes an hereditary peculiarity. Cases of sudden whitening of the hair as the result of extreme terror or anxiety have been reported. In a case of Addison's disease the hair, previously a pronounced blonde, became as black and coarse as that of a North American Indian. Circumscribed gray spots may be due to trophic changes produced by neuralgia or other disease affecting the fifth nerve.

The fact, if it is apparent, that the hair is dyed, may be an important clue in the diagnosis of chronic lead poisoning, as this metal enters into the composition of various hair dyes and washes. Hydrogen dioxide bleaches, pyrogallic acid darkens, the hair. The hair of those who work in copper may acquire a greenish colour, while that of cobalt miners and indigo handlers may exhibit a bluish tint.

(*b*) *Loss of Hair.*—This may be general or circumscribed.

General or diffused baldness, when not preceded by disease, is physiological, usually antedating grayness as an evidence of age, and its appearance at an early period is a somewhat frequent hereditary trait. Rapid loss of hair is a common occurrence after acute febrile diseases, more particularly typhoid fever, gout, and erysipelas. Falling out of the hair, either general or circumscribed, is one of the symptoms of syphilis. In chronic hydrocephalus the hair is almost invariably thin. Frequent and severe neuralgias of the fifth nerve are sometimes followed by loss of hair. In anæmia and phthisis pulmonalis, and usually in myxœdema, the hair may become scanty.

Circumscribed areas or patches of baldness may be due to ringworm (*tinea tonsurans*) or alopecia areata. Scars upon the scalp are usually destitute of hair. A marked bald patch upon the back of an infant's head is suggestive of rickets, because of the decided tendency in this disease to a constant rolling of the head upon the pillow. This condition is seen to a slighter extent in almost all infants.

VIII. **Edema or Swellings of the Face.**—(*a*) The face shares in the condition of general dropsy or anasarca, especially that which is due to renal disease. Puffiness or edema of the face as a whole may be due to emphysema (toward the close), pneumothorax, chronic interstitial pneumonia, and mediastinal tumours. In pertussis it may be present from the frequent interference with the return circulation caused by the violent expiratory efforts during paroxysms of cough. The face is more or less characteristically swollen in erysipelas, measles, variola, dengue, and trichinosis. The enlargement of the face in myxœdema is due to a thickened condition of the skin and subcutaneous tissues, which resembles edema but does not pit on pressure.

(*b*) Localized edematous and usually fugitive swellings of the face may be due to urticaria or angioneurotic edema, or may occur as an intercurrent symptom of exophthalmic goitre.

(*c*) Swelling and puffiness of the forehead may occur in glanders or thrombosis of the superior longitudinal sinus.

(*d*) Swelling over the upper jaw may be due to alveolar abscess, phosphorus necrosis, or disease of the antrum.

(*e*) Swelling of the lower jaw may be accounted for by alveolar abscess or actinomycosis.

(*f*) A swelling in front of the ear, extending downward behind the angle of the jaw, at first unilateral but later appearing also on the other side, is due to parotitis, usually epidemic (mumps). Owing to the position of the gland, the lower portion of the ear is rather characteristically pushed outward.

(*g*) A tender edematous swelling over the mastoid process may be due to involvement of the mastoid cells in the course of an otitis media, or to thrombosis of the lateral sinus.

(*h*) The cheeks are sometimes swollen as a result of the gingival conditions in scurvy, and in gangrenous stomatitis (*cancrum oris*) there is a great and brawny infiltration of the cheeks and lips. Furuncles and anthrax (malignant pustule) may also be the cause of inflammatory swellings seated upon some portion of the face.

IX. **Abnormal Movements of the Head.** (*a*) *Nodding Spasm.*—A rhythmic nodding movement of the head may be a form of habit spasm or of epilepsy in children. In the latter case it is accompanied by a momentary loss of consciousness. It may be indicative of rachitis, and occurs in hysteria, either alone or as a part of the salaam convulsion (rhythmic or hysterical chorea). Pulsating shaking of the head is occasionally seen in arteriosclerosis.

(*b*) *Spasmodic Torticollis* (*clonic*).—A spasmodic jerking of the head, recurring every few minutes, in which the head is brought toward one shoulder, and at the same time the face is rotated to the opposite side and the chin raised, is the clonic form of spasmodic torticollis. The shoulder may be simultaneously jerked upward to meet the head. The movements of the head in chorea are by contrast extremely irregular and bizarre, and the facial muscles are affected before those of the neck.

X. **Abnormal Fixity of the Head.**—(*a*) *Inability or disinclination to move the head*, especially if the patient is prone to support it by the hands, may be due to tuberculous disease of the cervical vertebræ, and if associated with dysphagia it points to disease of the articulation between the atlas and occiput.

(*b*) *Retraction of the Head.*—Drawing back and fixation of the head by contraction of the posterior cervical muscles, so that the occiput bores into the pillow, is a symptom of all forms of cerebral and cerebro-spinal meningitis, particularly when affecting the basal meninges. It is also seen in tetanus and strychnine poisoning as a part of the general tonic spasm. In nervous infants a transient retraction of the head accompanies a fit of crying from temper or pain, especially the pain caused by acute indigestion.

(*c*) *Other causes of abnormal fixity* are occipito-cervical myalgia, post-pharyngeal abscess, congenital and spasmodic (tonic) torticollis, rheumatism (acute, chronic, gonorrhœal), arthritis deformans, swollen and painful cervical glands, contracted cicatrices (especially from burns), and sprain of the cervical muscles or other traumatism of the neck.

XI. **Facial Spasm.**—Spasmodic contraction of the facial muscles may be tonic (continuous) or clonic (intermittent), unilateral or bilateral, and involving one or all of the muscles which are innervated by the facial nerve. In order to form an opinion of the cause of spasmodic affections of the face, it is advisable to take into consideration age, sex, and possible neuropathic diathesis, and to ascertain the presence or absence of disease of the eyes, teeth, skin, and possibly of uterine disease and intestinal parasites. In some cases the facial symptoms constitute a comparatively insignificant part of a disease of the nervous system.

The diseases or conditions which may be indicated by facial spasm are mimic spasm, habit spasm, convulsive tic, blepharospasm, nictitating spasm, exophthalmic goitre, chorea, epilepsy, tetanus, meningitis, hysteria, spasm following paralysis, irritation at the root of the facial nerve and lesion of its cortical centre.

(*a*) *Mimic Spasm.*—A more or less constant twitching of one side of the face with partial closing of the eye, occurring in an adult, is mimic spasm. It is usually bilateral, and ordinarily does not begin until adult age.

(*b*) *Habit Spasm.*—If in a neurotic child, usually a girl from seven to fourteen years of age, there is a sudden wink of the eye or a rapid drawing of the mouth to one side, or a sniff, occurring at irregular intervals and greatly intensified in severity by emotional causes, it is habit spasm.

(*c*) *Convulsive Tic.*—In an hereditarily neurotic child, irregular movements of the facial muscles, and frequently those of the arm, if accompanied by an explosive ejaculatory utterance of profane or obscene words (*coprolalia*), or the involuntary repetition of a word (*echolalia*), or the mimicking of an action (*echokinesis*), is convulsive tic or Gilles de la Tourette's disease.

(*d*) *Blepharospasm.*—A tonic (persistent) closure of the eye is ordinarily due to some ocular disease causing photophobia and spasmodic contraction of the orbicularis palpebrarum. Very seldom it is a symptom of hysteria.

(*e*) *Winking Spasm.*—Rapid clonic contractions of the orbicularis palpebrarum (winking or nictitating spasm) is, in the absence of ocular disease, either habit spasm or hysteria.

(*f*) *Exophthalmic Goitre.*—A clonic spasm of the levator palpebræ superioris resulting in successive rapid liftings of the upper eyelids is an occasional symptom (Abadie's sign) of exophthalmic goitre.

(*g*) *Chorea.*—The facial muscles are more or less affected by irregular jerking movements in chorea; but the diagnosis is made by the associated symptoms.

(*h*) *Epilepsy, Meningitis, Tetanus.*—In epilepsy there are tonic followed by clonic spasms of the facial muscles, but the simultaneous presence of general convulsions will establish the diagnosis unless it happens to be a variety of *petit mal.* In the latter case the diagnosis of epilepsy may be determined by the occurrence of a momentary loss of consciousness, biting of the tongue, or the presence of a preliminary aura. In meningitis and tetanus the facial spasm is usually tonic, and is overshadowed by the associated symptoms.

(*i*) *Hysteria.*—The facial spasm of hysteria is usually tonic, less commonly clonic, and requires for the diagnosis of its character a careful search for corroborative symptoms.

(*j*) *Spasm following Paralysis.*—Facial spasm or contracture, generally tonic and sometimes permanent, may follow facial paralysis. The latter, in the vast majority of cases, is unilateral. If the paralysis is hysterical the tonic contraction affects the muscles of the non-paralyzed side of the face; if due to other causes, the contracture ordinarily takes place in the paralyzed muscles during or subsequent to the recovery of their power.

(*k*) *Irritation at Root of Facial.*—Clonic unilateral spasm or twitching of one or more of the facial muscles may be caused by irritation of the facial at its emergence by the pressure of a tumour of the base of the brain, or an aneurism of the vertebral artery. The diagnosis is usually made *post mortem.*

(*l*) *Tic Douloureux.*—Spasmodic movements of the face may occur in this disease, but simply as incident to the pain. Movements of the eyes and facial muscles frequently complicate nodding spasm (*q. v.*).

XII. **Facial Paralysis.**—Paralysis of the muscles of the face is almost always unilateral, very rarely bilateral.

In unilateral facial paralysis the affected side of the face has a smooth, expressionless appearance, due to the obliteration of the wrinkles which normally give it character. The mouth is drawn to the sound side. There is an inability to pucker the lips as in whistling, and labial sounds are bunglingly articulated. In eating, food collects between the teeth and the cheek. During sleep the cheek flaps with inspiration and expiration because of the paralysis of the buccinators, and the nostril of the paralyzed side does not dilate upon forced inspiration.

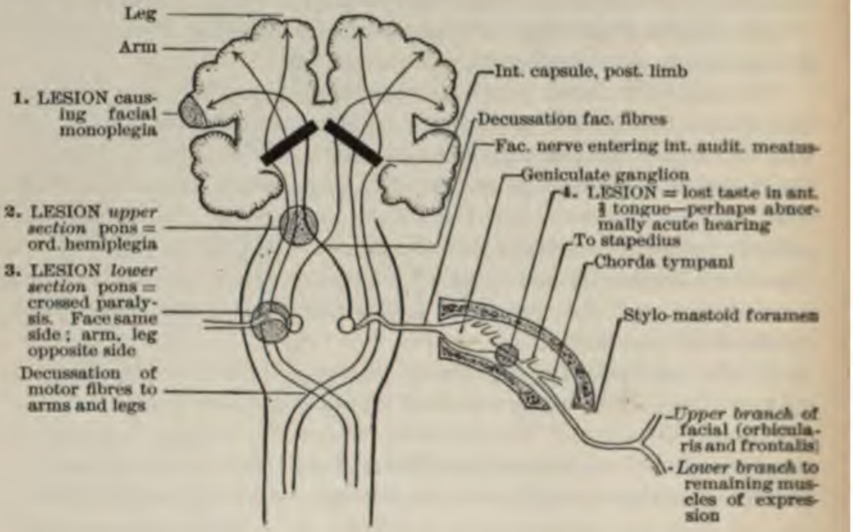
If the paralysis is of *peripheral* origin, involving the facial nerve only and not constituting a part of a hemiplegia, the eye can not be completely closed, and the forehead can not be wrinkled, owing to paralysis of the orbicularis palpebrarum and the occipito-frontalis. The tongue, if protruded, does not in reality deviate from the middle line, as may be determined by watching its relation to the central upper incisors, the genio-hyo-glossus not having been affected. The apparent deviation is due to the pulling of the mouth to the sound side.

If the paralysis is of *central origin*, the upper branch of the facial is but slightly affected, the power of closing the eye and wrinkling the forehead is largely retained, and the tongue deviates toward the paralyzed side, as the genio-hyo-glossus usually loses its power and its healthy fellow is unopposed.

The best test of facial paralysis is to request the patient to close the eyes tightly and to raise the upper lip so as to expose the teeth. If complete facial paralysis is present, the eye will not close, the eyeball rolls upward, and the difference between the two sides of the face becomes very noticeable. This device should always be employed, as otherwise, in the case of children or plump, smooth-faced adults, a slight facial paralysis may escape detection. Moreover, in the course of time secondary contractures may occur in the paralyzed muscles which will restore the wrinkles, and, unless the other muscles are exerted, may deceive the observer into mistaking the affected side for the sound one.

Having determined the presence of unilateral facial paralysis, it is desirable, if possible, to determine its cause. The facial nerve supplies all the muscles of the face, except those of mastication, which are innervated by the motor branch of the fifth and the stapedius, stylo-hyoid, buccinator, and platysma myoides.

Paralysis of the facial nerve (Fig. 44) may be *central* (supranuclear), due to a lesion affecting the cortical centre of the fibres which run from it to the nucleus; or *nuclear*, a lesion involving the nucleus itself; or *infranuclear*, caused by disease or injury of the nerve after it leaves the nucleus, either while it pursues its course through the pons, or while in the bony canal, or after emerging from the stylo-mastoid foramen and penetrating the substance of the parotid gland on the way to its final distribution.


Leg
Arm
1. LESION causing facial monoplegia
2. LESION *upper section* pons = ord. hemiplegia
3. LESION *lower section* pons = crossed paralysis. Face same side; arm, leg opposite side
Decussation of motor fibres to arms and legs
Int. capsule, post. limb
Decussation fac. fibres
Fac. nerve entering int. audit. meatus
Geniculate ganglion
4. LESION = lost taste in ant. ⅔ tongue—perhaps abnormally acute hearing
To stapedius
Chorda tympani
Stylo-mastoid foramen
Upper branch of facial (orbicularis and frontalis)
Lower branch to remaining muscles of expression

Causes affecting Centre, or Fibres to Nucleus.	Causes of Peripheral Paralysis affecting Nerve.			
	In pons.	Bet. pons and int. meatus.	In aqueduct.	At and beyond stylo-mastoid foramen.
Hemorrhage. Embolism. Thrombosis. Softening. Tumours. Abscess.	Hemorrhage. Tumour. Softening. Diphtheria. Bulbar paralysis. Locomotor ataxia.	Tumours. Gummata. Basal meningitis and endarteritis. Fracture at base.	Inflammation or necrosis of bone due to disease of ear. Fracture petrous portion of bone.	Injury. Parotid swelling. Multiple neuritis. Peripheral neuritis (rheumatic).

Fig. 44.—Showing the varieties and causes of paralysis of the facial nerve.

The first essential in determining the cause of a given case of facial paralysis is to ascertain whether it is of central or peripheral origin. With rare exceptions, if the lesion is central, the eye can be closed and the forehead wrinkled, especially under the influence of emotion. Moreover, the normal response to the faradic and galvanic currents is preserved, and the reaction of degeneration does not appear, because the nucleus still exerts its trophic influence upon the paralyzed muscles. On the other hand, if the lesion is peripheral, affecting the nucleus or the nerve trunk, the orbicularis and frontalis are powerless, the response to faradism disappears, and the galvanic excitability (*q. v.*) is qualitatively altered. The muscles degenerate because they are cut off from the trophic stimulus of the nucleus.

(*a*) *The Central Causes of Facial Paralysis.*—In the great majority of cases facial paralysis of central origin is associated with hemiplegia, and, like the latter, is due to intracranial hemorrhage, embolism, or thrombosis, with softening (less frequently tumour and abscess), affecting the cortex or the facial fibres as they pass down in the corona radiata or the internal capsule. The facial paralysis is on the same side as that of the arm and leg, because the fibres from the cortex to the nucleus decussate before entering the nucleus, and the facial muscles consequently bear the same relation to the cortex as do the muscles supplied by the spinal nerves. The determination as to which of the central lesions is causing the paralysis must be made by the history and associated symptoms. If there is facial palsy, presenting the characters of a central paralysis and not associated with hemiplegia, it is due to a lesion of the face centre in the cortex.

(*b*) *Nuclear and Peripheral Causes of Facial Paralysis.*—(1) Nucleus alone.—Lesions affecting the nucleus alone are not common but may occur as a result of hemorrhage, tumour, and chronic softening, or in the course of diphtheria, anterior poliomyelitis, and bulbar paralysis.

(2) Crossed paralysis.—A special form of motor disturbance is *crossed paralysis*—palsy of the face on one side and of the arm and leg on the opposite side, the facial paralysis being in this case on the same side as the lesion. If this condition is present, it is indicative of a lesion in the lower part of the pons, involving the nerve between its nucleus and the point upon the side of the pons from which it emerges. In this form of paralysis the location of the lesion is very definitely in the lower portion of the pons, for, if it is in the upper portion, the facial paralysis will be upon the same side as that of the arm and leg (ordinary hemiplegia), because in this case the facial fibres, like those of the arm and leg, are invaded above their point of decussation.

(3) At its *point of emergence* from the pons (Figs. 45 and 46) the nerve may be involved by tumours, gummata, basal meningitis, or endarteritis of syphilitic origin and fracture of the base. Syphilis is the most important factor in these basal lesions, which are always accompanied by evidences of involvement of other cranial nerves (Dana).

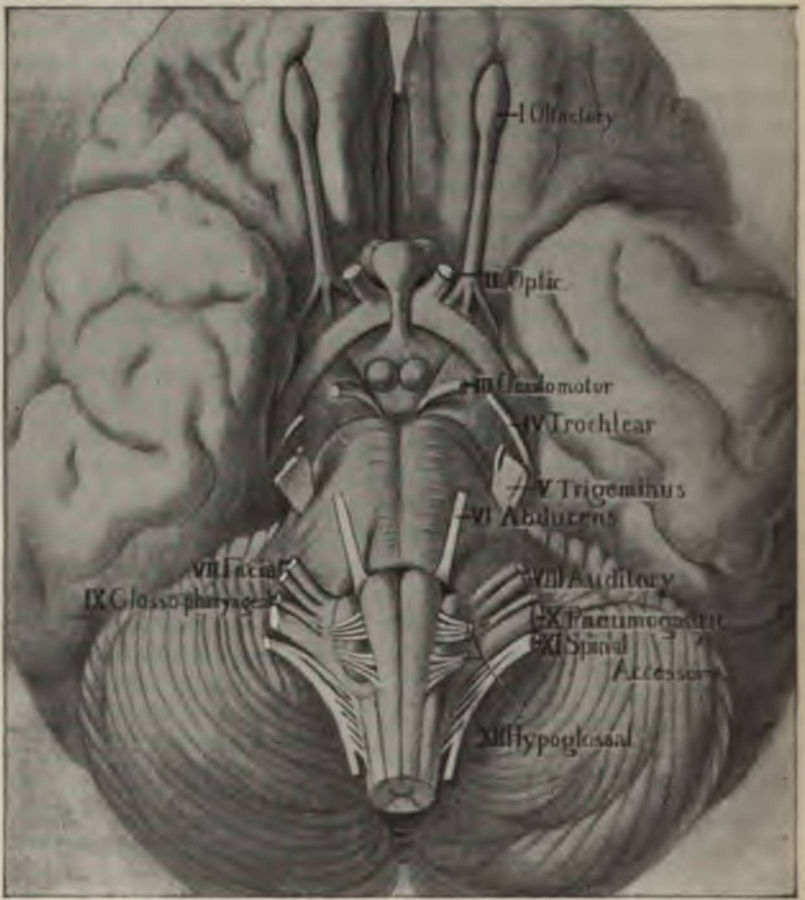


Fig. 45.—The base of the brain. Redrawn from Gray.

(4) During its *course through the bony canal* (aqueduct of Fallopius), the nerve may be paralyzed by fracture of the petrous portion of the temporal bone, or by inflammation or caries of the same, due to otitis media.

(5) At or after its *emergence from the stylo-mastoid foramen* the nerve may be injured by blows; by accidental cutting during the

removal of enlarged cervical glands, parotid or other tumours; and at birth by direct pressure of the forceps. In the latter instance it should be remembered that the paralysis may be due to intracranial hemorrhage, but in this case other paralyses usually coexist. The nerve may also be so pressed upon by swelling of the parotid gland that it loses its function.

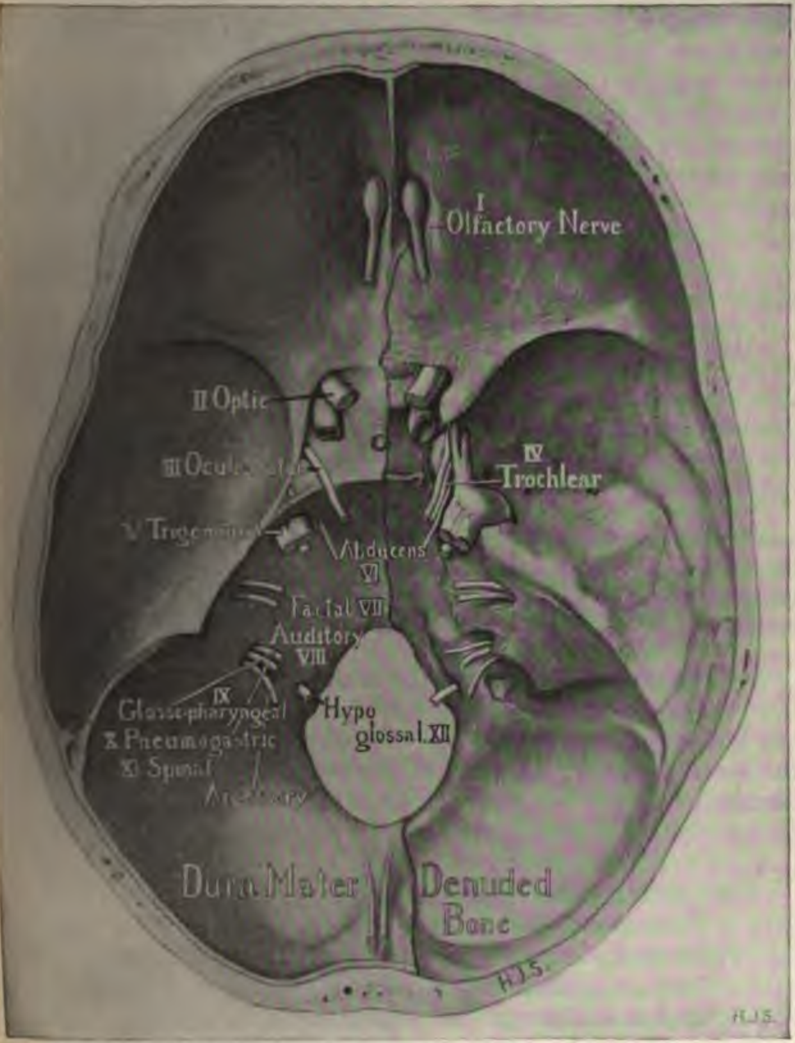


FIG. 46.—Points of exit of the cranial nerves from the skull. Redrawn from Henle.

(6) Paralysis of the facial may be present as a part of mul-

tiple neuritis, locomotor ataxia (lesion affecting the pons), and hysteria.

(7) As the most common cause of central facial paralysis is either hemorrhage or tumour, so the majority of cases of peripheral facial paralysis are due to exposure to cold and consequent diffuse rheumatic neuritis of the facial nerve (Bell's palsy, *q. v.*). The neuritis may in one instance affect mainly the peripheral terminations of the nerve, in another that portion lying in the Fallopian canal.

(8) To determine the particular form of disease which is responsible for the paralysis requires a review of the history and accompanying symptoms which may point to the conditions mentioned as possible causes—e. g., sudden hemiplegia and unconsciousness in an old person with atheromatous arteries; or vertigo, vomiting, convulsions, and paralysis of other cranial nerves in tumour of the brain.

The location of a peripheral lesion may be ascertained within certain limits by testing the sense of taste in the anterior two thirds of the tongue. If the sense of taste is partly or entirely lost, the lesion must be between the geniculate ganglion of the nerve and the point where the chorda tympani is given off. If it is not lost, the lesion is either central to the genu or peripheral to the chorda. In the greater number of cases it is peripheral.

The hearing power of the ear on the paralyzed side may be abnormally acute or less than normal. If abnormally sensitive, it indicates that the lesion extends into the Fallopian canal and affects the nerve to the stapedius, paralyzing it and allowing the unopposed tensor tympani to tighten the drum membrane. If the hearing is impaired, it is usually due to otitis media.

(*c*) **Bilateral or Double Facial Paralysis** (*Facial Diplegia*).—This is of very rare occurrence, and when present is caused by bilateral and symmetrical cortical lesions: tumour or gummatous disease at the base of the brain, disease of the pons—viz., a lesion at the point of decussation of the face fibres, disease of the basilar artery, acute and chronic bulbar paralysis, diphtheritic multiple neuritis, and double mastoiditis of otitic origin.

XIII. **Miscellaneous Affections of the Head and Face.**—(*a*) *Syphilitic or Tuberculous Ulceration.*—Indurated grayish spots upon the face which break down, giving rise to deep, scooped-out ulcers with hard, thickened borders, and leaving smooth, white, circular cicatrices, are ulcerated subcutaneous gummata. It is sometimes difficult to distinguish an ulcerating gumma from a tuberculous ulcer, but in the latter the ulcer is more shallow, the edges are flat and soft, and the scar reddish or purple. If the diagnosis is uncertain, an attempt may be made to find the tubercle bacillus or the therapeutic test may be applied.

(*b*) *Herpes Zoster.*—One or more groups of small vesicles seated upon an inflamed base, attended by a burning or neuralgic pain, and occurring along the course of one or more branches of the fifth nerve, is herpes zoster.

(*c*) *Distended Veins.*—Distended or enlarged veins upon the scalp may be due to tumours of the neck, thrombosis of the lateral sinus, or meningitis. They are especially prominent in chronic hydrocephalus.

(*d*) *Erysipelas of the Face and Head.*—Redness beginning over the bridge of the nose or at the site of an abrasion, spreading rapidly over the face and scalp, the advancing edge being well defined, causing great œdema of the face, eyelids, ears, and scalp, which sadly alters the patient's appearance, and attended with sudden, usually high, fever, is erysipelas (*Streptococcus pyogenes*).

(*e*) *Excessive Sweating* of the head in a child is suggestive of rachitis (*q. v.*), although it is observed to a considerable extent in many children who are not rachitic.

SECTION XV

THE EAR

I. **Pain.**—(*a*) *Otitis Media.*—By far the most frequent cause of severe pain in the ear (earache) is an otitis media secondary to scarlet fever, epidemic influenza, acute simple rhino-pharyngitis or tonsilitis, measles, diphtheria, and typhoid fever. It is most common in children and may explain an obscure infantile illness.

(*b*) *Other Causes of Earache.*—Pain in the ear may also be due to decaying teeth, alveolar abscess, mastoid disease, abscess of the meatus, foreign body in the ear, neuralgia, rheumatism of temporo-maxillary articulation, and, if in the right ear, aneurism of the innominate. Ear pain is sometimes due to ulcer or cancer of the tongue.

II. **Colour.**—(*a*) *Waxy.*—Abnormally white or waxy ears are usually due to conditions in which pallor (*q. v.*) is a symptom.

(*b*) A *blue-black* discoloration of the cartilages of both ears is a symptom of ochronosis (usually with alkaptonuria).

(*c*) *Blue.*—A livid or bluish tint of the external ear is usually indicative of general cyanosis and its causes (*q. v.*). A frost-bitten ear is at first blue, but when frozen is waxy white.

(*d*) *Ecchymosis with Swelling.*—A swollen black and blue pinna may be due to injury, but may also be an example of that curious trophoneurosis, *hæmatoma auris* of the insane.

III. **Shape.**—(*a*) *Stigmata of Degeneration.*—Ears unusually prominent, long, or misplaced, with absence of helix, antihelix, or lobule, are marks of degeneration or imperfect physical development.

(*b*) *Tophi.*—Small hard nodules, found usually on the borders of the ear, are chalky masses of sodium urate of gouty origin and may be so numerous as to cause deformity.

(*c*) *Abscess of Meatus, etc.*—If the external ear is swollen so that it stands out from the head, and if on inspection the meatus is found to be inflamed and almost closed, with absence of tinnitus and vertigo, it is an abscess of the external meatus. The calibre of the meatus may also be lessened by periostitis, exostoses, and sebaceous cysts.

IV. **Discharges.**—(*a*) *Blood.*—A fracture of the base of the skull may cause an aural hemorrhage, followed by the escape of a considerable amount of clear cerebro-spinal fluid from the subarachnoid space. Rupture of the tympanic membrane, either as a result of injury or consequent upon an otitis media, may be responsible for a discharge of blood—in the latter case mixed with pus. Hemorrhage from the ear, especially in the newborn, may be an evidence of hæmophilia. It may occur in caisson disease.

(*b*) *Pus.*—A flow of pus—otorrhœa—may be either acute or chronic, and, while it may be due to a polypus or abscess of the external meatus, it is, in the great majority of cases, an evidence of otitis media with or without complications. A slight moisture may be indicative of eczema or impacted cerumen in the meatus.

V. **Hearing.**—(*a*) *Tinnitus.*—Subjective sounds heard in the ear (*tinnitus aurium*), or with much less frequency vaguely located in the head (*tinnitus cerebri*), are of varying intensity and character. As a rule, the sound is heard on one side only. It is a very common symptom, and may be due to so many conditions that in a given case it is often difficult and sometimes impossible to determine its etiology.

The character of the sounds may be of service in ascertaining their cause (DANA), although too much reliance must not be placed upon this as a diagnostic factor.

If they are of a boiling, bubbling, gurgling, singing, or whistling quality, their origin is likely to be found in rhino-pharyngeal catarrh with involvement of the Eustachian tube and middle ear, or some source of irritation in the external meatus. If the sound is of a constant rushing or knocking character, it may be due to congestion, inflammation, or hemorrhage of the labyrinth. If the tinnitus is like a systolic bruit, synchronous with the heart beat, and is stopped by carotid compression, it is presumably due to vasomotor paralysis, aneurism, or inflammation of the middle ear. If the sound is of a ringing or roaring description, it is probably caused by chronic middle-ear catarrh, tympanic disease, syphilis, brain tumours, or meningitis.

If the noise is referred to the head (*tinnitus cerebri*) rather than the ear, and neither deafness nor aural disease be present, it is in all likelihood caused by arteriosclerosis or meningitis. If the tinnitus is of a complicated character—viz., musical sounds or words—it is indicative of some central lesion.

It is helpful to bear in mind that the *most frequent* causes of tinnitus aurium are: neurasthenia and the neuropathic diathesis, with vasomotor irregularity; diseases of the ear, as impacted cerumen, otitis with fluid in the middle ear and obstruction of the Eustachian tube; blood diseases, as anæmia (with coincident cerebral anæmia) and leucæmia; toxæmic states, as in gout, nephritis, alcoholism, and the overuse of tobacco; the arteriosclerosis of old people; and disturbances of the digestion.

Other and *less frequent* causes are as follows:

Hypertrophy of the heart and aneurism of the cerebral, basilar, or vertebral arteries may cause a pulsating tinnitus. Tinnitus may constitute the premonitory aura of an epileptic seizure or initiate an attack of catalepsy. It is one of the multiform manifestations of hysteria. Noises in the ear, due perhaps to local disease, may be the basis of aural hallucinations in delirium tremens and insanity. Associated with vertigo and deafness it is one of the prominent symptoms in Ménière's disease. An attack of migraine is frequently preceded or accompanied by sudden loud noises, perhaps like a pistol shot. Buzzing or ringing in the ears may precede an attack of intracranial hemorrhage or thrombosis. Tinnitus is a rare occurrence in organic disease involving the auditory nucleus or tract, e. g., tumour. It is not uncommon as a tinnitus cerebri in meningitis, especially chronic pachymeningitis following sunstroke, chronic alcoholism, or blows upon the head. Tinnitus with deafness may be due to the administration of the cinchona alkaloids, as well as salicylic acid and its compounds. Ergot in considerable doses may give rise to ringing in the ears because of its probable action in producing cerebral anæmia. Various subjective sounds, due to toxæmia, circulatory disturbances, or aural disease, may be present in some of the *infections*, particularly cholera, malarial, typhoid, and typhus fevers.

(*b*) *Deafness*.—In the majority of cases deafness depends upon disease of the tympanum, middle ear, or Eustachian tube. It may, however, be due to disease of the auditory nerve, its nucleus, or its cortical centre, in which case it is spoken of as "nervous deafness."

The distinction between ordinary deafness and the nervous form is made by testing with the watch or the tuning fork.

If the ticking of the watch or the vibrations of the tuning fork are heard faintly or not at all when held at varying distances from the ear (aërial conduction), but become distinctly audible when the watch or the handle of the fork is placed in contact with the skull or mastoid process (bone conduction), the deafness is of the ordinary variety and due to aural disease. If, on the other hand, watch and fork are heard indistinctly or not at all, both in contact and at a distance, the deafness is due to some lesion of the nerve or its connections. In the first case the nerve is normal and can appreciate vibrations brought by the bone, while, through some fault in the mechanism, aërial vibrations are not transmitted to the nerve endings. In the second case the nerve is at fault and can not appreciate vibrations, no matter how well they may be conducted.

Nervous Deafness.—The presence of nervous deafness having been determined, its possible causes must be remembered. The lesions producing it may affect the cortical centre, the nucleus and root of the nerve, or its terminations in the labyrinth.

The eighth nerve has a double function. The *cochlear* branch is the nerve of hearing; the *vestibular* branch, which supplies the semicircular canals, is the space sense nerve. The cortical centre for the auditory nerve is situated in the 1st and 2d temporal convolutions.

(1) Cortical lesions.—As a clinical curiosity nervous deafness of both ears may be caused by bilateral lesions of the cortical centres. Disease of the left cortical centre produces word deafness—inability to understand the meaning of words, although the sounds may be heard. A lesion of the right cortical centre may give rise to defective hearing in the left ear. Loss of hearing due to hysteria is of cortical origin. Headache, mental strain, and "nervousness" may produce a temporary diminution of auditory acuity, although the variation from these causes is usually in the direction of hyperacusis, or, more properly, dysacusis, when ordinary sounds act as irritants.

A lesion of the corpora quadrigemina, or the internal capsule, may involve the fibres leading to the nucleus and thus impair the power of audition. The particular lesions which may occur here are tumour, abscess, hemorrhage, or cortical meningitis.

(2) Lesions of the nucleus and the root of the nerve.—Disease of the nucleus itself is rare, but at the base of the brain (Figs. 45 and 46) the nerve may be compressed or injured by tumours, hemorrhage, fracture of the base, violent blows upon the head without fracture, and meningitis. Of these causes the more frequent are syphilitic

meningitis in the adult, and epidemic cerebro-spinal meningitis in children. In the latter the deafness may be permanent and, if in an infant, deaf-mutism may result. The nerve may undergo primary degeneration in locomotor ataxia.

(3) Labyrinthine disease.—In the great majority of cases nervous deafness is due to a lesion, either primary or an extension of middle-ear disease, affecting the terminations of the nerve in the internal ear—the end organ. There may be syphilitic disease; inflammation, hemorrhage, or tumour of the labyrinth; tuberculous disease or injury of the petrous bone; and atrophy of the nerve endings in locomotor ataxia. Quinine, salicylic acid, or the salicylates may be responsible for the deafness by causing labyrinthine hyperæmia. Ménière's disease, as limited by some, is due to a lesion of the labyrinth. The constant noise and jarring incident to certain occupations (railway engineers, boiler makers) may produce nervous deafness. Loud reports or explosions have a similar effect, usually temporary unless the concussion is sufficiently violent to inflict a traumatism. Some of the fevers, notably typhoid and typhus fevers, the septicæmic form of pneumonia, and diphtheria, are attended with marked impairment of hearing without middle-ear disease.

The determination of the special lesion in nervous deafness must be made from a comparison of the associated signs and symptoms, viz., those of tumour or abscess of the brain, intracranial hemorrhage, or basal meningitis, affecting the cortex, nucleus, or nerve root, together with a careful examination of the ear in labyrinthine disease. Sudden total deafness is very suggestive of syphilis of the internal ear. Hysterical deafness is to be distinguished by its affecting one ear only; by its suddenness of onset, usually following strong emotion or shock; by its incompleteness, the failure concerning mainly high and low notes; by the equal diminution in air and bone conduction; by the presence of hysterical stigmata (*q. v.*), and perhaps by a return of hearing as sudden as its loss.

Non-nervous Deafness.—Of all cases of deafness by far the greatest number are due not to affection of the nerve, but to some defect in the auditory mechanism for conveying vibrations to the end-organ.

The diseases and conditions of the ear which will usually be found responsible for loss or impairment of the power of hearing are middle-ear inflammation resulting from simple or specific infectious rhino-pharyngitis, postnasal adenoids, enlarged faucial tonsils, nasal polypi, and nasal stenosis from any cause. Wax in the meatus, aural polypi, parotitis, or other inflammations or growths may produce deafness by closing the external meatus.

(*c*) *Hyperacusis.*—An extraordinary acuteness of the sense of hearing (*hyperacusis*) may be symptomatic of hysteria, facial paralysis with loss of power in the stapedius muscle, and hypnosis.

Allied to hyperacusis is *dysacusis*—the causing of unpleasant sensations by ordinary sounds—which may be significant of the cerebral congestion of fevers, or an irritable condition of the nervous system during convalescence from febrile diseases or in debilitated persons. It is a rather frequent complaint in hysteria, neurasthenia, hypochondriasis, migraine, and severe headaches. It may be present in epilepsy, meningitis, and tetanus.

SECTION XVI

THE EYE

THE signs and symptoms referable to the eye are of importance, not only with reference to the disorders of the nervous system but also in connection with diseases of other organs. These signs and symptoms will be enumerated in the usual order of a clinical examination.

I. THE EYELID

(*a*) *Swelling, Puffiness.*—The eyelids, particularly the lower, are swollen in general anasarca, and are usually the earliest seat of œdema of renal origin. Anæmia is not infrequently attended by swelling of the lids. A bloated appearance of the eyes, possibly with ecchymoses, is common in the later stages of a severe pertussis. The effect of full doses of arsenic in causing puffiness of the lids is familiar. Angioneurotic œdema may be seen in this locality, and is perhaps analogous to the œdema of the lids and forehead which infrequently occurs in neurotic adolescents.

Because of the anatomical connection between the vessels of the face and those of the cranial cavity (Figs. 47 and 48), cerebral thrombosis may give rise to a swelling of one or both eyelids, with some protrusion of the eyeball, the puffiness subsequently extending to the face.

Erysipelas and glanders, the former especially, may be the cause of swelling so great as to prevent opening of the eye. Severe coryza, hay fever, and iodism may induce noticeable swelling of the lids. So, also, the lids are swollen in measles, variola, and occasionally in varicella.

(*b*) *Hordeolum.*—A minute, painful abscess at the edge of the eyelid is a sty or hordeolum. If occurring in successive series, they may be indicative of the overuse of defective eyes, or more frequently of digestive or genital disorders. If small, single, and superficial, they are of purely local origin. They must be differentiated from chalazion (cyst of a Meibomian gland), a slow-growing tumour which may become inflamed and suppurate.

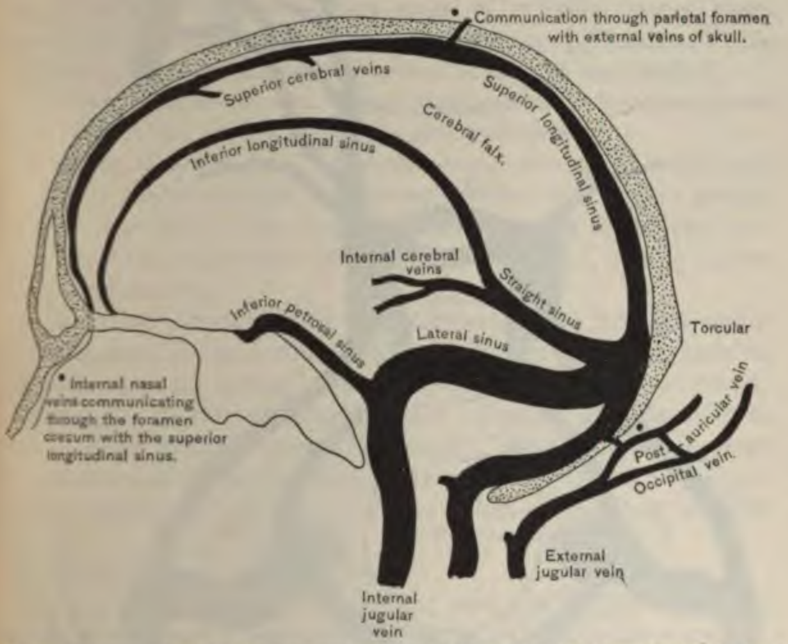

Communication through parietal foramen with external veins of skull.
Superior cerebral veins
Superior longitudinal sinus
Cerebral falx.
Inferior longitudinal sinus
Internal cerebral veins
Straight sinus
Inferior petrosal sinus
Lateral sinus
Torcular
Internal nasal veins communicating through the foramen cæcum with the superior longitudinal sinus.
Post. auricular vein
Occipital vein
External jugular vein
Internal jugular vein

Fig. 47.—Diagram showing the communications (indicated in the figure by black dots) existing between the superior longitudinal and lateral sinuses and the external veins. Redrawn from Leube.

A cystic swelling, of some standing, toward the inner canthus, is a mucocele (chronic dacryocystitis), which may become inflamed and suppurate. And there may be an acute inflammation of the lachrymal sac following measles, scarlet fever, ophthalmia, or other diseases in which there is conjunctival inflammation.

(*c*) *Blepharitis.*—If the edges of the eyelids are reddened, thick, inflamed, and crusted with secretions, possibly showing minute ulcers or pustules, the condition is a blepharitis resulting from a previous ophthalmia or an attack of measles. It is usually indicative of anæmia and a catarrhal, perhaps tuberculous, diathesis.

(*d*) *Verruca.*—Warts upon the eyelids are not common, and are only of importance in old people because of the possibility of commencing epithelioma.

(*e*) *Ulcers.* 1. *Epithelioma.*—In an elderly person a shallow, flat ulcer on the nasal side of the lower lid, usually concealed by a scab,

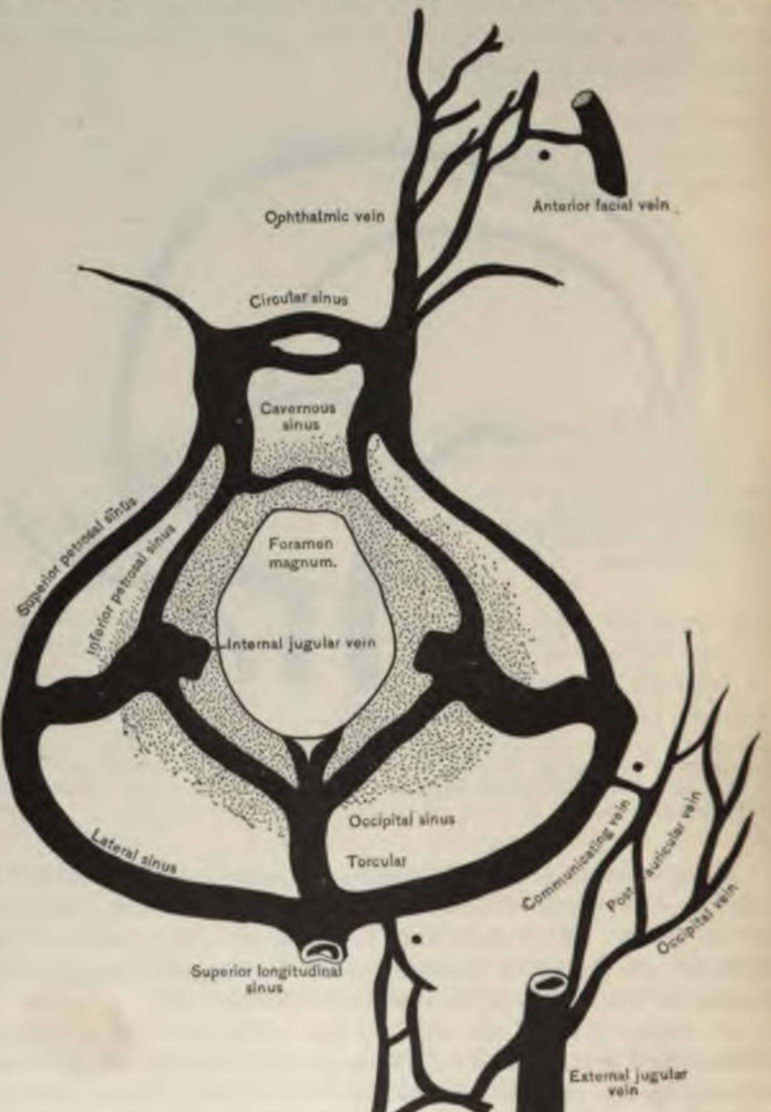


FIG. 48.—Diagram showing the communications (indicated in the figure by black dots) between the lateral and cavernous sinuses and the external veins. Redrawn from Leube.

and having lasted for several years, is quite certainly an epitheliomatous ulcer.

2. *Syphilis.*—The initial lesion of syphilis may occur on the eyelid in the shape of a small, moist, slightly ulcerated surface, with considerable induration and swelling. The diagnosis can usually be made with certainty only by the secondary symptoms. A very rare lesion is an ulcer in the tertiary stage of syphilis, deeper, more inflamed, and punched out in appearance than in epithelioma, which is practically the only disease with which it may be confounded.

(*f*) *Tophi.*—As in the ear, so may small nodules of sodium urate form in the eyelids, significant of the gouty diathesis.

(*g*) *Colour of the Lids.* 1. *Xanthelasma.*—One or more soft and very slightly elevated yellow patches on the eyelids of elderly people, varying in size from a mere point to the size of a finger nail, and situated near the inner angle of the eye (if unilateral, usually on the left), is xanthelasma.

2. *Duskiness.*—A darkening or duskiness of the lids and under the eyes is seen in some women during menstruation and early in pregnancy, also in menorrhagia and long-continued leucorrhœa. Duskiness may be very prominent in the anæmia and pallor of brunettes, or after fatigue, mental excitement, loss of sleep, or severe pain, and in exhausting diseases. Dark circles round the eyes constitute a time-honoured, if not always reliable, symptom of self-abuse.

(*h*) *Blepharospasm.*—(See Index.)

(*i*) *Lagophthalmos.*—The unnaturally open eye may be due to the contraction of a scar, to loss of power in the orbicularis palpebrarum, facial paralysis, or exophthalmic goitre. In the latter disease it is conjoined with protrusion of the eyeball (proptosis) and inability to follow with the upper lid a downward movement of the eyeball (von Graefe's sign).

Incomplete closure of the lids during sleep is a common symptom of exhausting diseases, particularly in children. As the eyeballs are normally turned upward while sleeping, the sclerotic is thus exposed, giving a ghastly expression to the face. A similar rolling up of the eye and imperfect closing of the lids is frequently seen in hysterical individuals.

(*j*) *Ptosis.*—Drooping of the upper eyelid (see Index).

II. THE CONJUNCTIVA, SCLEROTIC, AND CORNEA

(*a*) **Colour.** 1. *Yellow Sclerotic.*—Seen in jaundice, and may be due to fatty deposits, triangular in shape, apex at the cornea, and running laterally toward the angles of the eye.

2. *Bluish-white or Pearly Sclerotic.*—This is found in the anæmias, in phthisis, in nephritis, and, by contrast with the discoloured face, in Addison's disease.

3. *Inflamed or Injected Conjunctiva.*—This may be caused by gonorrheal infection or by the bacillus of diphtheria, in which case it is attended with a profuse purulent discharge or the formation of pseudo-membrane. A certain amount of conjunctivitis is present in measles, hay fever, coryza, and influenza of the catarrhal type. Typhus fever presents a brilliantly injected conjunctiva, as does yellow fever to a lesser degree. Meningitis, either simple or tuberculous, is a not infrequent cause of inflammation and discharge, perhaps unilateral. Facial paralysis, by causing undue exposure of the eyeball, may give rise to redness and inflammation.

Neuralgia of the fifth nerve and glaucoma produce conjunctival congestion; so also do full doses of arsenic or potassium iodide, especially if there is an idiosyncrasy. Ulcers of the cornea are attended with reddening of the eye. The possible presence of a foreign body should be remembered.

Subconjunctival hemorrhage or ecchymosis may, after the receipt of an injury, indicate a fracture of the base anteriorly, the blood passing forward into the orbit. The non-surgical causes are the violent straining which may occur in pertussis, severe vomiting, obstinate constipation, or heavy lifting. Subconjunctival ecchymosis may also occur in the epileptic seizure and in asthma or severe dyspnœa. They may be hemorrhagic infarcts and significant of ulcerative endocarditis.

(*b*) **Dryness and Moisture of the Eye.**—The eye may become dry and glazed in collapse or the typhoid status, as well as in disease attended by lagophthalmos.

Lachrymation—an increased secretion of watery fluid—usually accompanies irritation or inflammation of the conjunctiva, and accordingly is present in measles, influenza, pertussis, and typhus fever in the early stages, hay fever, asthma, coryza, trifacial neuralgia, facial paralysis, iodism, and foreign bodies in the eye. The moist eye of the chronic alcoholic is familiar. The tears may overflow the edge of the lids (*epiphora*) because of displacement of the puncta lachrymalis or obstruction of the duct. Cold winds and irritating vapours stimulate the flow of tears.

(*c*) **The Cornea.**—1. *Arcus Senilis.*—Fothergill made a distinction between the true and the false arcus senilis. The true arcus is an ill-defined, grayish, partial or complete ring at the circumference of the cornea, the cornea itself being somewhat hazy. It was formerly wrongly considered to be an indication of beginning arteriosclerosis, gout, chronic nephritis, and the degeneration of old age. The false arcus is a sharply delineated ring of a clear yellow or yellowish-white colour, due to a deposit of fat. Neither one is of special diagnostic significance.

2. *Keratitis.*—Interstitial keratitis is the only variety which is of general medical interest, because of its usual cause—syphilis. The cornea has the appearance of ground glass, with small clearer spots here and there through which the pupil may be indistinctly seen. It is usually symmetrical, and occurs between 5 and 15 years of age. Confirmatory signs should be looked for in the lips, teeth, nasal bones, and general appearance.

It is not to be forgotten that a hazy or steamy cornea, with severe pain around a reddened eye, possibly with nausea and vomiting, and a tense globe, are the symptoms of glaucoma.

3. *Ulcers of Cornea.*—A reddened, painful, photophobic eye, with a clear cornea, which on close inspection shows a slight loss of substance at one point, is an example of a corneal ulcer. This may be found in connection with exophthalmic goitre, and is due to the constant exposure of the eye consequent upon the inability of the lids to cover the protruding eyeballs.

The unilateral inflammation of the eye in meningitis may lead to a corneal ulcer. In disease of the first division of the fifth nerve, with resulting anæsthesia of the cornea and conjunctiva, the cornea may undergo intractable ulceration, due probably to trophic alterations, although latterly the importance of the trophic element has been denied.

III. THE PUPIL

The points to be observed about the pupil are its state of dilatation or contraction, inequality (anisocoria), response to light, response to accommodation, and the skin reflex.

I. **Physiological Conditions.**—(*a*) *Diameter of the Pupil.*—This, under ordinary circumstances, is 4 millimetres, and may be measured by holding a properly graduated rule close to the eye. Commonly the observer is able to detect any important abnormality (both small, both large, unequal) in this respect without artificial aid.

(*b*) *Response to Light.*—The responsiveness of the pupil to light is determined for each eye separately by asking the patient to look at the light from a window. Still keeping the eyes in the same direction, the hands of the physician are placed over and close to, but not touching, the eyes. One hand is then suddenly removed and the behaviour of the pupil noted. Normally the pupil contracts on exposure to light—the *direct reflex.* This hand being replaced, the other pupil is tested in a similar manner. In normal eyes the pupil of the covered eye will also contract—the *indirect reflex*. If particular care in this examination is desirable, artificial light in a dark room should be employed. While the patient with eye covered looks toward the darkest part of the room, the light is brought suddenly before the uncovered eye, at a distance of 3 or 4 feet, so that the pupillary accommodation reflex may be eliminated.

(*c*) *Response to Accommodation.*—As the pupil grows smaller when the eyes converge and the ciliary muscle contracts in the act of accommodation for near objects, this function should also be examined. Let the examiner place his finger tip 12 or 15 inches from the eye, nearly in a line with some object at least 20 feet away, and direct the patient to look first at the finger and then at the distant object until it is decided whether or not the pupil contracts when looking at the finger.

(*d*) *Skin Reflex.*—The pupil also dilates if the skin of the neck is pinched or pricked, because by so doing the cervical sympathetic is irritated.

(*e*) *Physiology of the Iris.*—Contraction of the iris is effected by circular or sphincter fibres which are controlled by a reflex nervous mechanism (Fig. 49), while dilatation is secured by a set of nerve fibres which oppose the action of the oculomotorius upon the circular muscles of the iris.

The balance of the pupil is preserved by the constant action of these nerve mechanisms, which keep the radial and circular muscle fibres of the iris in tonic antagonistic contraction. Irritation of the sympathetic or dilator mechanism, as by pinching the neck, overcomes the constricting mechanism and the pupil grows larger. Paralysis of the sympathetic allows the oculomotorius to act unopposed and the pupil contracts. Similarly stimulation of the oculomotor mechanism will cause contraction, while paralysis produces dilatation of the pupil.

II. **Pathological Conditions.**—There may be, therefore, three varieties of paralysis of the iris, or iridoplegia (with pupils small or of a medium size), as follows:

Loss of response to *light*—reflex iridoplegia.

Loss of response to *accommodation*—accommodative iridoplegia.

Loss of response to *irritation of the skin of the neck*—absent skin reflex.

The abnormalities of the iris and pupil, together with their diagnostic indications, may be stated as follows:

(*a*) *Iris and Pupillary Outline.*—Inflammation of the iris may be indicative of gout, rheumatism, or syphilis. If iritis is present, the iris, normally blue or gray, becomes of a greenish, muddy hue, the pupil responds sluggishly to light and, if the disease is unilateral, is smaller than the other, because of the iritic congestion and consequent swelling. There is a narrow zone of pinkish hyperæmia around the edge of the cornea. If the iris is brown or black, little change of colour occurs. While one iris may normally differ in colour from the other, if one is green and the other blue, the existence of an iritis should be strongly suspected. Rheumatic iritis is usually unilateral and is prone to relapse ; so also is the rarer gouty iritis. Syphilitic iritis occurs only during the secondary stage of the disease, is commonly bilateral, and rarely relapses.

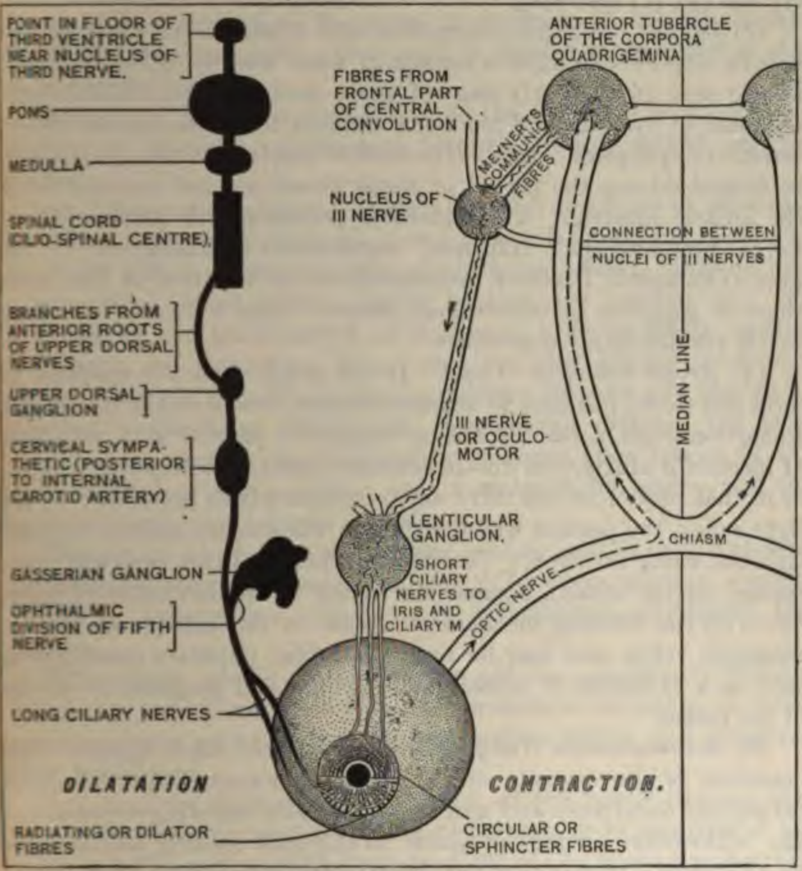

POINT IN FLOOR OF THIRD VENTRICLE NEAR NUCLEUS OF THIRD NERVE.
PONS
MEDULLA
SPINAL CORD (CILIO-SPINAL CENTRE).
BRANCHES FROM ANTERIOR ROOTS OF UPPER DORSAL NERVES
UPPER DORSAL GANGLION
CERVICAL SYMPATHETIC (POSTERIOR TO INTERNAL CAROTID ARTERY)
GASSERIAN GANGLION
OPHTHALMIC DIVISION OF FIFTH NERVE
LONG CILIARY NERVES
DILATATION
RADIATING OR DILATOR FIBRES
FIBRES FROM FRONTAL PART OF CENTRAL CONVOLUTION
MEYNERTS COMMUNIC FIBRES
NUCLEUS OF III NERVE
ANTERIOR TUBERCLE OF THE CORPORA QUADRIGEMINA
CONNECTION BETWEEN NUCLEI OF III NERVES
MEDIAN LINE
III NERVE OR OCULO-MOTOR
LENTICULAR GANGLION.
SHORT CILIARY NERVES TO IRIS AND CILIARY M.
OPTIC NERVE
CHIASM
CONTRACTION.
CIRCULAR OR SPHINCTER FIBRES

Fig. 49.—Nervous mechanism of the iris.

- - - - Path of sensory stimulation through optic tract.
- - - Path of motor impulse through third nerve to sphincter fibres, causing contraction of pupil.
Solid black = path of impulses from point near nucleus of third nerve and cilio-spinal centre through sympathetic to dilator fibres, causing dilatation of pupil.

An irregular outline of the pupil is due to iritis and subsequent adhesions between the posterior surface of the iris and the anterior surface of the lens (posterior synechiæ). A very small pupil is rarely quite circular in shape.

(*b*) *Anisocoria.*—Inequality of the pupils may be present in healthy persons and in those whose eyes refract unequally. For the special pathological relations of unequal pupils see paragraphs (*f*), (1) and (*g*), (1) following.

(*c*) *Hippus.*—A rapid contraction and dilatation of the pupil on sudden exposure to light is normal in some healthy individuals. It is also seen in the early stage of acute meningitis, in disseminated sclerosis, in hysteria, and has been claimed to be somewhat characteristic in epileptics. In Cheyne-Stokes' respiration the pupils may be dilated during the period of rapid breathing and contracted in the apnœic interval. A considerable percentage of phthisical patients present rapid, transient, unsymmetrical dilatation of the pupils (Rampoldi, Destree), presumably due to irritation of the upper thoracic ganglion by tuberculous glands. This statement has been partly verified in my experience.

(*d*) *Argyll-Robertson Pupil.*—If the pupil does not respond to light and shade, but does to accommodation, it constitutes the Argyll-Robertson pupil, a condition most frequently present as a symptom of locomotor ataxia, and due to sclerotic changes in Meynert's fibres, or in that portion of the third nerve nucleus which presides over the light reflex, the portion which regulates the accommodative contraction not being involved. The pupil in this condition is usually contracted (spinal miosis), because the lesions in the cervical cord interfere with the dilating mechanism. Late in the disease the accommodation reflex also may be lost. A similar pupillary condition is seen as a symptom of intracranial syphilis and progressive paresis of the insane.

(*e*) *Accommodative Iridoplegia with Preserved Light Reflex.*—This condition is the opposite of the Argyll-Robertson pupil. It is of infrequent occurrence and may denote a lesion affecting a portion of the oculo-motor nucleus in which lie the cells causing accommodative reaction. It may also, but not always, exist with the cycloplegia (paralysis of the ciliary muscle) which occurs after diphtheria.

(*f*) *Dilated Pupils.*—Dilatation of the pupils (mydriasis) may be due to irritation of the dilating mechanism overcoming the contractors, or paralysis of the contractors allowing unopposed dilatation.

(1) Unilateral Dilatation.—One pupil may be dilated as the result of disease of the optic nerve, as in optic atrophy; or lessened transparency of the media, as in cataract and glaucoma. If the pupil of the diseased eye does not react directly to light, but does react when

the light is thrown into the sound eye (consensual or indirect reaction), it shows that the disease is in the optic nerve or tract and that the oculo-motor nerve of the diseased side, its nucleus, and nuclear connections with the corpora quadrigemina, and through the latter with the opposite optic tract, are not involved. Unilateral dilatation may be due to irritation of the cervical sympathetic by a tumour of the neck, by aneurism of the aorta, or aneurism of the innominate (pupil of the right eye). One pupil is dilated in paralysis of one third nerve (*q. v.*).

Unilateral dilatation, unless due to the local use of mydriatics, requires a thorough examination before excluding serious organic disease.

(2) Bilateral Dilatation.—Double atrophy, amaurosis or blindness from any cause, will obviously give rise to dilatation and failure of the light reflex. Mydriasis may result from cerebral anæmia and poorly filled blood vessels, largely because the centres lack the stimulus of the blood-supply, as in anæmia, syncope, shock, nausea, and aortic regurgitation. The pupils are also dilated in neurasthenia, hysteria, and strong emotion; severe dyspnœa and asphyxia; cerebral hemorrhage, thrombosis, tumour and abscess; the later stages of meningitis and diphtheritic paralysis, from lesions of the oculo-motor nerve or nucleus; and in melancholia, trance, and the coma following an epileptic convulsion.

The drugs which have a mydriatic action, local or constitutional, are aconite, alcohol, atropine, chloral, chloroform, cocaine, conium, duboisine, hyoscyamus, nitrous oxide, stramonium, strychnine, and tobacco.

When both pupils are dilated the eye has a peculiarly brilliant expression, which may be particularly noticeable in mania, in fevers attended with active delirium, exophthalmic goitre, and poisoning with belladonna.

(*g*) *Contracted Pupils.*—Contraction of the pupils (miosis) may be due to irritation of the oculo-motor system or to paralysis of the dilators. Congestion of the vessels of the iris by causing an increase in bulk of the latter will produce the same effect.

(1) Unilateral Contraction.—Small size of one pupil may be congenital, but, like aniscoria (unequal pupils) in general, should be looked upon as a likely indication of more or less serious trouble.

The pressure of an aortic aneurism, if sufficiently great to paralyze the sympathetic (dilator) fibres in the upper thorax, may explain unilateral miosis. The same symptom may be caused by locomotor ataxia, general paresis of the insane and other lesions which may at times be largely unilateral, affecting the cervical cord. Unilateral cerebral lesions so placed, and of such a character as to irritate the oculo-motor centre, may cause unsymmetrical miosis.

(2) Bilateral Contraction.—A certain degree of miosis and sluggish contraction of the pupil is normal in old age, and the pupils are contracted during sleep in a healthy individual.

Congestion of the iris, such as occurs in the injected eye of typhus fever, causes miosis; so also with mitral insufficiency and other conditions producing venous congestion. The pupils are contracted in retinitis and when the condition of photophobia exists.

Bilateral disease of the cervical cord and the cervical spine, by paralyzing the sympathetic dilator fibres, may allow miosis. Symmetrical miosis may thus be significant of locomotor ataxia in particular, disseminated sclerosis, general paresis, spinal meningitis, tumour or hemorrhage of the cord, bulbar paralysis, hemorrhage into the pons, and tuberculous disease of the cervical vertebræ. Irritating lesions of the brain, by stimulating the third nerve centre, will cause miosis, notably cerebral meningitis, cerebral or dural hemorrhage, and tumour or abscess of the brain. It is present in the early stage of sunstroke, and is also a symptom of the uræmic state.

Certain drugs have a very characteristic action in contracting the pupil, particularly opium and its alkaloids. Others are eserine, pilocarpine, and chloral during its primary action.

(*h*) *Absent Skin Reflex.*—If the pupil does not dilate on pinching or pricking the skin of the neck, or using the faradic brush in the same region, it may be due to the atrophy of the iris in a glaucomatous eye or to posterior synechiæ following iritis. It is not infrequent in general paresis.

IV. THE EYEBALL

The symptoms which may be observed in examining the eyeball relate to pain in and around the eye, its protrusion or recession, its position and its degree of mobility.

I. **Pain.**—Pain in and around the eye is most frequently due to trigeminal neuralgia or migraine. With reference to prompt treatment, the pain of acute glaucoma should never be mistaken for either of these. The presence of dimmed vision, a reddened eye, hazy cornea, dilated pupil, and greatly increased tension, perhaps with nausea and vomiting, will declare the case to be one of glaucoma. Inflammatory diseases of the iris or conjunctiva and foreign bodies may explain a painful eye.

Photophobia is a form of eye pain excited by exposure to light. It is present to varying degrees in the conjuctivitis of measles, epidemic influenza, the early stage of pertussis and severe coryza, or the formation of the vesicles of variola and varicella upon the cornea. Ocular or retinal hyperæsthesia is a form of photophobia not dependent upon local inflammatory disease of the eye, and is usually, although not always, due to functional disturbance of the optic tract and nerve or the cortical centres for vision. It is a frequent symptom in neurasthenia, hysteria, migraine, and at times the hypnotic state, and is not unusual in the acute indigestions of children. It may be present in cinchonism and in the early stages of typhus fever and meningitis. It has occurred in albuminuric retinitis and aortic insufficiency (Osler).

II. **Protrusion of the Eyeball.**—*Exophthalmos* or protrusion of the eyeball, when bilateral, has as its most common explanation exophthalmic goitre. The eyes may protrude in spasmodic asthma or other conditions attended by severe dyspnœa. Thrombosis of the superior longitudinal sinus may be attended with some exophthalmos. A patient now under observation has a marked exophthalmos, apparently as a result of enormous cardiac hypertrophy due to aortic and mitral insufficiency. There may be a moderate proptosis in paralysis of the ocular muscles. It should be remembered that undue exposure of the sclerotic will give an impression that the eyes are protuberant.

Swellings or tumours may displace the eyeballs forward, usually on one side, perhaps on both sides, as in great enlargement of the lachrymal gland and aneurism, exostosis, or cancer of the orbit, or tumours of the upper maxilla. One or both eyes may be pushed downward and forward by subperiosteal hemorrhages into the upper portion of the orbit, which may be present as one of the lesions of scurvy.

III. **Recession of the Eyeball.**—*Enophthalmos*, or sinking of the eyeballs into the orbit, if bilateral may be due to absorption of the cushion fat of the orbital cavity such as takes place in phthisis pulmonalis, marasmus, diabetes, typhoid and other continued fevers, the cancerous cachexia, and wasting diseases in general. It may also be present in consequence of a rapid and exhausting drain upon the blood, as in cholera, severe diarrhœa and large hemorrhages, or a deficient orbital blood supply in collapse.

Unilateral enophthalmos may be indicative of a lesion of the cervical sympathetic or the intracranial sympathetic ganglia and plexuses interfering with nutrition and causing atrophy of the orbital connective tissues or paralysis of Müller's orbital muscle. In high degrees of hypermetropia, a condition in which the antero-posterior diameter of the eye is abnormally short, the eyes may have a sunken appearance.

IV. **Position of the Eyeball.**—Normally during sleep the eyes are *turned upward*, so that the cornea is well protected. The same position may obtain during hysterical coma or convulsions, and in an epileptic seizure. In ptosis (paralysis of the levator palpebræ) and in chronic hydrocephalus, where the bulging forehead prevents the proper raising of the upper lid, the eyes *look downward* and the head must be tipped back in order to see an object on or above a horizontal plane. If both eyes are *turned toward the same side* it constitutes conjugate lateral deviation (*q. v.*).

V. **Disturbances of Ocular Mobility.**—The functions of the nerves and muscles of the eye are sufficiently obvious from a study of the diagram (Fig. 50), lack of space forbidding a detailed description. A special word of explanation is here given with reference to the normal conjugate lateral movements of the eyes—i. e., the turning of both eyes to one side in order to see an object placed laterally. In order to accomplish this, it is obligatory that the internal rectus of one eye and the external rectus of the other should act consentaneously. The co-ordinate action of the two, although supplied by separate nerves on opposite sides, is accomplished by the communicating fibres (posterior longitudinal bundle) which run from the nucleus of the sixth nerve on one side to the nucleus of the third nerve on the other side. An impulse travelling from the left cortex is received by the sixth nucleus on the right side, which being excited immediately forwards the impulse, not only along its own nerve to the right external rectus, but also along the communicating fibres to the nucleus of the third nerve on the left side, and it, in turn, transmits it to the left internal rectus. The right external rectus and the left internal rectus simultaneously contract, thus turning both eyes to the right.

Symptoms Indicative of Disturbances of the Ocular Muscles. (1) *Ptosis.*—Drooping of the upper eyelid with an inability to raise it (ptosis) is due to paralysis of the levator palpebræ. As the levator palpebræ is innervated by the second portion of the oculo-motor nucleus, ptosis is dependent upon some interference with the function of the third nerve, its nucleus, or its cortical centre. It may exist alone, but is ordinarily combined with paralysis of the third nerve. The pathological possibilities of this symptom are as follows:

If bilateral, in a woman, and associated with spasm of the orbicularis upon attempting to raise the eyes, and presenting other symptoms of the causative affection, it is probably of hysterical origin. If existing from birth, and the patient is unable to roll the eyes upward, it is due to congenital defect or absence of the levator muscle. Transient double ptosis, slight or marked, may occur in the morning upon waking, in anæmic, neurotic, or overworked women; and as a result of the administration of conium or gelsemium. Double ptosis may be caused by idiopathic muscular atrophy if the facial muscles and the levator palpebræ are affected. Drooping of one lid and contracted pupil, with heat, redness, and œdema of the skin on the same side constitutes pseudo-ptosis, and is caused by a paralyzing lesion of the sympathetic nerve. It has also been attributed to disease of the corpus striatum. The ptosis in this case is referable to the paralysis of the muscular fibres of Müller which exist in the connective tissue of the orbit and, when active, assist in the retraction of the lid. Very seldom a unilateral ptosis, following a slight apoplectic attack, is referable to a cortical lesion, without involvement of the other branches of the third nerve. In severe neuralgia of the fifth nerve, and in tetanus, ptosis may exist, possibly because of transferred irritation from the fifth nerve acting as an inhibitory agent upon the oculo-motor nucleus.

Aside from the conditions which have just been recapitulated, ptosis is usually associated with paralysis of other ocular muscles due to definite lesions or functional affections of the third nerve—i. e., it is simply one of several symptoms indicating oculo-motor paralysis (*q. v.*).

(2) *Strabismus.*—Squint or strabismus is an inability to bring the visual axis of both eyes to bear at the same instant upon one point, the visual axis of one eye constantly deviating, as a rule horizontally, sometimes vertically, from the object inspected.

If the squinting eye turns toward the temple, it is a divergent or external strabismus; if toward the nose, it is a convergent or internal strabismus. Vertical squint may be upward or downward. By asking the patient to look at the tip of the examiner's finger, it is usually easy to decide as to which eye squints (the sound eye fixing itself upon the finger), and whether the squint is convergent, divergent, upward, or downward. If the strabismus be slight and uncertainty arises, the finger should be held directly in front of the face, at about 18 inches distance, and the patient asked to gaze at it. The apparently normal eye should then be covered by a card. The uncovered eye, if it be the squinting one, will immediately alter its position so as to fix itself upon the finger, thus demonstrating that it had not before been properly directed.

Strabismus, with reference to its origin, may be of two kinds, concomitant and paralytic. The latter form is that which is of importance in internal medicine.

In *concomitant* strabismus the squinting eye will retain its mobility, moving readily in every direction concomitantly with its companion, the degree of strabismus remaining substantially the same. The principal causes of concomitant strabismus, which need but a simple mention, are overaction of certain muscles consequent upon refractive errors, and imperfect vision of an eye with resulting disease of the muscles.

In *paralytic* strabismus the squinting eye loses, to a greater or less degree, its power of movement—i. e., its range of motion is restricted as compared with the sound eye. It is due to a loss of power in one or more of the external ocular muscles. If, for instance, the internal rectus of the left eye is paralyzed, and the patient attempts to keep the eyes fixed upon the finger as it is passed slowly from his left to his right, it will be seen, as the finger passes the middle line, that the left eye lags behind the right and a divergent squint develops, becoming more marked the further the finger moves to the right. At the same time double vision, or diplopia, becomes manifest to the patient. Furthermore, in the same instance, if while the patient is looking at the finger held to his right, the sound right eye is screened by a card and an attempt is made to fix the object with the left eye, the right eye behind the card will be seen to deviate still further to the right. This is termed the secondary deviation, and is dependent upon the physiological law that when two muscles are acting toward the same end and one of them lacks power, if an increased impulse is sent to the weaker, the stronger one shares the additional stimulus and contracts more vigorously. As paralytic squint is simply a symptom of paralysis or weakness of the ocular muscles, its diagnostic indications are considered elsewhere (Index—Paralysis, Causes of Ocular).

Closely connected with and dependent upon paralysis of the ocular muscles and strabismus is the subjective symptom—

(3) *Diplopia.*—Double vision depends upon the fact that unless the visual axes are correctly adjusted, each to the other, the images of the objects do not fall upon identical points in each retina. Under normal conditions each image falls upon the macula lutea of each retina, combining to form a single image in the sensorium.

If the proper relation of the visual axes is altered by a wrong position of an eye, due to defective action of the ocular muscles, and the images are received upon dissimilar retinal parts, the images are perceived by the mind as of separate origin—i. e., double. The image perceived by the sound eye is the true image, that perceived by the deviated eye is the false image. It should be remembered that diplopia may be present, but not manifest, unless the eyes are turned in such a direction as to require action by the paralyzed muscle or muscles. Thus, in the case already described (paralysis of the left internal rectus), as illustrating paralytic squint, diplopia may not be present if the eyes are turned to the left, as the palsied muscle is not called upon to contract; but if the eyes are required to turn to the right, the deficient activity of the muscle causes strabismus, and consequent diplopia.

Aside from the double vision which is produced by the action of alcohol, belladonna, conium, gelsemium, and spigelia upon the ocular nerves, diplopia, like strabismus, is a symptom of paralysis of the ocular muscles, and its diagnostic significance will be discussed in connection with the latter. Any lesion of the brain, cord, or basal meninges (inflammation, hemorrhage, tumour, softening, sclerosis) which causes paralysis of one or more of the external ocular muscles may be attended with diplopia.

Double sight with one eye (uniocular diplopia) may occur in cataract and astigmatism, due to irregular refraction, and in brain tumour as a psychical aberration.

(4) *Erroneous Projection and Ocular Vertigo.*—Our judgment of the relation of external objects to our own body is largely based upon the position of the eyeballs, and a knowledge of the latter comes through the degree of innervation required by its muscles. If the eye looks forward, any object seen by it is recognised as being directly in front of us. If the eye is turned to the left, an object seen is recognised as lying to our left side. The relative position of the two objects with regard to each other, and also with regard to the body, is judged by the effort which requires to be made in turning the eye from one object to another and the resulting range of motion of the eyeball. If a muscle is weak, the movement of the eye does not correspond in extent to the effort made in innervating it, and a mistaken judgment as to the position of the body with reference to surrounding objects will result. As the balance of the body is largely preserved by a perception of its relation to its environment, if the perception is uncertain or incorrect, dizziness (ocular vertigo) will ensue. In order to detect it, the sound eye must be covered or closed, as it is rarely present if both eyes are open, or if the affected eye is shut.

5. *Conjugate Deviation.*—If both eyes are turned strongly and persistently toward one side, either right or left, the condition is termed conjugate deviation. This condition may be due either to spasm or paralysis of the internal rectus of one side and the external rectus of the other side, and the eyes may be turned toward the lesion or away from it, depending upon its location and whether it is irritative (causing spasm) or destructive (causing paralysis). This symptom depends upon the normally associated action of the internal rectus of one side and the external rectus of the other in effecting horizontal co-ordinated motion of both eyes to the right or left, as explained upon page 208. Referring to Fig. 50, the lesions which cause this symptom are as follows:

If there is conjugate deviation to the *right*, it indicates:

(*A*) An *irritating* lesion, as at *B*, involving the left cortical centre, or the fibres leading from it through the corona radiata or internal capsule; or, as at *C*, affecting the fibres from the cortex in the right side of the pons after decussation, whereby the nucleus of the right sixth nerve and by it the nucleus of the left third nerve are both stimulated and the right external and left internal recti are made to contract, thus turning the eye to the right.

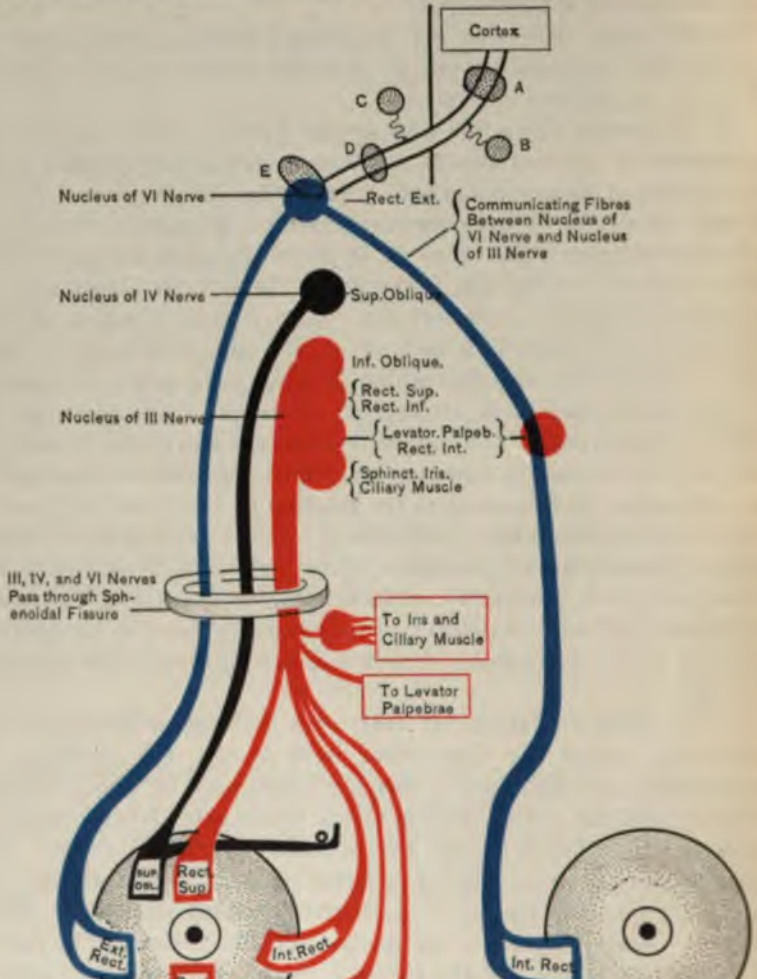


Fig. 50.—Diagram showing the ocular muscles and their nerve supply.

(*B*) A *destructive* lesion of the corresponding right cortical centre or its efferent fibres, or of the same fibres in the left side of the pons, causing paralysis of the left externus and right internus, allowing unopposed action of the right externus and left internus, turning the eyes also to the right.

If there is conjugate deviation to the *left*, it indicates by a similar mechanism:

(*A*) An *irritating* lesion of the right cortex or cortical fibres, or the same fibres in the left side of the pons, or

(*B*) A *destructive* lesion as at *A* (left cortical or cerebral), or at *D* (right side of pons).

The lesions producing conjugate deviation are, in particular, cerebral hemorrhage, tumour, or meningitis. If the lesion is a left-side hemorrhage at *A* and causes right hemiplegia, the eyes look toward the lesion; but if the lesion is or becomes irritant and spasms or convulsions occur, the eyes turn away from the lesion. On the other hand, if right hemiplegia is present as the result of a destructive tumour in the pons at *D*, the eyes look away from the lesion; but if it is or becomes irritant, and spasms and convulsions take place, the eyes look toward the lesion.

The determination as to the nature of the lesion must be made by the associated symptoms of brain tumour, meningitis, apoplexy, sclerosis, or abscess.

6. *Nystagmus.*—A rapid involuntary oscillation or slow movement of both eyeballs, usually from side to side (lateral nystagmus), sometimes vertical or rotatory. It is a clonic bilateral spasm of the ocular muscles, due to some irritation, either functional or organic, affecting the ocular muscle centres. If very slight, it may be intensified by directing the patient to look steadily at an object held well to one side or the other; or if the nystagmus be vertical or rotatory, by directing the eye upward or downward, or circumducting the eye by moving the object.

Rarely nystagmus is congenital from the irritation of hereditarily defective vision, or from cataract or refractive errors. It is present in many cases of blindness or defective sight caused by optic atrophy, amaurosis, and corneal opacities. It may be found in albinos. It is frequently seen in epileptic or other convulsions, and may form a part of the symptomatology of neurasthenia, hysteria, chorea, and some cases of insanity. There is a "miner's nystagmus," a manifestation of irritable weakness of certain of the ocular muscles arising from the strain put upon them by looking obliquely upward while lying upon the side and using the pick in a narrow vein of coal.

The most important diagnostic associations of nystagmus, however, are with Friedreich's ataxia, disseminated (insular) sclerosis, and brain tumour, especially growths involving the cerebellum, pons, or crus. Acute basal meningitis is apt to present it. Occasionally it is seen in the terminal stage of locomotor ataxia and in chronic hydrocephalus. Its cause, in a given case, must be determined by the associated symptoms.

7. *Irregular or Spasmodic Movements of the Eyes.*—Non-rhythmical, irregular movements of the eyes may occur in connection with special ocular paralyses or insufficiencies and errors of refraction. Ménière's disease, when due to lesions of the labyrinth, is sometimes accompanied by forced movements of the eyes. Chronic hydrocephalus and meningitis (basal irritation) may give rise to irregular spasmodic movements of the eyeballs. In hysteria the internal rectus and levator palpebræ may be tonically contracted, the eyes being opened, convergently squinted and uprolled. Divergent squint is said not to exist in hysteria (GOWERS).

8. *Loss of Power of Accommodation.*—Paralysis of the ciliary muscle (cycloplegia) is responsible for loss of the power of accommodation. In this condition distant objects can be clearly seen but near objects are indistinct. It must not be confused with reflex iridoplegia (loss of the pupillary light reflex) or accommodative iridoplegia (loss of the pupillary contraction for near vision). The first and last both depend upon the integrity of the same portion of the oculo-motor nucleus. Cycloplegia is one of the symptoms of complete third-nerve paralysis, is a common symptom in diphtheritic paralysis, and is also seen in multiple sclerosis.

(*c*) **Diagnosis and Causes of Ocular Paralysis.**—Paralysis of one or more ocular muscles is termed ophthalmoplegia.

Ophthalmoplegia *interna* concerns the internal muscles, viz., the ciliary muscle, and the circular fibres of the iris; *externa*, all the other (or external) ocular muscles; *total*, both external and internal; *partial*, a limited number of ocular muscles. There may be much variation in the number of muscles involved as well as differences in the completeness of the paralysis.

In the majority of cases the external eye muscles can be adequately tested by asking the patient to follow the finger moved in different directions, and the intraocular muscles by examining for the light reflex of the pupil and asking the examined to read small print. If a more careful investigation is needed, as with the slighter degrees of paralysis and the ocular asthenopias, the patient should be referred to a competent ophthalmologist.

1. *Paralysis of the Third Nerve.*—If there is ptosis, slight exophthalmos, external strabismus, diplopia, and a dilated pupil which reacts neither to light nor to accommodation, and if when an object is moved in various directions the eye fails to follow it in an upward, inward, or downward direction, but will move outward and a little downward by means of the external rectus and superior oblique, there

is complete oculo-motor paralysis. In many cases some of the muscles escape, the levator and superior rectus or the iris and ciliary muscle being alone affected, or ptosis existing with a dilated pupil.

The signs indicating paralysis of the various single muscles innervated by the third nerve, excepting the ciliary muscle and the iris, are:

For the rectus superior.—The affected eye lags behind its sound fellow in following the object of regard above the horizontal line, the divergence increasing the higher the object is raised, and there is at the same time slight divergent strabismus from the action of the inferior oblique if the latter is unaffected.

For the rectus internus.—If the moving object is brought horizontally across toward the sound eye, the diseased eye will fail to follow it beyond the middle line, or will do so with a wavering, irregular motion due to the action of the superior and inferior recti, if the latter are healthy.

For the inferior rectus.—If the point of fixation is carried below the horizontal line, the affected eye fails to follow it, and there may be slight convergent strabismus from the action of the superior oblique.

For the inferior oblique.—Paralysis of this muscle alone is extremely infrequent and is difficult to detect. On looking at an object above the horizontal line there is diplopia, with slight deviation of the eye downward and inward.

The most frequent causes of oculo-motor paralysis are cold and syphilis, the former producing a rheumatic peripheral neuritis; the latter giving rise to a basal meningitis or a gummatous growth involving the roots of the nerve.

Partial paralysis may be due to a cortical lesion affecting the inferior parietal lobule. By producing anæmia or hyperæmia of the nucleus, undue exposure to light, migraine and the excessive use of alcohol, morphine and tobacco may cause a temporary paralysis. There is a rare form of complete oculo-motor paralysis, affecting women, recurring at intervals of one or several months, and coming on suddenly with headache and vomiting or a well-marked migraine.

As organic disease of the third-nerve nucleus is usually associated with disease of the fourth and sixth nuclei an oculo-motor paralysis, without loss of power of the external rectus and superior oblique, is, as a rule, functional.

While the nerve is penetrating the crus it may be involved by tumour or other lesion of the crus, in which case there will be partial or complete third-nerve paralysis conjoined with hemiplegia on the opposite side of the body, a combination characteristic of unilateral disease of the crus. If in addition there are unilateral paralysis and atrophy of the tongue, the lesion is localized in the inner and inferior aspect of the crus involving the cerebral fibres passing to the nucleus of the hypoglossal or motor nerve of the tongue.

Ptosis occurring on one side, disappearing and then appearing on the other side (alternating), is usually due to variable syphilitic lesions in the neighbourhood of the nucleus.

At and after its emergence from the pons (Fig. 45) on its way to the sphenoidal fissure, the third nerve may be involved by basilar meningitis, frequently of syphilitic origin, or by gummatous deposits or tumours.

The lesions of locomotor ataxia which cause ptosis may be in the same locality. If due to disease at the base, the oculo-motor paralysis is usually bilateral and apt to be accompanied by interference with the function of the other cranial nerves. Third-nerve paralysis may also be one of the symptoms of primary muscular atrophy and upper bulbar palsy.

Oculo-motor paralysis may be indicative of pressure in the cavernous sinus (Fig. 51), or inflammation at the sphenoidal fissure (Fig. 52), or injury (fracture) in either locality.

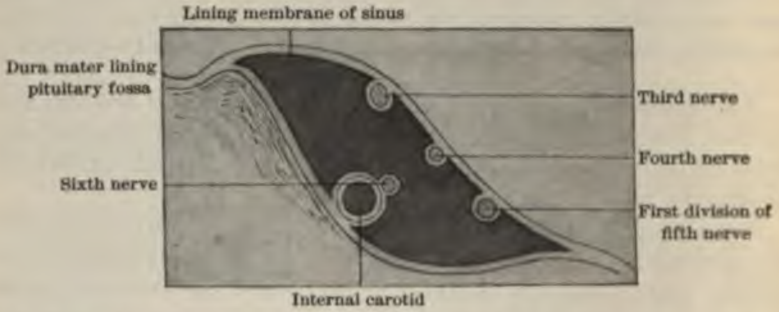


FIG. 51.—Plan showing the relative position of the structures in the right cavernous sinus, viewed from behind. Redrawn from Gray.

If loss of accommodation, loss of light reflex, and external squint succeed an attack of diphtheria it is usually indicative of a neuritis of the third nerve. Neuritis of this nerve may also occur in the course of locomotor ataxia.

Finally, tumours of the orbit may be responsible for this form of ocular paralysis.

2. *Fourth-nerve Paralysis.*—The fourth nerve supplies the superior oblique, and paralysis of this muscle is somewhat difficult of detection. If paralyzed, when the fixing object is moved downward below the horizontal line the diseased eye fails to follow it, there is slight convergent strabismus, and the patient has double vision while looking down.

The diplopia is an extremely annoying symptom, as the eye is so constantly employed in writing, reading, and walking, all of these occupations requiring down-turning of the eye. The head is apt to be carried forward and toward the sound side.

Isolated paralysis of this nerve is very infrequent. When present it may be indicative of pressure upon its trunk as it winds around the outer surface of the crus to reach the antero-lateral edge of the pons (Fig. 45). The pressure may be due to the exudation of basilar meningitis, tumour or aneurism at the termination of the basilar artery bearing against the crus.

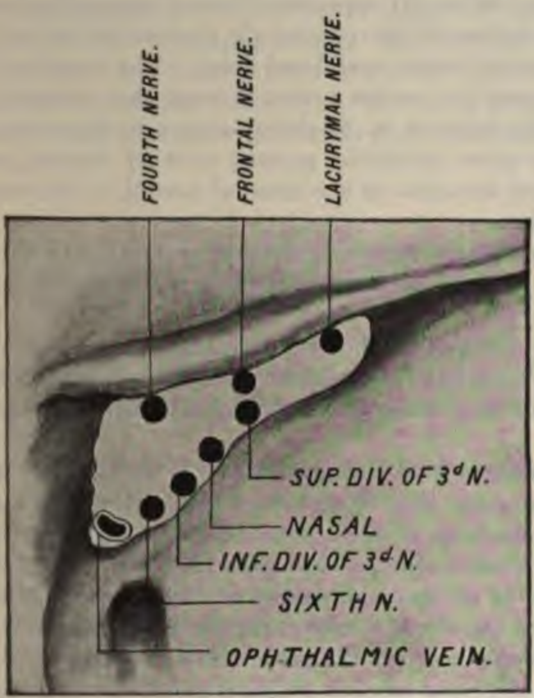


FIG. 52.—Showing relation of structures passing through sphenoidal fissure. Redrawn from Gray

If the symptoms of cerebellar disease coexist with fourth-nerve palsy, it is indicative of a lesion of the anterior part of the cerebellar hemisphere on the same side (STARR). There may be paralysis of this nerve, presumably due to a peripheral neuritis as the result of epidemic influenza, rheumatism (cold), gout, and diabetes, as well as alcoholism and lead poisoning. It may be a transient symptom of neurasthenia.

3. *Sixth, involving Associated Action of the Third Nerve.*—A lesion of the nucleus of the sixth nerve, as at *E*, Fig. 50, may cause conjugate deviation of the paralytic form, the mechanism of which has been previously explained.

4. *Third, Fourth, and Sixth Nerves.*—Total ophthalmoplegia may be indicative of disease in the cavernous sinus of the sphenoidal fissure (Figs. 50 to 52), especially if there is anæsthesia of the peripheral distribution of the ophthalmic division of the trifacial nerve (skin of forehead, upper eyelid and nose). The branches of this division accompany the ocular nerves through the sphenoidal foramen, and it may be involved in morbid processes in these localities—i. e., syphilitic or other periostitis, gumma or other tumour, aneurism or arterio-venous aneurism of the internal carotid, or thrombosis of the cavernous sinus.

Ophthalmoplegia, external, internal or total, may be a symptom of acute or chronic polioencephalitis superior (upper bulbar palsy, nuclear palsy).

When paralysis of all the ocular muscles is associated with facial paralysis without hemiplegia, it is probably due to a basal lesion, as the facial, like the ocular nerves, is liable to involvement by disease at the base.

(*d*) **Asthenopia and Insufficiencies of the Ocular Muscles.**—If complaint is made that close use of the eye, such as that involved in writing, reading, or sewing, causes them to feel strained, hot, and uncomfortable, and perhaps gives rise to headache in the forehead, vertex, or occiput, the source of the trouble may be found in a weakness of the ocular muscles, particularly the recti.

If this weakness exists the patient can not keep the visual axes of the eyes in proper relation without an effort, either conscious or unconscious.

If normal strength of the ocular muscles is present it is called orthophoria. If some of the muscles are weak it is termed heterophoria.

The varieties of heterophoria are: *esophoria,* weakness of the externi, causing a tendency to convergence of the visual axes; *exophoria*, a tendency to divergence of the visual axes from weakness of the interni; *hyperphoria*, a tendency of the visual axis of an eye to deviate upward.

None of these deviations are sufficiently obvious to constitute strabismus. As all errors of refraction should be corrected before making the special tests, it is always advisable to have the examination made by a competent ophthalmologist.

While the immense importance of muscular asthenopia in causing various forms of nervous disease has been vigorously urged by certain writers, the consensus of opinion among the masters of ophthalmology and neurology appears to be that its etiological influence has been greatly overrated. According to the more conservative view it is not a factor in causing chorea, hysteria, or epilepsy; but in neuropathic individuals it may intensify or render more frequent attacks of migraine, trigeminal neuralgia, occipital and cervical headaches, vertigo, and perhaps choreiform movements of the upper facial muscles. It is probable that correction of refractive errors will relieve the troublesome symptoms more effectually than tenotomies.

V. VISION

The disorders of vision which are of diagnostic importance are hemianopia, alterations in the colour fields, amblyopia, and amaurosis.

I. **Minor Disorders.**—If objects seen have a yellow tint, it may be due to jaundice or the administration of santonin. In exhausted neuropathic women or children, overuse of the eyes may cause everything to turn red. The small, beaded, semitransparent threads or dots (muscæ volitantes), seen in looking at some clear expanse of light, are of small diagnostic importance, but they appear to be most abundant in cerebral anæmia or hyperæmia, hysteria, functional disorders of the stomach and liver, and cardiac hypertrophy. Flashes or small luminous points of light before the eyes occur most commonly in acute indigestion, and may constitute the aura of epilepsy. Migraine may be preceded by scotomata resembling a cloud, the edges of which are brilliantly lighted or coloured, the "flittering scotoma" of German writers. It is said that such appearances occasionally precede an attack of intracranial hemorrhage or thrombosis, and may be present in hypochondriasis, insanity, delirium tremens, typhus fever, and meningitis.

II. **Alterations in the Field of Vision.**—The alterations in the size and shape of the visual fields which are of more or less value in diagnosis are: (*a*) hemianopia, (*b*) contraction of the visual fields for light, and (*c*) contraction of the colour fields. In connection with hemianopia, blindness due to disease of the optic nerve or its central connections will be considered.

(*a*) **Hemianopia.**—The condition variously termed hemiopia, hemianopia, or hemianopsia is that in which there is blindness of one half of the field of vision. It is due to functional or organic disease affecting the optic nerve or its central connections. The optic-nerve mechanism is shown in Fig. 53.

Hemianopia, or blindness of one half of the visual field, is of different varieties, and, together with the qualifying terms employed, refers to the field of vision from the point of view of the patient—not to the retina. Thus, right temporal hemianopia means that the outer or temporal half of the field of vision of the right eye is blind, so that if the eye is fixed upon a point directly in front objects to the right of the point of fixation will not be perceived. But it must be remembered that blindness of the temporal half of the field of vision is due to loss of function of the inner or nasal half of the retina, because of the crossing of the light rays in the media, and so with other varieties.

If the hemianopia affects both visual fields, it is *bilateral;* if corresponding halves (both right or both left), it is *homonymous* or lateral. If both temporal or both inner fields are implicated, it is *heteronymous*. Furthermore, the inner fields are termed *nasal;* the outer, *temporal*. Very rarely the upper or lower halves of the visual field are effaced, and it is then called *superior* or *inferior* (sometimes altitudinal) hemianopia. Hemianopia may be partial, only a portion of the half field being blank. The unaffected half may retain its normal dimensions or, as in some cases, be reduced in size.

To determine the presence of hemianopia each eye must be tested separately, the resting eye having been covered by a card. The patient, being placed with his back to the light, is told to fix the uncovered eye upon that part of the physician's face (placed about two feet away) which is most nearly on the horizontal plane of the eye. The finger or a bit of white paper is then brought, first from one side, then from the other, to the median line. If the patient can not see the paper until it has passed nearly or quite to the line of fixation, hemianopia exists. The vertical extent of the field is tested in a similar manner by bringing the object from above downward and below upward. To get an *accurate* outline of the visual and colour fields, it is necessary to employ the perimeter.

Hemianopia in the majority of cases is due to organic disease of the brain, such as hemorrhage, softening, inflammation, or tumour, but may be of functional origin in connection with gout, lithæmia, migraine, and occasionally hysteria. It is of considerable value in localizing certain lesions but the determination of the nature of the causative lesion depends upon the associated symptoms. The diagnostic indications of the various forms of hemianopia are as follows:

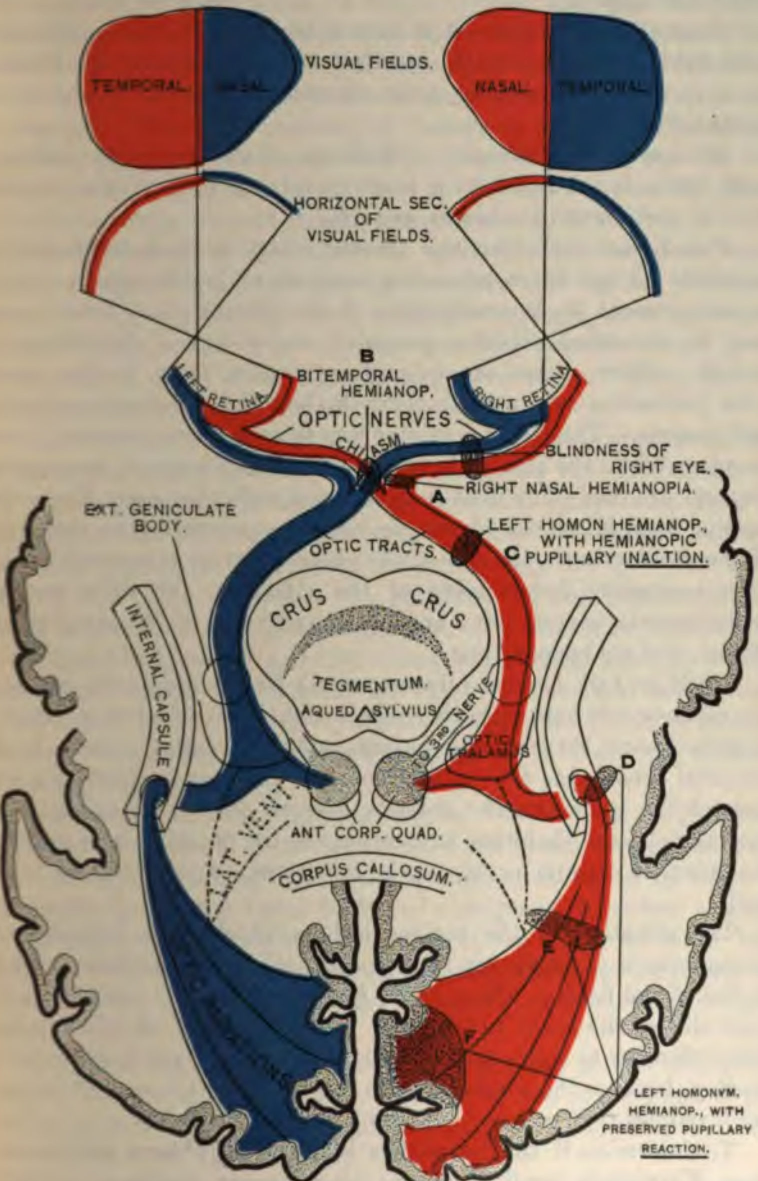


Fig. 53.—Diagram showing optic tracts, visual fields, and lesions causing hemianopia.

Unilateral Nasal Hemianopia.—Blindness of the nasal half of the field, an extremely rare occurrence, indicates a very limited lesion at the outer angle of the chiasm, as at *A* (Fig. 53, to which subsequent references apply).

Binasal Hemianopia.—Also rare is blindness of the nasal half of both fields, requiring for its manifestation two symmetric lesions, as at *A*, on both outer angles of the chiasm, or the outer sides of both optic nerves.

Bitemporal Hemianopia.—Blindness of the temporal halves of both fields is produced by a lesion involving the anterior angle or central portion of the chiasm, as at *B*.

The disease (affecting the chiasm) which is most frequently responsible for the three preceding varieties of hemianopia is tumour or enlargement (as in acromegaly) of the pituitary body, with which may be associated diabetes, proptosis, and a flow of fluid from the nostril. Other causes are tubercles, tumours, cysts, basilar meningitis, periostitis, exostosis, fracture of the body of the sphenoid bone, and gumma. The latter may give rise to evanescent recurring hemianopia. When the chiasm is affected the lesion is apt to be progressive, and gradually involves both crossed and uncrossed fibres, thus causing total blindness of one or both eyes, according to the extent of the destruction. Lesions of the chiasm may be attended by symptoms indicating involvement of the olfactory, the fifth, and the ocular-muscle nerves—viz., anosmia, anæsthesia of conjunctiva and cornea, and ocular paralysis.

Right or Left Lateral (*Homonymous*) *Hemianopia.*—Blindness of the right or left half of both fields is significant of a lesion situated at some point between the chiasm and the cortical centre in the occipital lobe—viz., the optic tract, the pulvinar or posterior gray mass of the thalamus, the anterior corpora quadrigemina, the fibres passing from the thalamus and corpora to the occipital lobe either in the internal capsule or the optic radiations, or the cortical centre itself.

The situation of the various lesions which cause homonymous hemianopia is of course on the opposite side from the defective half of the visual fields. Thus, *C*, *D*, *E*, and *F* represent lesions on the right side of the brain causing left hemianopia. It should be noted with reference to right hemianopia that there is much difficulty in reading, because the words lying to the right of the point of fixation are invisible.

To determine if the lesion lies between the chiasm and the corpora, Wernicke's pupil symptom, "hemianopic pupillary inaction," may be of service if it can be obtained. A beam of light must be thrown upon the blind half of the retina, and the resulting contraction or non-contraction of the pupil noted. If the pupil does not react (pupillary inaction) it shows that the reflex arc, retina to corpora, corpora to third nerve, to iris, is injured, and that the lesion must lie anterior to the corpora in the optic tract. On the other hand, if the pupil does react, the reflex arc is intact, and the lesion lies posterior to the corpora in the internal capsule, optic radiations, or cuneus. Pupillary inaction, if found, is a valuable localizing symptom, but it is obviously hard to determine it because of the difficulty in causing the ray of light to impinge only on the blind half of the retina without stimulating the seeing half.

The diseases which may affect the optic tract are neoplasm, syphilitic meningitis, gummata and tuberculous meningitis. In these cases the crus may be implicated, causing hemiplegia, or, as the result of basal lesions, ocular-muscle paralyses. The absence of any form of aphasia in right hemiplegia is against a central lesion and in favour of an affection of the tract.

As in the majority of cases of hemianopia there is organic disease of the brain, hemiplegia and hemianæsthesia on the same side with the hemianopia are not uncommon associated symptoms; and, in left-side lesions, aphasia. If athetosis coexists with lateral hemianopia it is significant of a lesion involving only the posterior gray mass of the optic thalamus, the pulvinar. If the hemianopia involves a quadrant and not a semicircle, or is otherwise incomplete, and if mind blindness or word blindness coexists, the lesion is usually cortical. Dimness of vision in one eye, and a marked contraction in the visual field of the other, together with mind blindness (seeing but not recognising objects), is a combination indicative of a lesion of the angular gyrus.

Hemianopia in rare instances may be due to hysteria, in which case the conjunctiva is usually anæsthetic and hemianæsthesia is present. But by far the most common alterations of vision in hysteria are contraction of the visual fields and changes in the colour sense.

Altitudinal Hemianopia.—Blindness of the upper or lower half of the visual field may be caused by a lesion affecting the upper or lower portions of the chiasm and is usually associated with optic neuritis.

Unilateral superior or inferior hemianopia may be due to a lesion respectively of the lower or upper portion of the cuneus, and if the lesions happen to be symmetrical and bilateral—a clinical curiosity —the hemianopia will be double.

Total blindness of one eye may be due to a lesion of one optic nerve (Fig. 53), or disease of one occipital lobe, and if in addition there is hemianopia of the opposite eye, it may be due to disease of the decussating fibres in the centre of the chiasm together with the direct fibres of one lateral angle. Total blindness of both eyes will be caused by disease affecting the entire chiasm, or bilateral lesions of the cuneus. It is presupposed that no disease of the retina or other ocular structures sufficient to account for the loss of sight is present.

(*b*) **Contraction of the Visual Fields for Light.**—In order to determine the presence of this symptom one invokes the aid of the oculist and his perimeter, as it can not be ascertained by any less accurate test. Leaving out of consideration glaucoma, optic atrophy, or other organic disease, by far the most important indications of a nearly uniform contraction of the visual fields relate to the presence of hysteria and traumatic neuroses. As a rule, one field presents a greater contraction than the other. In neurasthenia, while there may not be a true concentric limitation of the visual field, yet the fatigue of continued testing will sometimes develop a decided temporary diminution in its size.

(*c*) **Contraction of the Visual Fields for Colour.**—The colour fields are tested by the perimeter, using bits of coloured paper. Passing from the circumference to the centre of the field, the perception of white objects is found to have the widest extent. A little nearer blue is perceived, then red, then green (Fig. 54).

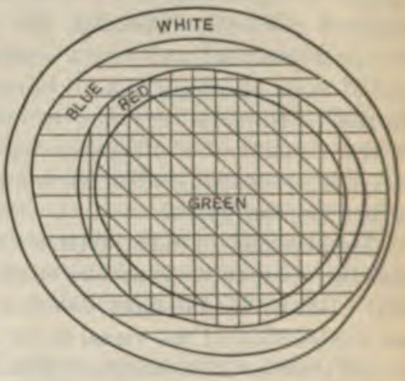


FIG. 54.—Visual colour field of right eye.

Limitation of the colour fields is a common symptom in hysteria and traumatic neuroses. Sometimes a transposition of the normal fields for different colours will occur, one taking the place usually occupied by another. A central blank or scotoma for red or green is a constant symptom of tobacco or other toxic amblyopia, usually due to a retrobulbar neuritis. Like vision in general, the colour perception may be much reduced or abolished by optic neuritis or atrophy.

III. **Amblyopia, Amaurosis.**—Amblyopia is dimness of vision or partial blindness, while amaurosis signifies a total loss of sight, *without appreciable lesions.* The causes producing these forms of partial or total blindness are, as a rule, of a functional character, affecting either the centres or the retina or, if organic, do not produce noticeable alterations in the appearance of the latter.

The loss of vision is usually sudden, bilateral, and, in most cases, temporary. The most frequent causes are uræmia, diabetes, tobacco, and certain drugs, large hemorrhages, migraine, and hysteria. When occurring in uræmia it ordinarily follows coma or convulsions, is sudden and usually lasts but one or two days. It may supervene in a similar manner in diabetes, and in all cases of amblyopia the urine should be examined for sugar. In tobacco amblyopia the onset is slow, the loss of sight is most marked in the centre of the visual field, and on testing the colour field a central blank or scotoma for red and green is found to exist. Quinine and the salicylates may cause sudden amaurosis, and it sometimes occurs in alcoholism. Severe hemorrhages, particularly those from the stomach, may be responsible for a sudden failure of sight. Migraine may exhibit as one of its symptoms a fugitive blindness, perhaps of only one half the visual field (hemianopia). The amaurosis of hysteria is associated with emotional states and other characteristic symptoms, and no matter how complete the apparent loss of sight may be, the pupillary reflex, both direct and consensual, is retained.

Other and less frequent causes are blows upon the eye or the head without visible injury, shock from lightning or the dynamo current, and poisoning from lead. In cases in which strabismus or a high degree of astigmatism with hypermetropia has existed from an early age, one eye may be accidentally discovered to possess very little power of vision.

IV. **Ophthalmoscopic Signs of Extraocular Diseases.**—Valuable diagnostic information may be obtained by an examination of the interior of the eye, but it is an open question whether the general practitioner, who makes an occasional ophthalmoscopic examination, can rely upon his own interpretation of what he sees. As a rule, the examination should be made by a specialist.

The conditions of general diagnostic value, as distinguished from purely local ocular diseases, are: retinal hemorrhage, optic neuritis, optic atrophy, pulsating retinal arteries, embolism or thrombosis of the central artery and tubercles of the choroid.

(*a*) **Retinal Hemorrhage.**—Apoplexy of the retina occurs most commonly in elderly people with degenerated arteries. Cardiac hypertrophy from valvular disease or chronic nephritis may be the causative condition. So also may the gouty diathesis (Hutchinson) and hemorrhagic blood states, as in scurvy, hæmophilia, purpura, and grave anæmias. Retinal extravasations may also be significant of malarial fever, ulcerative endocarditis, pyæmia, suddenly suppressed menstruation, the menopause, and leucæmia, although in the latter a retinitis is more common.

(*b*) **Optic Neuritis.**—Inflammation of the optic nerve, with congestion, hemorrhage, and exudation into its substance, may affect the nerve posterior to the globe, *retrobulbar neuritis;* the nerve and its end, *papillitis;* the retina, *retinitis.* In course of time there may be connective-tissue proliferation and consequent atrophy of the nerve fibres, giving rise to secondary (consecutive) *optic atrophy.*

(1) *Retrobulbar Neuritis.*—In this form of inflammation of the optic nerve the retina itself is but slightly affected. If acute, there is pain in the globes with rapid loss of sight, but in the more chronic forms the visual defects consist of scotomata (blind spots in the visual field) for colour or light. It is indicative of the abuse of alcohol or tobacco (amblyopia), lead poisoning, diabetes, and syphilis, and is sometimes due to rheumatic inflammation.

(2) *Papillitis.*—Inflammation of the papilla is in reality a descending optic neuritis with a very marked swelling of the papilla, the "choked disk" of earlier writers. It is the extreme grade of papillitis to which reference is had in the following paragraphs. Papillitis of a much less intensity is present as a part of retinitis or neuro-retinitis. The vision in many cases remains nearly normal, while in others there may be limitation of the visual fields or scotomata for light and colour.

Papillitis of the extreme grade, and usually affecting both eyes, is of great value as a symptom of brain tumours, occurring as it does in $\frac{4}{5}$ of all cases, without reference to the size of the growth. It does not accompany growths in the medulla (HUGHLINGS JACKSON), is not very common in lesions of the motor cortex, and is most frequent in cerebellar neoplasms. Next in frequency it is symptomatic of tuberculous meningitis (80 per cent of all cases), and it may also be present in the non-tuberculous and suppurative forms of the same disease. It is also found in a considerable proportion of cerebral abscesses.

(3) *Retinitis and Neuro-retinitis.*—As retinal disease is usually secondary to some extraocular cause, it is in the majority of cases a bilateral affection. If the retina is chiefly involved, it is a retinitis; but if the papilla, as usual, presents evidence of disease, it constitutes a neuro-retinitis.

The varieties and diagnostic significance of this condition are as follows:

Hemorrhagic Neuro-retinitis.—In this form the retinal hemorrhages are abundant. It occurs in general atheromatous degeneration of the arteries, in cardiac hypertrophy, in cardiac valvular disease, especially the insufficiencies, and in aneurism. It may also be a symptom of diabetes and chronic nephritis, and sometimes happens in sudden suppression of menstruation or chronic discharges.

Albuminuric Neuro-retinitis.—It occurs in from 15 to 20 per cent of all cases of chronic Bright's disease, most commonly with the interstitial variety. The papillitis may be so marked that it is difficult to determine whether it is of renal or intracranial origin.

Syphilitic Retinitis or Choroido-retinitis.—In inherited syphilis retinitis pigmentosa may occur. As a secondary symptom of acquired syphilis both retina and choroid are more likely to be affected. Purulent choroiditis may be a symptom of pyæmia, ulcerative endocarditis, cerebro-spinal meningitis, and other infective diseases.

Anæmic Neuro-retinitis.—Inflammatory and hemorrhagic alterations in the retina and papilla may be due to large hemorrhages, pernicious anæmia, and severe chloro-anæmia.

Leucæmic Neuro-retinitis.—In 25 to 30 per cent of cases of leucæmia retinitis is present.

Finally, neuro-retinitis may be found in lead poisoning, diabetes, diphtheria and other infectious diseases, the malarial cachexia, chronic hydrocephalus, and, as a unilateral symptom, in disease of the bones of the orbit or erysipelatous inflammation of the orbital tissues. It may exist without an assignable cause.

(*c*) **Optic Atrophy.**—Atrophy of the optic nerve may be primary or secondary.

(1) *Primary Atrophy*, not preceded by papillitis or neuro-retinitis, is in the majority of cases a symptom of spinal-cord disease, especially locomotor ataxia. Less frequently it occurs with disseminated sclerosis, lateral sclerosis, and general paralysis. There is an hereditary form affecting young males. Other causes are large hemorrhages, diabetes, the specific fevers, chronic alcoholism, and lead poisoning.

(2) *Secondary Atrophy.*—Secondary or consecutive atrophy is usually consequent to an optic neuritis, the causes of which have been previously rehearsed. Retrobulbar neuritis, syphilitic choroido-retinitis, and retinitis pigmentosa may also be responsible for consecutive atrophy. In some cases of chronic hydrocephalus a greatly distended third ventricle may press upon the chiasm and produce similar changes.

(*d*) **Pulsation of the Retinal Arteries.**—Visible pulsation may be found due to aortic insufficiency, great cardiac hypertrophy, or vasomotor instability as in exophthalmic goitre and other conditions in which there is abnormal throbbing of the arteries.

(*e*) **Embolism and Thrombosis of the Central Artery.**—Obstruction or thrombosis of the central artery causes a sudden loss of vision, commencing at the circumference of the visual field and extending to the centre. It is frequently indicative of cardiac or vascular disease—viz., ulcerative endocarditis, valvular disease, particularly mitral stenosis, atheroma of the large arteries, aneurism of the aorta, thrombosis of the pulmonary veins, and very rarely it occurs in chorea. In a very small proportion of cases the sudden loss of sight in chronic nephritis is said to be due to this accident.

(*f*) **Tubercles in the Choroid.**—These are significant of tuberculous meningitis or general miliary tuberculosis.

SECTION XVII

THE NOSE

I. **Examination of the Nose and Nasal Chambers.**—Having a good light (Argand, Welsbach, electric) upon the right of the patient, on a level with or a little higher than his mouth, desire him to sit nearly erect on a straight-backed chair in such a manner that he is supported without lolling back. The observer sits facing the patient, knees outside if a man, to the patient's right if a woman. A forehead mirror, 3 to 3½ inches in diameter, with a central opening, should be employed. As a rule, it is better to wear the mirror in front of the left eye, as the otherwise obscuring glare of the light is thus avoided.

Having directed the reflected light upon the nose, note its shape, colour, presence of excoriation, fissure, or eruption. Tilt up the tip of the nose, and inspect the lower portion of the septum as to symmetry and the existence of ulcers or raw surfaces. Closing first one, then the other nostril, desire the patient to breathe rather deeply in order to determine their patency and whether or not the alæ fall in unduly during inspiration.

Then warm and introduce a nasal speculum (bivalve, duckbill, or wire). The first of the intranasal structures to be seen, provided the patient's head is not retracted, is usually the rounded end of the inferior turbinate body, from which this structure may be followed backward for a varying distance. Note if it appears swollen and abnormally large, and if so always test its consistence with a probe in order to determine whether the enlargement is bony, firm, or a soft vascular tumidity. Depress the head slightly so as to see the inferior meatus, where foreign bodies, if present, are usually found; then tip it backward in order to bring the middle turbinate (which begins considerably farther back than the inferior) and middle meatus into sight. The latter is the usual location of polypus and the place where the thick pus of antral, frontal, or ethmoidal disease is found. If the inferior turbinal is so large that it interferes with the examination of the parts behind and above, apply a pledget of cotton soaked in a 5 to 10 per cent solution of cocaine or suprarenal capsule to the part, which will speedily reduce the swelling so far as it is due to vascular engorgement.

Finally, turn the patient's head slightly from side to side in order to examine the septum for abnormal deviations, ridges, spurs, ulcerations, or perforations. It should be borne in mind that the septum is rarely evenly placed between the two nostrils, so that, as a rule, one cavity is larger than the other. Test all projections with the probe to discover their consistence, extent, and whether opposing projections are in firm contact, thus causing erosions or turbinal pressure. The superior turbinate can not be seen.

The examination thus far has been from the front—anterior rhinoscopy. Inspection from the back, or posterior rhinoscopy, requires the use of a small (¼ to ½ inch in diameter) laryngoscopic mirror. The tongue depressor (usually indispensable) should be applied, and the patient instructed by precept and example to breathe quietly through the nose. Then, having warmed the mirror, introduce it, reflecting surface upward and parallel to the tongue depressor, until the soft palate is approached, when it should be turned so that it passes edgewise between the uvula and the left tonsil to a point below and behind the soft palate. Having again turned the mirror so that the reflecting surface looks upward and forward, the posterior nares should be brought into view. The surface to be examined is of such shape and extent that it can not be seen all at once. The mirror must be turned and shifted in a manner which can only be acquired by practice.

The most striking object first seen is the posterior edge of the septum (Fig. 55), sharp and yellowish in colour below, broadening and becoming more distinctly red above. To either side may be seen two bluish-red or gray-red swellings—the posterior extremities of the middle turbinates. Below are the inferior turbinates, of which the upper halves only are usually seen; above are visible the superior turbinates.

The colour, shape, and size of the turbinates and the presence of pus or polypi should be determined. By turning the mirror to one side or the other, keeping it low down with its back resting almost against the tonsil, the openings of the Eustachian tubes may be seen as rounded red prominences with central yellowish depressions. Posterior to these are the fossæ of Rosenmüller. Finally, the mirror should be turned upward in order to observe the vault of the pharynx, the surface of which normally somewhat resembles that of the faucial tonsil. The most important abnormality here is the adenoid or lymphoid growth so commonly found to be the cause of habitual mouth-breathing in children. Digital examination of the upper pharynx is often required to determine the exact size and location of the growth, and is indispensable in children who are too young to be under control. The child's head is held either by an assistant or, better, by the physician, who sits or stands by the patient and embraces and steadies the head with his left arm. The right forefinger, palmar surface up, is then passed in at the angle of the mouth, invaginating the cheek between the teeth of the patient to prevent biting. The finger, having been carried back, hugging the tonsil, is hooked up behind the palate to the upper pharynx, where it explores the pharyngeal vault, septum, turbinates, and Eustachian prominences.

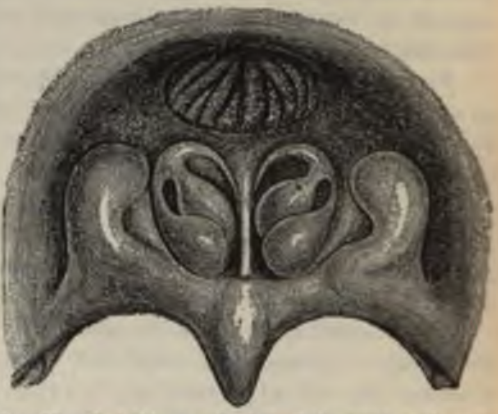


Fig. 55.—Post-rhinoscopic view of the septum, choanæ, Eustachian tube mouths, soft palate, and pharynx vault (after Heymann).

Posterior rhinoscopy is rarely easy, generally difficult, and sometimes impossible, even in the hands of the expert.

II. **Pain in or around the Nose.**—Burning or smarting sensations usually attend coryza or other acute catarrhal inflammations; and if great pain in the nasal chambers is present, it may be a symptom of a syphilitic lesion, glanders, or impacted foreign body. Severe pain just above the root of the nose is due to inflammation of the frontal sinus; in the nose and cheek, of the antrum; referred to the nasal cavities and ear, of the Eustachian tubes. A sensation of dryness is a common complaint in the early and late stages of coryza and a persistent and annoying symptom of atrophic rhinitis.

III. **Shape, Colour, and Ulceration of Nose.**—The nose becomes coarse and broad in cretinism and myxœdema. A depressed, sunken, "saddle" nose may be due to a previous injury, but if there is no history of traumatism, syphilis should be suspected. The nose is broadened or distorted and displaced by growing tumours in the nasal cavities or the adjacent facial bones. A nose which is pinched and insignificant relatively to the lower part of the face may be indicative of nasal obstruction and consequent mouth-breathing.

A chronic redness of the nose due to dilated capillaries, perhaps also with some papules, may be significant of chronic alcoholism, or is an acne rosacea from other causes. A similar redness may be caused by chronic digestive disorders, amenorrhœa, and, exceptionally, by chronic hypertrophic rhinitis. Acute redness of the nose, with pain and swelling, especially of one ala, may be the result of a small pustule or boil, to be seen just within the nares; or may be a beginning erysipelas. In the latter case the redness rapidly extends, and there is a marked elevation of the bodily temperature.

A superficial ulceration of the wing of the nose in a young person, usually painless, beginning as a reddish papule, spreading in various directions, and healing in one portion while breaking down in another, is probably a *tuberculous ulcer*. In an elderly patient a small, hard, scabbing ulcer, somewhat painful, gradually extending, and perhaps with glandular involvement, is an *epithelioma*. In all cases, both children and adults, an ulcer of the margin of the nose or multiple circular ulcers should cause a careful search elsewhere for evidences of *syphilis*.

IV. **Sneezing.**—This is a spasmodic expiration, caused usually by direct, rarely by reflex, irritation of the sensory nerves of the nose, and occurs as an early symptom of coryza, measles, pertussis, asthma, and hay fever. Small doses of the iodides in susceptible individuals, and large doses in many others, will induce it, as well as the inhalation of various irritants like pepper, snuff, powdered ipecac, euphorbium, or veratrum, and solutions of zinc chloride. It has been attributed to gout, and prolonged paroxysms of sneezing have been asserted to be of hysterical origin. Syringing or manipulation of the external auditory canal will sometimes provoke a reflex sneeze.

V. **Acting Nares; Regurgitation of Fluids.**—In some sensitive and neurotic individuals the nostrils dilate with each inspiration, especially under mental excitement. Aside from this, acting nares indicate marked dyspnœa (*q. v.*) of various origin, and among other conditions are very noticeable in emphysema, asthma, pneumonia, obstructive diseases of the larynx, and the broken compensation of cardiac valvular lesions. It is a particularly useful symptom in the form of pneumonia in children which begins with such marked gastro-intestinal symptoms that the underlying pulmonary condition is overlooked. The respiration may not be very rapid, but the dilating nostrils often give the first clew to the real nature of the case.

If upon attempting to swallow fluids a portion escapes through the nostrils, one of three conditions is usually present—cleft palate, diphtheritic paralysis of the soft palate, or bulbar paralysis.

VI. **Nasal Stenosis.**—Difficulty in breathing through the nostrils, the stenosis coming on somewhat rapidly, may be symptomatic of an acute coryza, hay fever, nasal diphtheria, foreign bodies in children (one nostril), or the prodromal stage of variola, typhus fever, or glanders. A more chronic and slow-coming nasal obstruction affecting one or both sides is usually referable to hypertrophic rhinitis, postnasal lymphoid growths, polypi, or a deviated septum. If accompanied by "the snuffles" in infants, hereditary syphilis should be suspected.

VII. **Discharges from the Nose.**—Nasal discharges may be clinically (*a*) non-offensive, watery, mucous, muco-purulent, or purulent; (*b*) offensive; (*c*) bloody or composed of blood alone, according to the nature and seat of the causal condition.

(*a*) A *watery* discharge, in some cases very profuse, marks the beginning of acute coryza, hay fever, the catarrhal form of epidemic influenza, pertussis, measles, iodism, and typhus fever. In the later stages of some of these the discharge grows thick and *muco-purulent.* Watery fluid may flow from one nostril during an attack of trigeminal neuralgia. An occasional watery discharge, with a persistent obstruction of one or both nostrils, may be due to nasal polypi. A recurring flow of *pus* from one nostril, particularly if brought on by lying upon or leaning over toward the side opposite to that which is discharging, is probably an antral abscess, and the probability is increased if bad teeth are present. In children the possibility of a foreign body should not be overlooked. Finally, irritating gases or powders will produce a smart watery flow.

(*b*) *Offensive discharges* may be significant of an impacted foreign body. Coming on in the course of pharyngeal diphtheria and irritating the parts with which it comes in contact, it may indicate nasal infection before membrane can be discovered in the nasal cavities. An extremely offensive continuous discharge from both nostrils, which may be accompanied with greenish-gray crusts, is symptomatic of atrophic rhinitis, or may be due to syphilis, caries or necrosis of the bones, or glanders. It may be a sequel of scarlatina. The patient is usually unconscious of the fetid odour. Cancer or lupus affecting or encroaching upon the nasal chambers may also be responsible for discharges possessing an unpleasant smell.

(*c*) A *discharge of blood* from the nose (epistaxis) may be a result either of local or general causes. It is usually a capillary oozing and upon inspection the bleeding area is seen to be congested, ecchymotic, or superficially ulcerated. A common site of the bleeding spot is upon the cartilaginous septum. Less frequently it arises from the posterior end of the middle turbinated body or, in children with adenoids, from the vault of the pharynx or at times from the accessory sinuses. Careful specular inspection under a good light, clots having been removed by swab or spray, is necessary to determine the exact location of the hemorrhagic point. If epistaxis occurs during sleep the blood may run into the pharynx and, after having been swallowed, may be vomited or, if clinging to the pharynx, be hawked up, thus simulating hæmatemesis or hæmoptysis. Nosebleed from local causes is usually unilateral, but if due to blood changes or general diseases is apt to take place from both sides.

Epistaxis is ordinarily not sufficiently severe to produce constitutional symptoms, but after operations on the nose or in cases of hæmophilia may be so profuse or continue so long as to seriously threaten life, although an actual fatal result is extremely rare.

1. Nasal Causes of Epistaxis.—Aside from traumatic causes, caries or necrosis of the nasal bones, ulceration from a foreign body or other causes, polypus, new growths, varicosities, and chronic nasal catarrh may be responsible for epistaxis. Arteritis and alcoholism may render the vessels liable to rupture.

2. General Causes of Epistaxis.—People of full habit may have a rather frequently recurring nosebleed which appears to be conservative. Delicate children suffer from it, especially at puberty and after exertion under a hot sun. It is a common complaint of mountain climbers at considerable altitudes, and inhalation of very hot or very cold air may induce it. In suppressed menstruation it may be vicarious. The chronic anæmias are frequently attended by epistaxis, and it is a symptom of leucæmia, purpura hæmorrhagica, and scurvy. It is the most ordinary form of hemorrhage in hæmophilia. Less commonly it may be the result of cardiac hypertrophy and valvular disease, enlarged bronchial glands, pleurisy with large effusion, emphysema, and the strain of pertussis.

It is a symptom of diagnostic value in the prodromal stage of typhoid fever, and is an occasional event in other infections, as erysipelas, malarial fever, measles, scarlet fever, dengue, relapsing fever, pyæmia, and acute yellow atrophy of the liver; also in phosphorus poisoning. As an infrequent happening it occurs in ascites, peritonitis, appendicitis, enlarged spleen, large ovarian cystomata, uterine fibroids, chronic nephritis, and cerebral thrombosis. Finally, it may be impossible to assign a cause for its presence.

VIII. **The Sense of Smell.**—The olfactory bulbs and their associated parts are in reality portions of the brain. The peripheral nerves arising from the bulb are distributed to the mucous membrane of the upper portion of the nasal septum and the superior and middle turbinates. The centre for the sense of smell is said to be in the uncinate convolution (FERRIER).

To test the function of the olfactory nerve, non-irritating substances must be used, as pungent vapours affect mainly the trigeminal nerve. Suitable odorous bodies are musk, oil of cloves, or peppermint in convenient containers. It should be remembered that the perception of any single odour is lost in 3 or 4 minutes, but is regained by 1 minute of rest. It is essential that the odorous material should enter the nostrils as a vapour or in a state of fine division, and the act of smelling is usually assisted by "sniffing," which is a modified inspiration. It is also necessary that the mucous membrane should be moist in order that the odorous particles shall enter into solution on the surface of the mucous membrane.

The clinical symptoms relating to the sense of smell are *anosmia*, loss of the sense of smell; *hyperosmia*, increased sensitiveness of the sense of smell; and *parosmia*, subjective perversion of the olfactory sense.

(*a*) **Anosmia.**—The loss of the sense of smell is, in the majority of instances, due to local disease of the nasal mucous membrane—viz., chronic rhinitis, particularly the atrophic variety, and polypi or other new growths. The inhalation of irritating vapours or extremely foul odours may temporarily or permanently abolish the sense of smell. Paralysis of the trigeminal will impair the olfactory power of the affected side because of the deficient secretion and consequent dryness of the mucous membrane which is thus induced. Loss of the power of smell may be a symptom, purely neurotic, of neurasthenia and hysteria.

Less frequently the sense of smell is abolished because of injury or lesion of the olfactory bulb or tract. Falls or blows upon the head, affecting the nerve in its course, may have as their only symptom a persistent anosmia. Caries of the bones supporting the tract or bulb, and basal meningitis or tumours involving the nerve, may be instrumental in causing olfactory anæsthesia. Possibly from atrophy of the nerves anosmia may be one of the symptoms of locomotor ataxia. It is sometimes congenital and results from imperfect development of the olfactory nerve tissue. Partial anosmia may be due to a lesion of the uncinate gyrus or disease of one hemisphere.

(*b*) **Hyperosmia.**—Hyperæsthesia or abnormal sensitiveness of the sense of smell is, in its slighter degrees, not an uncommon symptom in neurotic individuals. More rarely the sensitiveness is extreme and is symptomatic of hysteria and neurasthenia.

(*c*) **Parosmia.**—Olfactory sensations without a physical basis, the apparent odours usually being unpleasant or offensive (*kakosmia*), are not infrequent as hallucinations in the psychoses. A bad odour, like that of burning rags or feathers, may constitute the premonitory aura of epilepsy. Very rarely, after head injuries, ordinary odours are perverted, so that a usually agreeable perfume is perceived as foul. Finally, subjective kakosmia has been associated in a limited number of cases with tumour of the hippocampus.

SECTION XVIII

THE MOUTH

UNDER this heading will be given the diagnostic evidence which may be derived from an examination of the lips, buccal surfaces, gums, teeth, and tongue.

I. **The Lips.**—Thick or thin lips are often a racial characteristic, as in the African or American Indian. Full lips in white races are said to mark a phlegmatic temperament or to indicate a tendency to the worship of Bacchus and Venus; while thin-lipped individuals are apt to be nervous and of somewhat acrid temper. The lips are thickened and coarse in myxœdema and cretinism.

Their *colour* is often significant of anæmia and other conditions in which the skin is pallid, and the first evidence of cyanosis is often seen in them, their slight blueness attracting attention to the existence of cardiac disease.

If the lips are *open*, and particularly if they are dry and cyanosed, it is apt to be indicative of dyspnœa, due to disease of the heart or lungs, especially the chronic forms—e. g., emphysema or failing compensation in valvular lesions. It may be due to disease within the mouth, stomatitis, glossitis, cancrum oris, phlegmonous tonsilitis, or some form of nasal stenosis. If the lips are loose and pendulous it is suggestive of diphtheritic paralysis or chronic bulbar palsy. The open mouth is seen in all conditions of great prostration, and in idiots and some cases of insanity.

Trembling or *twitching of the lips* may be a symptom of general paralysis or chronic bulbar palsy. Convulsive raising of the upper lip is an occasional evidence of severe abdominal pain.

Unilateral deviation of the lips, the angle of the mouth being drawn to one side and downward, if not due to loss of teeth on the opposite side or to the contraction of scars, is indicative of facial paralysis, the latter either existing alone or as a part of a hem plegia.

A brawny, hot *swelling* of the lip may be a small abscess or more extensive and serious carbuncular inflammation. The lips a swollen as a part of the disease in the fortunately rare cancrum or and the swelling may be due to the taking of corrosive poisons, which case the interior of the mouth will also be swollen and reddened. Bites of insects may explain a swelled lip, and among other causes are alveolar abscess, bitten lip in epileptic or other convulsion, and angeioneurotic œdema.

Foam upon the lips is a common symptom during an epileptic seizure, and sometimes in the later stages of cerebral apoplexy. It should be borne in mind that a bit of soap in the mouth is employed by malingerers in simulating epileptic convulsions.

Miscellaneous Affections of the Lips.—*Herpes.*—Vesicles upon the lips are especially common in malarial fevers, pneumonia, and acute coryza, as well as other febrile diseases.

Fissures.—Cracks or fissures (rhagades), or the scars resulting from them, if occurring in infants or children, must be regarded with suspicion as suggestive of inherited syphilis. The vertical crack in the middle of the lower lip, which is occasionally seen, is not of special significance.

Chancre.—A single, small ulcer upon the lip, with an indurated base and accompanied by enlargement of the submental glands, is likely, if in a young person, to be the initial lesion of syphilis.

Mucous Patches.—Flattened, warty outgrowths, strictly delimited, coated with a gray matter, and found at the angles of the mouth, are the mucous patches of the secondary stage of syphilis.

Epithelioma.—A somewhat irregular ulcer, usually upon the lower lip, gradually enlarging, recurrently scabbing over and becoming denuded, at times originating from a wart, is probably an epithelioma, and, if of some standing, the glands beneath the jaw are enlarged. The occurrence of such an ulcer in a middle-aged or old person, its progressive increase in size and the late involvement of the glands, will differentiate it from the initial lesion of syphilis.

II. **The Buccal Cavity.**—**The Odour of the Breath.**—Variations from the normal in this respect may be of considerable value in diagnosis.

In hydrocyanic-acid poisoning the breath may smell of peach kernels or bitter almonds, and an alliaceous or garlicky odour is present in phosphorus poisoning, provided in both cases that too long a time has not elapsed. The narcotic odour of the opium preparations may explain a profound stupor, and the characteristic fumes of ether, chloroform, and alcohol are sufficiently familiar.

Local conditions in the mouth may be accountable for an unpleasant or foul odour of the breath, as in those to whom the cult of the toothbrush is unknown. If sordes have collected upon the teeth, the breath is apt to be stale and musty. A foul odour attends in some degree all forms of stomatitis and glossitis, most fetid in the mercurial and gangrenous forms, less so in scurvy. Caries of the teeth, necrosis of the jaw, pharyngeal or tonsillar diphtheria, follicular tonsilitis, and lacunar concretions are local conditions which may explain the existence of unpleasant emanations.

An intensely fetid breath, in the absence of other sufficient causes, may be due to gangrene of the lung, pulmonary actinomycosis, bronchiectasis, and pyothorax or pyopneumothorax with a fistulous opening into a bronchus.

A common cause of bad breath is some form of gastritis, especially that caused by chronic alcoholism, and it is sometimes due to constipation. In children with gastric disorders it is often merely a sour smell.

A hot, "feverish" breath is common in febrile disorders, and the disagreeable odour is most noticeable in typhus fever, measles, and scarlet fever. An ammoniacal, "urinous" odour is not uncommon in the more severe grades of uræmia. A heavy, sweetish odour, like stale beer or overripe apples, and due to the presence of acetone or diacetic acid, is perceived in bad cases of diabetes mellitus, often preceding or accompanying diabetic coma. Finally, a slight cadaveric smell is at times perceptible in the breath of those who are critically ill.

Puffing Cheeks.—In most forms of coma the cheeks are lax and puff outward with each expiration. The same thing is seen during sleep in toothless elderly persons. Facial paralysis, alone or as a part of a hemiplegia, is the usual cause of outward blowing or flapping of *one* cheek.

Petechiæ: Pigmented Spots.—Small extravasations are occasionally seen upon the buccal mucous membrane and, when present, may be caused by one of the grave anæmias, hæmophilia, purpura hæmorrhagica, and scurvy; or they may be the hemorrhagic infarcts of ulcerative endocarditis.

Brownish or yellowish pigment patches on the buccal and palatal surfaces and the inner aspect of the lips are significant of Addison's disease; and the mucous membrane may exhibit discoloured purplish patches in those saturated with silver salts. Oral inflammations in the coloured races may be followed by pigmentary changes.

The yellow of jaundice and the bluish tint of cyanosis are more or less obvious in the oral mucous membrane.

Exanthems, Erythema, Vesicles.—The rash of measles is often seen upon the pharyngeal and palatal surfaces prior to its appearance upon the skin, and Koplik has described, as absolutely characteristic of measles, small red spots with a minute blue-white centre upon the inner surface of the cheeks, which disappear as the rash develops. The angry redness of scarlet fever involving the entire oral and faucial surfaces is very striking. A slighter redness is present in simple erythematous stomatitis, and the rare occurrence of a salivary calculus with the accompanying redness should be remembered. Vesicles when present may be the beginning of an aphthous stomatitis, herpes, or the eruption of varicella or smallpox.

White Spots, Ulcers, Sloughs.—Small grayish ulcers with reddened margins, which have begun as vesicles, situated upon the inside of the lips and cheeks and the edges of the tongue, are the lesions of aphthous stomatitis, the common "canker sores." Small ulcers on the hard palate in infants may be due to the friction of the rubber nipple; or, if in very weak newborn infants, situated symmetrically on either side of the median line and usually increasing in size, constitute a form of oral disease reported by Parrot. Ulcers on the buccal mucous membrane are also seen in mercurial stomatitis and the sore mouth of syphilis. White, curdlike patches, beginning on the tongue and spreading to the inside of the lips and cheeks, and, if detached, leaving a normal or slightly ulcerated surface, form the disease known as thrush (parasitic stomatitis). Deep ulceration, with the formation of sloughs, may be caused by corrosive poisons, gangrenous stomatitis, and glanders. A case of peliosis rheumatica is reported by Hare, in which sloughing of the cheek occurred.

Dryness of the Mouth.—The secretion of saliva may be checked by various causes. That dryness of the mouth is produced by fright or excitement is a fact within the experience of most individuals. So also with the dry mouth of febrile states. One of the unpleasant symptoms of mouth-breathing is the stiff, dry tongue of the morning awakening. Persistent dryness of the mouth is often significant of diabetes or chronic gastritis, and is not infrequently present in chronic nephritis. Finally, a dry mouth may constitute the disease known as xerostomia (Hutchinson).

Salivation and Dribbling.—The amount of saliva which is secreted in 24 hours is, under normal circumstances, from 2 to 3 pints. Ptyalism or hypersecretion of saliva, in which this amount is greatly exceeded, may occur in early pregnancy and sometimes attends the menstrual period. All forms of stomatitis may cause an increase. The pain due to dental causes, alveolar abscess, or trigeminal neuralgia, and the process of dentition itself, will induce an excessive formation of saliva. It occurs infrequently in hysteria and other psychic neuroses, and in hydrophobia. Ptyalism is sometimes present in the early stages of variola and typhus fever, and has occurred during convalescence from typhoid fever. Thick saliva accumulates in quinsy and mumps.

Certain chemical substances and drugs will produce an increased flow of saliva—viz., acids, aconite, alkalies, antimony, cantharides; copper, gold, iodine, and mercury compounds; muscarine, pilocarpine, and tobacco. Too large a dose of mercury or an unusual susceptibility to its action is the most common example of drug salivation which is met with in practice.

Dribbling of saliva may occur, although the amount is not increased, because of inability to retain it, in idiocy, facial paralysis, diphtheritic paralysis, and chronic bulbar palsy.

III. **The Gums.**—*Colour.*—The pallor of the gums is noticeable in all forms of anæmia. A greenish-blue line at the edges of the gums is significant of poisoning by copper. A blue or gray-blue line, with a grayish deposit upon the teeth, is indicative of lead poisoning, but may be absent if the teeth are well kept. A blue or red line is seen in mercurial stomatitis, while a purple coloration occurs in scurvy. A red line along the gingival margins in young adults was formerly supposed to indicate the imminence of tuberculosis, and may be present in diabetes and the cancerous cachexia, or as a symptom of a chronic affection of the teeth and their sockets (pyorrhœa alveolaris). Lack of cleanliness is not an uncommon cause of marginal redness.

Red, Spongy, or Ulcerated Gums.—Carious or ill-kept teeth, with abundant tartar, may produce red and spongy gums. Gangrenous stomatitis usually involves the gums to a very marked degree, and the less severe forms of oral inflammation will cause a general redness in which the gums will share. Spongy, red, and bleeding, perhaps ulcerated, gums occurring in artificially fed infants may be due to scurvy. A similar condition in an older person may be the result of an idiosyncrasy for mercury or an abuse of the drug.

Swollen or spongy gums are also met with in some cases of digestive disorders, leucæmia, phthisis pulmonalis, diabetes, purpura, and phosphorus poisoning. Ulceration along the line of the gums, rarely extending to the cheeks or tongue, is particularly characteristic of ulcerative stomatitis. Finally, localized or general redness and swelling may attend the eruption of the teeth.

IV. **The Teeth.**—**Dentition.**—The temporary or deciduous teeth appear with greater regularity as to order than as to time. The average order and times of eruption are as follows (Holt):

(1) Two lower central incisors	6 to 9	months.
(2) Four upper incisors	8 to 12	"
(3) Two lower lateral incisors and 4 anterior molars	12 to 15	"
(4) Four canines	18 to 24	"
(5) Four posterior molars	24 to 30	"

At 1 year a child should have	6	teeth.
At 1½ years a child should have	12	"
At 2 years a child should have	16	"
At 2½ years a child should have	20	"

The permanent set arrive as follows:

First molars	6	years.
Incisors	7 to 8	years.
Bicuspids	9 to 10	"
Canines	12 to 14	"
Second molars	12 to 15	"
Third molars	17 to 25	"

Early Dentition.—Eruption of the teeth in advance of the usual dates is not of special significance. It has been noted in children who are the subjects of hereditary syphilis, and also as part and parcel of the precocity exhibited by infants with a transmitted predisposition to tuberculosis.

Delayed Dentition.—The most common cause of delay in teething is rachitis, together with other conditions or diseases involving malnutrition, particularly if occurring in the first 5 or 6 months of the infant's life, so that the delay, if observed, is more significant of the past than the present status. A late appearance of the teeth may be indicative of cretinism. A case of mine had but 4 teeth at 2 years of age.

Difficult Dentition.—There has been, and is, much difference of opinion in regard to the influence of teething in causing disease. The physician is too apt to coincide with the views of the mother or nurse in this respect. The safe rule is to exhaust every other possibility before concluding that the symptoms which may be present are caused solely by dental irritation. The departures from the normal in otherwise robust children which may safely be considered as direct results of the irritation due to erupting teeth are: loss of appetite, disturbed sleep, fretfulness, slight fever (100° to 101°), and either constipation or a slight diarrhœa. In feeble or poorly nourished infants the attacks may be more severe, amounting to an acute indigestion with high fever, the temperature remaining elevated for 2 or 3 days. All other possible causes—e. g., improper food, exposure, fatigue, tonsilitis—must be carefully excluded, and on inspection the gums over the advancing teeth should be red, swollen, and tender, ordinarily with some salivation.

Shape and Structure of the Teeth.—*Notched.*—If the permanent upper central incisors are somewhat rounded and peglike, tapering from gum to edge, with a single shallow and discoloured notch in the edge, it is good but not infallible evidence of inherited syphilis. These teeth are apt to be small, placed somewhat irregularly, and stand apart from one another (Fig. 56). If keratitis and middle-ear disease coexist, a positive diagnosis of syphilis may be made.

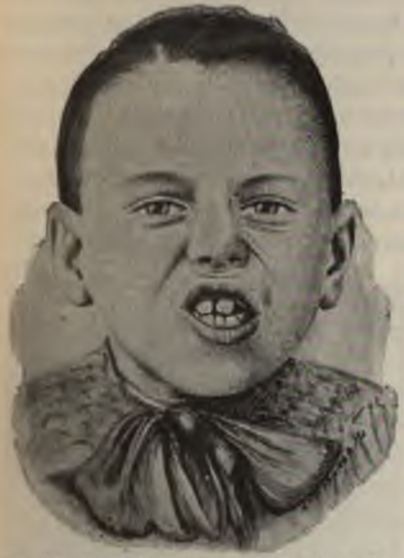


Fig. 56.—Syphilitic "screw-driver teeth;" boy nine years old (Holt).

Dentated or Furrowed.—If the edges of the teeth are dentated, malnutrition or struma is likely to be the cause. Grooves or furrows running transversely across the teeth are indicative of an acute illness during infancy or childhood sufficiently severe to interfere with their nutrition. Pitted teeth, the molars exhibiting the greatest changes, are caused by the various forms of stomatitis.

Loosened and Decayed Teeth.—Loosening of the teeth in their sockets is associated with spongy, ulcerated, or bleeding gums (*q. v.*). Movability of the teeth is therefore usually due either to mercurial stomatitis, pyorrhœa alveolaris, scurvy, purpura hæmorrhagica, phosphorus poisoning, or gangrenous stomatitis.

Early, extensive or rapid dental caries is most commonly due to rachitis, but occurs also in pregnancy, diabetes, and chronic phosphorus poisoning. The influence of carious teeth in causing bad breath and dyspepsia from imperfect mastication, and adenitis by furnishing a source of irritation or infection, should not be overlooked.

Sordes.—A collection of foul material upon the teeth (sordes), sometimes stained with blood oozing from the gums, is seen in conditions of prostration, particularly febrile diseases in which the typhoid status is marked.

Grinding of the Teeth.—Gritting or grinding of the teeth in children during sleep is popularly supposed to indicate "worms," but is usually due to some gastro-intestinal disorder. It also occurs in neurotic children who sleep uneasily, and has been noted in certain maladies of the nervous system, as meningitis, intracranial tumours, hydrocephalus, anterior poliomyelitis, epilepsy, and chorea.

Defective Mobility of the Jaw.—Inability to open or close the mouth may be due to spasm or paralysis of the muscles of mastication. The masseter, temporal, and other muscles concerned in the act of chewing are innervated by motor fibres from the fifth nerve (trigeminus), and *spasm* and *paralysis* of these muscles indicate some interference, organic or functional, with the action of this nerve.

Spasm.—Trismus or lockjaw, a tonic spasm of the masseter and temporal muscles whereby the jaws are held firmly together, may occur as a symptom of trismus neonatorum, tetanus, strychnine poisoning, hysteria, epilepsy, or disease of the brain, and is sometimes a reflex from dentition, gastro-intestinal ailments, and intestinal parasites.

Paralysis.—In this case the masseter and temporal do not contract, and there is inability to masticate on the affected side. Paralysis of these muscles may indicate hemorrhage into the pons, basal lesions (meningitis, tumour, caries) or neuritis.

Pain and swelling from disease of the maxillæ, mumps, quinsy, and trichiniasis may prevent the opening of the mouth and interfere with mastication.

V. **The Tongue.**—An inspection of the tongue is a part of the clinical examination which is seldom neglected by the physician. In general, the tongue should be investigated with reference to its colour, size, coating, dryness, lesions and mobility. Occasionally the condition of the sense of taste requires examination.

Colour and Pigmentation of the Tongue.—The colour of the tongue (including coloured areas or patches), as distinguished from the colour of its coating, is of some diagnostic value. It is pallid in anæmia and bluish in cyanosis. A bright-red tongue may be due to the exanthemata, particularly in the early stage of scarlet fever; or to inflammation of the tongue itself (glossitis), differing from the darker, raw-beef tongue of the adynamic states. Petechiæ, ecchymoses, and infarcts may be found on the tongue, and have the same significance as if found elsewhere. Dark purple or blackish deposits of pigment may indicate an old glossitis, or constitute the discolorations of Addison's disease. A black tongue, as if stained with iron, if not traced to the taking of foreign substances, is an example of the disease termed nigrities. The tongue is yellowish in jaundice, and the yellowness is particularly marked on the under surface of its tip. A series of clearly outlined yellowish-white spots along the edges of the tongue constitutes xanthelasma.

More or less uniform discolorations of the tongue and its coating may be caused by the ingestion of various substances, corrosive or non-corrosive. Among the corrosive substances, ammonia, corrosive sublimate, sulphuric, carbolic, and oxalic acids whiten the tongue; hydrochloric, nitric, and chromic acids produce a yellow colour; acid nitrate of mercury, caustic potash, and caustic soda redden it. Evidence of destructive action is usually present. With reference to non-corrosive drugs or foods, the tongue is stained black by bismuth, charcoal, and iron; red or purple by red fruits or wine; yellow or brown by rhubarb, tincture of opium, tobacco, licorice, or chocolate.

Size of the Tongue.—1. *Enlargement.*—Great enlargement of the tongue is easily determined. A slight increase in size can be assumed to be present if the edges of the tongue are indented by the teeth, the indentations existing only if the tongue is swollen beyond its normal limits.

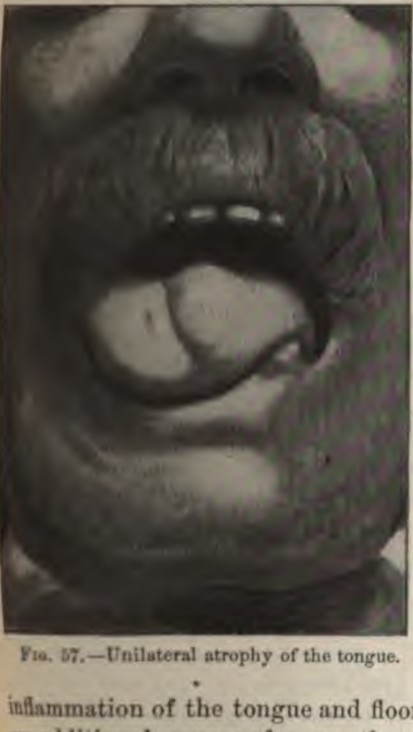


FIG. 57.—Unilateral atrophy of the tongue.

A great enlargement of the tongue is met with in acromegaly and myxœdema. If associated with inflammatory symptoms it is an acute glossitis, due usually to irritant poison or sepsis. The inflammation and swelling may be unilateral (hemiglossitis), and in this case a neurotic origin has been alleged. Variola, foot and mouth disease (aphthous fever), salivary calculus, inflamed ranula, and actinomycotic, erysipelatous, or other inflammation of the tongue and floor of the mouth (angina Ludovici) are additional causes of a greatly enlarged or swollen tongue. Tumours of the tongue are also responsible for an irregular and sometimes great increase in its size. A slow-growing, painless nodule in the tongue may be a gummatous deposit. A moderate increase in the size of the tongue, as indicated by the imprint of the teeth upon its edges, is seen as a symptom in anæmia, chronic gastric catarrh, stomatitis, scurvy, and typhus fever. If cyanosis from venous obstruction is present, the tongue will be swollen and somewhat edematous.

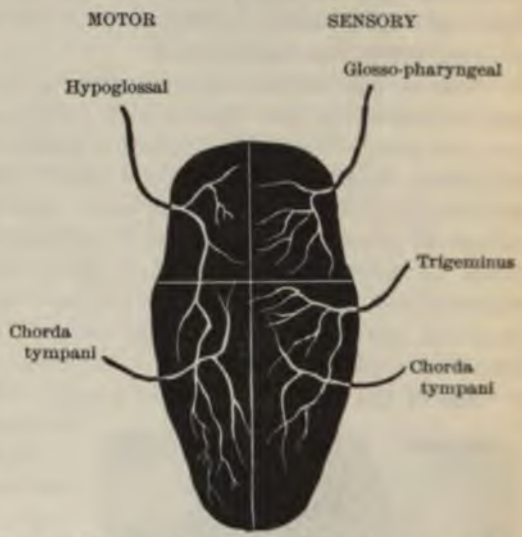


Fig. 58.—Diagram showing the motor and sensory supply of the tongue.

2. *Shrinking or Atrophy of Tongue.*—The tongue is small after a profuse hemorrhage, and may become noticeably lessened in size in the later stages of typhoid fever.

In atrophy of the tongue, the organ has a shrunken appearance, and its mucous membrane is thrown into folds. It is due to some affection of the hypoglossal or motor nerve of the tongue involving its nucleus or its peripheral portion. It is always conjoined with paralysis, and the paralysis and atrophy affect one or both lateral halves of the tongue according as the lesion is unilateral or bilateral. Unilateral atrophy of the tongue (Fig. 57) is sometimes associated with facial hemiatrophy. If the cortical connections of the nucleus are involved there will be paralysis but no atrophy, or if the latter be present it is slight. A contracted condition of the tongue, due to the absorption, under treatment, of a lingual gumma, may be mistaken for paralytic atrophy unless the possibility of such an error is remembered.

Spasm, Tremor, and Paralysis of the Tongue.—The motor supply of the muscles of the tongue is for the most part derived from the hypoglossal nerve (Fig. 58). The same nerve also supplies the muscles which fix and depress the hyoid bone in chewing and swallowing. Two muscles are not supplied by the hypoglossal—namely, the lingualis inferior, which is innervated by the chorda tympani branch of the seventh (facial) nerve, and the palato-glossus by motor fibres from the fifth nerve.

Spasm of the Tongue.—Spasm of the tongue is a rare symptom. It may be tonic, in which case the tongue is contracted and rigid; or clonic, the tongue jerking and twitching irregularly, or being protruded and retracted very rapidly, 40 or 50 times a minute (Osler). The movements usually involve both halves of the tongue, but may be unilateral. Clonic spasm of the tongue is usually a part or symptom of chorea, hysteria, and the epileptic convulsion, or is associated with mimic tic. Stuttering is a spasmodic affection of the lingual muscles. A curious disorder, of which a few instances have been reported in those who use the voice incessantly in public, is a spasm of the tongue on attempting to speak, a condition analogous to writer's cramp. Lingual clonic spasm is an occasional symptom in disseminated sclerosis, general paralysis, and melancholia. In rare cases irregular spasmodic movements of the tongue are due to hypoglossal irritation without discoverable cause. Reflex irritation of the fifth nerve has been held responsible for lingual spasm, and, in some instances, disease of the central nervous system produces it.

Tonic spasm of the tongue is indicative of hysteria or reflex irritation through the fifth nerve, occurring in nervously weak and debilitated persons. It may coexist with tonic spasm of other voluntary muscles in Thomsen's disease (myotonia congenita).

Tremor of the Tongue.—A coarse tremor or trembling of the tongue is most frequently symptomatic of chronic alcoholism, and is seen as well in delirium tremens. It is usually present in the typhoid state when the tongue is protruded, and occurs also in bromism. It may exist as a part of paralysis agitans. A fibrillary tremor or fine twitching of the muscular bundles of the tongue may be seen in disseminated sclerosis, general paralysis, and in bulbar paralysis when the lingual muscles begin to atrophy. Finally, the tongue trembles in neurotic persons, particularly if excited or agitated.

Paralysis of the Tongue.—If one side of the root of the tongue as it lies in the mouth is higher than the other, and if when protruded it deviates toward the same side, *unilateral* lingual paralysis exists. There is apt to be some interference with the speech, and a slight difficulty in chewing and swallowing. If the tongue lies motionless in the floor of the mouth, the patient being unable to protrude it, and the functions of speech, mastication and deglutition are greatly impaired, there is *total* lingual paralysis. In both cases there may or may not be atrophy and fibrillary twitching. If atrophy does not take place it indicates a cortical or supranuclear lesion; but if one or both sides of the tongue are shrivelled, the lesion is nuclear or infranuclear.

Bilateral paralysis without atrophy, a rare finding, is due to pseudo-bulbar paralysis, a condition associated with symmetrical lesions (softening) of both cortical centres of the tongue. Bilateral paralysis with atrophy is usually a symptom of true bulbar paralysis, and occasionally of progressive muscular atrophy. Unilateral paralysis without atrophy is, in the majority of cases, a part of a hemiplegia. If the fibres of the nerve are involved in their course through the medulla after leaving their nucleus, the lingual paralysis will be on the side opposite to the hemiplegia, and the tongue, when protruded, deviates toward the sound side.

Lingual paralysis, with atrophy either of one or both sides of the tongue according to the situation or extent of the lesion, may also exist as a symptom of general paralysis, chronic lead poisoning, locomotor ataxia (rarely), embolism or thrombosis of the vascular supply of the nuclei, basal meningitis, tumour of the base, syphilitic or other disease of the bone containing the anterior condyloid foramen or of the first cervical vertebra, tumours of the upper portion of the cord, and wounds of the neck. A hospital tramp in my service simulated total glossoplegia very accurately for the sake of a night's lodging.

Scars, Fissures, and Ulcers of the Tongue.—*Scars* upon the tongue may be the result of healed ulcers, epilepsy, bulbar palsy, accidental biting of the tongue during restless sleep, careless chewing, or a fall while the tongue is between the teeth. Before deciding as to the cause a careful inquiry for a history of syphilis should be made, as they may be the result of healed specific lesions.

Fissures of the tongue are not infrequently of normal occurrence, especially in elderly people. At other times they are significant of the habitual use of irritating food or drink which has caused a chronic glossitis. The median longitudinal fissure is generally the deepest. Very deep inflamed fissures may be due to dissecting glossitis, which in some instances is syphilitic. Fissures or losses of substance at the edges of the tongue are usually of syphilitic origin, but may be due alone to the irritation of a rough or broken tooth. Fissures are also met with as the result of erysipelatous inflammation, chronic hepatic disease, chronic dysentery, and diabetes mellitus.

Ulcers of the tongue are usually due to aphthous stomatitis, less frequently to the ulcerative form. They are acute and painful. Very shallow, red, and glazed ulcerated surfaces occur in chronic superficial glossitis. Multiple ulcers, gray, indolent, stellate, with enlarged cervical glands, may be tuberculous, and are almost always secondary to tuberculous disease elsewhere. Somewhat similar multiple ulcers may be syphilitic, but then the glands are rarely enlarged. Multiple small ulcers, preceded by vesicles, may be found in smallpox, varicella, measles, and erysipelas, as well as in pemphigus, herpes, and eczema of the tongue.

A single ulcer with a hard base and enlarged cervical glands may be either the initial lesion of syphilis or an epithelioma. In the former case the age of the patient is usually under forty, the lesion is on the tip of the tongue, it is not very painful and improves under treatment. A mucous patch may become ulcerated, and a gumma may break down, forming a deep sore. A single ulcer, if opposite a rough or jagged tooth, may be simply the result of continued traumatism, but the presence of an irritating tooth does not exclude syphilis. An ulcer on the frenum linguæ may occur during the course of pertussis from the thrusting of the under surface of the tongue against the lower incisors during the paroxysms of cough.

Miscellaneous Symptoms and Affections of the Tongue.—*Eczema* of the tongue gives rise to a condition variously known as geographical tongue, annulus migrans, or wandering rash. Ring-shaped patches, red and denuded of epithelium, are seen on the tongue, and under observation are found to spread at the edge while healing at the centre, coalescing and forming irregular areas with curved outlines.

The *smoker's patch* is a red, yellowish or pearly and slightly elevated plaque, smooth and not ulcerated, usually situated upon the dorsum of the tongue near its tip.

Leucoplakia buccalis (*ichthyosis*, *leucoma*, *leucokeratosis*, *buccal psoriasis*) consists of slightly elevated, thickened patches of irregular shape, not ulcerated, white, smooth, and hard upon palpation. They may furnish the starting point of an epithelioma.

General Diagnostic Appearances of the Tongue.—These relate to the character of the fur or coating which may be present, or its disappearance; to the colour of the tongue, and particularly to its degree of moisture. The coating consists of accumulated epithelium, microorganisms, and food detritus. Dryness of the tongue, when not caused by mouth breathing or coma, is of very considerable importance as an indication of general adynamia and prostration. The following states of the tongue are of value in diagnosis and prognosis:

(1) A thin, white, even furring of the tongue is normal in many healthy individuals, particularly those who are in the habit of smoking, and in mouth breathers. It may be indicative of nasopharyngeal

catarrh or mild gastric disorders. Such a coating is always found in moderately febrile states.

(2) A flabby, swollen, indented tongue, covered with a uniform yellow, pasty fur, is indicative of catarrhal gastritis or gastro-duodenitis, usually of some standing. Heavy smokers and drinkers show a similar fur on rising in the morning, and it is also produced by the continuation of a moderate fever.

(3) A narrow tongue, with a deep median fissure, on each side of which is a thick, rough fur, the tip and edges of the tongue being red and denuded, is characteristic of the typhoid status, whether arising from typhoid fever or not, although it occurs particularly in the latter disease.

(4) If the tongue just described becomes dry, brown, and fissured, is tremulously and slowly protruded and the patient must be told to retract it, the typhoid status is well marked and ominous of evil. So also is it if the coating desquamates and—

(5) The tongue becomes dry, red, and glazed, the so-called "beefy" tongue. A change from either of these conditions to cleanliness and moisture is a favourable omen.

(6) A broad, flabby, gray tongue, with reddened, irregular spots, resembling in shape the lesions upon a worm-eaten leaf, and occurring in a child, is indicative of a peculiar form of gastro-intestinal catarrh (mucous disease of children).

(7) If the tongue is covered with a white fur through which project greatly swollen and bright-red fungiform papillæ, it is the so-called "strawberry tongue," seen most frequently in the early stage of scarlet fever, but not pathognomonic of this disease, as it may be present in other acute specific infections. Some writers bestow this term upon a tongue which has lost its coating and shows a uniformly red surface with projecting papillæ (raspberry tongue), but the foregoing description conforms more nearly to the fancied likeness.

(8) A white, creamy fur is often seen upon the tongue of patients who are upon an exclusive milk diet. Chalk rubbed upon the tongue is said to have been employed by malingerers for the purpose of simulating gastric disease.

(9) Unilateral furring of the tongue may result from neuralgia of the second and third divisions of the trigeminus and from fracture involving the foramen rotundum, through which the second division passes. It is also noted with unilateral lingual paralysis (e.g., in hemiplegia), which interferes with the frictional cleansing of the organ.

(10) Small and limited furrings of the tongue may indicate local irritation from a rough tooth or an inflammation, as of one tonsil.

(11) A grayish coating of the tongue in adults or a white coating in children may be due respectively to an unusually extensive growth of leptothrix or thrush.

Taste.—The 4 primary taste sensations are bitter, sweet, acid, and salt. There is some disagreement among physiologists as to the nerves concerned in the conduction of gustatory sensations, due to the fact that there are considerable variations in the course of the taste fibres. It may be said that usually these fibres run both in the glosso-pharyngeal and trigeminus, the former supplying the posterior third of the tongue, the latter the anterior two thirds (Fig. 58). Nevertheless, complete loss of taste has resulted from trigeminal disease alone, more rarely from lesions affecting only the glosso-pharyngeal nerve, so it must be admitted that the gustatory fibres may at times be present solely in one or the other. Some of the taste fibres may pass by way of the chorda tympani, as a partial loss of taste may be present in facial paralysis.

The sense of taste is tested by directing that the tongue be protruded and kept so while drops of various solutions are placed upon the anterior and posterior surfaces of the dorsum upon each side of the median line. For bitter, a solution of quinine is employed; for sweet, sugar; for salt, sodium chloride; for acid, vinegar. Ordinarily a solution of sugar is sufficient to detect any impairment of taste. The metallic taste caused by the passage of a weak galvanic current through the tongue may be utilized as a convenient test.

The disorders of the gustatory sense are *ageusia* (loss or impairment of the sense of taste) and *parageusia* (perversion of the sense of taste).

Ageusia.—If the taste sense is impaired or absent it may be due to local unhealthy conditions of the mucous membrane of the tongue involving the taste buds or end organs of the gustatory fibres, as in a furred or coated tongue, or a tongue which has been exposed to the action of irritating condiments. If the tongue is dry the taste is much lessened or abolished. As the sense of smell plays a considerable part in the production of the sensations ordinarily referred to the sense of taste, a loss of the latter may be complained of in the various diseases of the nose (coryza, polypus, etc.) which cause anosmia (*q. v.*). In all these cases the ageusia is bilateral and general.

Aside from local conditions of the mouth and nose, a loss of the taste sense, especially if unilateral and localized, is indicative of disease of the glosso-pharyngeal nerve (posterior third of the tongue); or, as is more commonly the case, of trigeminal disease (anterior two thirds of the tongue). Slight unilateral impairment of the sense of taste (*hemiageusia*) is usually due to facial paralysis. Large doses of the bromides blunt the sense of taste.

The lesions affecting the taste may be basal meningitis, tumours, and injuries. Ageusia may be one of the manifold symptoms of hysteria, which, with facial paralysis, is the commonest cause of a partial loss of the taste sense. In every case associated symptoms must determine the diagnosis.

Parageusia.—Purely subjective perversions of the sense of taste are usually indicative of hysteria, but may be hallucinations of the insane or constitute the aura of epilepsy.

Various "bad tastes" are frequently a source of complaint. In gastro-duodenal catarrh (biliousness) a coppery taste is often mentioned, and in jaundice the mouth is bitter. When the tongue is furred or coated from any cause the taste is diversely described as bad, foul, sweetish, or sour. The administration of certain drugs, such as potassium bromide and iodide or tartar emetic, may give rise to abnormal taste sensations.

SECTION XIX

THE PALATE, TONSILS, AND PHARYNX

TECHNIC OF EXAMINATION

To examine the pharynx, a tongue depressor is usually required. For house visits, especially in cases of contagious diseases, a spoon with a broad, smooth handle is preferable because of the danger of conveying infection by the use of a portable depressor. Light from a window, lamp, candle, bicycle lamp, or match may be utilized. For office work, the head mirror and Argand or Welsbach burner, or electric light, with a suitable condenser, will be used, so also a right-angled tongue depressor, the width of which should be at least one inch.

To use the tongue depressor, desire the patient to open his mouth, but not to protrude the tongue over the lower teeth. Ask him to sound *ah* (*a* as in father), thus lowering the posterior half of the tongue. Then carry the blade of the depressor in until it passes well (but not too far) over the highest part of the dorsum of the tongue. By pressing down and at the same time pulling somewhat forward with the instrument, the tonsils and pharynx may be fully exposed.

Note first the general colour of the fauces and throat as a whole. Observe the shape of the hard palate and the existence of a perforation of the soft palate, swelling of the uvula, vesicles or ulcers, and perhaps test for paralysis or anæsthesia of the palate.

Note enlargement of the tonsils (acute or chronic), the presence of exudate, evidences of suppuration, ulcers (tuberculous, syphilitic, cancerous), and use a probe to determine the consistence and adherence of exudate or follicular plugs if present.

With reference to the pharynx, note enlarged follicles, veins, pulsation, exudate, ulcers, bulging of the posterior wall (retropharyngeal abscess), and, if required, anæsthesia of the mucosa and spasm or paralysis of the pharyngeal muscles.

An abnormal general redness of or discrete eruptions upon the palate, fauces, and pharynx may be due to simple inflammations of the mucous membrane, measles, scarlatina, rötheln, roseola, epidemic influenza, erysipelas, chronic gastritis, irritant poisons, iodism, and belladonna poisoning. The vesicles of varicella, variola, and herpes may also be seen. So also with petechiæ, infarcts, and extravasations of blood or bleeding surfaces.

An abnormal general pallor of the same surfaces is noted in the anæmias, and the yellow colour of jaundice may also be seen here.

I. **The Palate.**—(*a*) A high, narrow, and arched hard palate constitutes one of the stigmata of degeneration.

(*b*) *Perforation* of the soft palate or its adhesion, either partial or complete, to the posterior pharyngeal wall, is usually the result of syphilitic ulceration. Ulcers of the palate in the adult are almost always tertiary syphilitic manifestations.

(*c*) *Bilateral paralysis* of the soft palate is to be suspected if there is regurgitation of fluids through the nose on attempting to swallow, and if there is failure to pronounce correctly certain sounds which require the approximation of the soft palate to the posterior pharyngeal wall—e. g., the patient says "beng" for "beg." If the paralysis is unilateral these symptoms are wanting. Inspection of the soft palate during the utterance of the long "*ah*" sound will show under normal circumstances that both sides of the palate arch upward. If the whole palate remains motionless during phonation there is bilateral paralysis; if but one side moves, it is unilateral. The soft palate is in all probability innervated by the accessory portion of the eleventh nerve (spinal accessory) by way of the pharyngeal plexus of the pneumogastric, although it is proper to state that some authorities consider the pneumogastric itself to be the motor nerve of the palatal muscles.

Paralysis of the soft palate is most commonly due to diphtheritic neuritis, but is also caused by bulbar paralysis, tumours, basal meningitis, and vertebral caries.

(*d*) *Anæsthesia* of the hard and soft palates indicates disease involving the second division of the fifth nerve, from which the sensory supply of the palatal region is derived.

(*e*) *Vesicles* arranged in annular shape upon the soft palate, or perhaps upon the pharyngeal wall, and attended with a disproportionate amount of pain, constitute an example of that rare disease, herpes of the throat.

(*f*) *Swelling of the uvula* is common in all inflammatory conditions of the pharynx and tonsils. It may become edematous in nephritis, severe anæmias, and conditions of debility. A bloody extravasation into the uvula may occur in peliosis rheumatica or in diseases which cause hemorrhagic infarcts elsewhere.

II. **The Tonsils.**—(*a*) *Acute swelling* of the tonsils, fever and marked constitutional symptoms being present, with a punctate, perhaps confluent, white or grayish exudate occupying the tonsillar crypts or surfaces, may be due to follicular tonsilitis, suppurative tonsilitis, tonsillar diphtheria, scarlet fever, or measles.

(*b*) If the exudate, as in (*a*), becomes confluent and spreads to the pillars of the fauces, the soft palate, and pharynx, it is, in the vast majority of cases, diphtheria.

(*c*) If the exudate, as in (*a*), remains stationary or disappears, and the soft palate on one side becomes reddened, tumid, and greatly swollen, pushing the tonsil to or beyond the median line, it is a suppurative tonsilitis (quinsy).

(*d*) Permanent enlargement of the tonsil is due to chronic inflammation, and is frequently associated with postnasal lymphoid growths. The symptoms and signs of mouth breathing are usually to be found.

(*e*) If there are deep ulcers upon both tonsils, circular in shape and with a gray surface, the remaining portion of the tonsil presenting a normal appearance, it is almost certain that they are of syphilitic origin.

(*f*) Irregular, grayish, painful ulcers upon the tonsils, occurring in the later stages of pulmonary phthisis and associated with similar ulcers upon the pharynx and larynx, may be considered tuberculous, although their appearance upon the tonsils is a rare event.

(*g*) Deep, spreading ulceration upon an enlarged tonsil, from which an offensive, sanious discharge issues, is, if it occurs in an elderly person, in all probability cancerous.

(*h*) Whitish plugs in the lacunæ of chronically enlarged tonsils may be composed principally of leptothrix threads or are small concretions.

III. **The Pharynx.**—(*a*) Congestion of the pharyngeal mucous membrane is seen in acute and chronic inflammations of the pharynx, and passive or venous hyperæmia may be significant of cardiac valvular disease, aneurism, or mediastinal tumour.

(*b*) Uvula, fauces, and pharynx may pulsate (communicated carotid throbbing) in aortic insufficiency and profound anæmia, also (same conditions) rhythmic turgescence of soft parts may be seen.

(*c*) A dry, reddened condition of the pharynx, with slight constitutional symptoms, is an acute catarrhal pharyngitis. There may be an indistinct punctate exudate analogous to that which is present in follicular tonsilitis.

(*d*) A patient complaining of sore throat and intense dysphagia, with slight or absent objective signs, has in all probability a rheumatic pharyngitis.

(*e*) A relaxed dry or moist mucous membrane showing dilated veins and numerous projecting, rounded follicles on the posterior wall of the pharynx, is an example of chronic pharyngitis.

(*f*) A swollen and injected mucous membrane, with severe constitutional symptoms and ending in suppuration, is a phlegmonous pharyngitis, fortunately of rare occurrence.

(*g*) A membranous exudate upon the pharynx is in a large majority of cases diphtheria, and should be regarded as such unless the diagnosis is disproved by a bacteriological examination. Membranous inflammations not due to the Klebs-Loeffler organism—the diphtheroid affections—are for the most part the result of infection by the *Streptococcus pyogenes*.

(*h*) Ulcerations, rounded in shape, yellow, sloughy, and surrounded by a reddened zone, if occurring in an adult, are frequently due to syphilis. In the absence of other definite symptoms the therapeutic test will decide the question. Small superficial ulcers are usually follicular, and occur in connection with chronic pharyngitis. Irregular ulcers, with undefined boundaries and yellowish-gray floors, if found in a patient far gone with phthisis, are undoubtedly tuberculous. Small round or oval ulcers of the pharynx may be found toward the close of typhoid fever. Ulcers also occur in diphtheria, lupus, and cancer.

(*i*) Bulging forward of the posterior pharyngeal wall, with the rapid onset of dysphagia and dyspnœa, particularly in children, is due to an acute retropharyngeal abscess. If chronic and without urgent symptoms it is probably an abscess caused by cervical caries affecting the bodies of the vertebræ.

(*j*) *Motor and Sensory Disorders of the Pharynx.*—(1) *Anæsthesia* of the pharynx is indicative of some interference with the sensory functions of the glosso-pharyngeal and pneumogastric nerves, and is found in diphtheritic neuritis, hysteria, and bulbar paralysis. Globus hystericus is a functional disorder of the ninth nerve.

(2) *Spasm* of the pharyngeal muscles on attempting to swallow is a functional affection of the motor portions of the same nerves. It may be present in neurotic and hysterical individuals, in hydrophobia true or false, in tetanus and strychnine poisoning. It is probably an element in globus hystericus.

(3) *Paralysis* of the pharynx, if bilateral, causes difficulty in swallowing, the food is not passed into the esophagus, and portions may enter the larynx. If the paralysis is unilateral, there is little if any difficulty in deglutition. Pharyngeal paralysis indicates an involvement of the nuclei of the ninth and tenth cranial nerves or of the nerves in their course, as in bulbar paralysis, Landry's paralysis, and neuritis; or basal lesions, as meningitis, tumour, or aneurism.

SECTION XX

DYSPHAGIA

DYSPHAGIA, difficulty or pain in the act of swallowing, attends a variety of conditions, the more important of which are as follows:

Mouth and Fauces.—Glossitis, cancer or other tumour of the tongue, and the various forms of stomatitis may cause painful or difficult swallowing. So also with tonsilitis, follicular or suppurative, tonsillar concretions, and the various forms of pharyngitis, including rheumatism of the pharynx and retropharyngeal abscess. Diphtheria, and the eruptions of scarlet fever (erythema), measles (macules), varicella, variola, and herpes (vesicles) may be responsible for dysphagia. Difficult swallowing may be due to spasm or paralysis of the palate and pharynx, the causes of which have just been considered. Trichiniasis may cause difficulty in chewing and swallowing because of the stiffness and soreness of the muscles due to the presence of the parasite.

Larynx.—Ordinary laryngitis causes some uneasiness in swallowing. Dysphagia may be due to cancer of the larynx, and the atrocious pain attending tuberculous ulceration is well known.

Esophagus.—The diseases of the esophagus which lead to difficulty or pain in deglutition are: spasm (œsophagismus), inflammation (œsophagitis), in which case pain is felt in the episternal notch and behind the upper sternum, cancer, stricture, and impacted foreign body.

Pressure from Outside.—Dysphagia, in the absence of demonstrable lesions in the mouth, pharynx, larynx, and esophagus, may be caused by the pressure of an enlarged thyroid gland, thoracic aneurism, enlarged bronchial glands, lymphadenoma, mediastinal tumour, and large pericardial or pleural effusions.

SECTION XXI

EXAMINATION OF THE LARYNX AND LINGUAL TONSIL

THERE is required a strong light (Argand gas burner, student lamp, Welsbach burner, electric light) with a suitable condenser (Mackenzie's) and various sizes of laryngoscopic mirrors. The shank of the laryngoscopic mirror should be stronger and much less flexible than some of those which are now in the market. The mirror for ordinary use should be about 1 inch or a little less in diameter. A forehead mirror, 3 to 3½ inches in diameter, with a central perforation, worn preferably over the left eye, is employed to direct the light. The patient sits erect, the examiner facing him. The light is placed to the patient's right on a level with his mouth. The laryngeal mirror is warmed, its heat tested on the examiner's hand, and the light directed upon the patient's mouth. He is then desired to throw his head slightly backward and, seizing the tongue between his thumb and forefinger, a handkerchief having been interposed to prevent slipping, to roll it out, forward and downward over the edge of the lower teeth. At the same time ask him to sound *ah* (*a* in father), which lowers the root of the tongue and raises the soft palate. The throat mirror, held delicately like a pen, reflecting surface down, is then passed over the dorsum of the tongue without touching anything until its back rests against the uvula and soft palate. The mirror is then to be carried upward and backward, pushing the soft palate and uvula before it, until it rests almost against the posterior pharyngeal wall. By slightly raising or lowering the handle of the mirror the epiglottis, larynx, and surrounding parts may be brought into view.

The base of the tongue should be examined, by bringing the mirror somewhat forward, for the presence of hypertrophy of the lingual tonsil, a mass of rounded projections bulging backward into the space between the tongue and epiglottis (glosso-epiglottic fossa); for red and painful swelling, with or without punctate or membranous exudate, somewhat similar to that which is seen in lacunar tonsilitis; and for ulceration.

The epiglottis varies considerably in shape and position, and in some cases may interfere seriously or totally with a good view of the larynx. It should be inspected, its anterior surface in particular, for cysts or edematous swelling. Passing backward from either side of the base of the epiglottis to the arytenoid cartilages are the ary-epiglottic folds. Outside of these folds lie the pyriform sinuses.

In the larynx the white gleam of the true vocal cords is usually the first thing to catch the eye. The funnel shape of the larynx and the depth of the vocal cords (1 inch) below the margin of the ary-epiglottic folds are not appreciated from a study of the usual cuts, because of the lack of perspective of the latter. The true cords are pearl-white in colour, reaching from the apex of the thyroid cartilage anteriorly to the arytenoid cartilages posteriorly. Their anterior ends are often hidden from view by the epiglottis, but if the patient is requested to sound a high-pitched, almost falsetto, *eh*, the epiglottis is lifted forward and the vocal cords come together, lying parallel and almost in contact. During respiration the vocal cords diverge posteriorly, leaving between them a triangular opening, base to the rear. On each side of the true cords the dark openings of the lateral ventricles may or may not be seen, and a little above and to the outside of these are the reddish ventricular bands (the false vocal cords). At the posterior extremities of the vocal cords are two knoblike elevations or prominences, the arytenoid cartilages. When the vocal cords are widely separated during forced inspiration the trachea may be seen as far down as its bifurcation, including the openings of the primary bronchi.

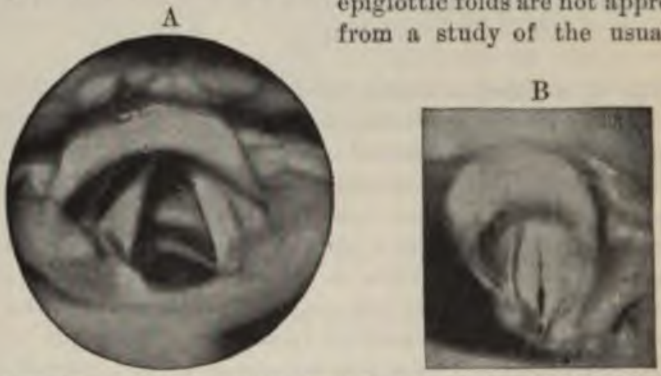


FIG. 59.—Photographs of normal larynx.—A, during respiration; B, during phonation.

My thanks are due to Dr. Thomas R. French, of Brooklyn, for permission to insert two of the unique photographs of the larynx, made by him, and hitherto unpublished. One (Fig. 59, A) is taken during respiration, the other (Fig. 59, B) during phonation.

In studying the larynx and its parts, one observes the presence of swelling or ulceration. Tumours on, above, or below the vocal cords may be noted. In tuberculosis there may be swelling of the epiglottis and ary-epiglottic folds and, if laryngeal phthisis exists, multiple ulcers may be seen, especially on the fold between the arytenoid cartilages (interarytenoid fold). Syphilitic or cancerous ulcers are usually single. A gray-pink or reddish colour of the vocal cords is indicative of laryngitis, and pinkish nodules may be present on the edges of the cords. General pallor of the mucous membrane of the larynx may attend certain cases of laryngeal tuberculosis; general redness is present in most inflammatory or syphilitic laryngeal diseases.

Laryngeal Paralysis.—Finally, the mobility of the vocal cords should be determined by causing the patient to phonate (*ah* or *eh*) and to breathe somewhat rapidly, observing the manner in which the vocal cords approach and depart to and from each other and the middle line (Fig. 60, to which subsequent references apply). The movement of each cord should be estimated separately. Under normal circumstances the cords meet in the middle line during phonation (A) and separate widely while breathing (B), especially during inspiration. All the muscles of the larynx are supplied by the recurrent laryngeal except the crico-thyroid, which receives its motor innervation from the superior laryngeal branch of the pneumogastric.

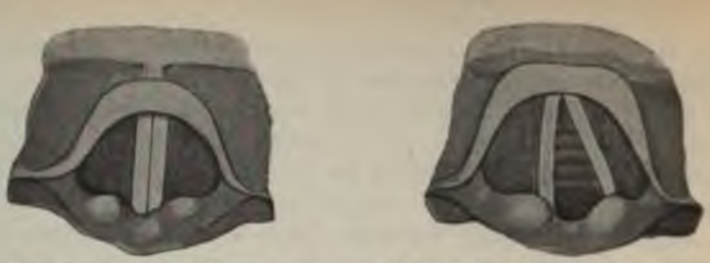
(1) If there is *total bilateral paralysis* of the recurrent nerves, the cords remain in the "cadaveric position," i. e., midway between the median line and extreme abduction (C). They do not move inward during phonation, nor outward during inspiration.

(2) If the paralysis is *unilateral* (usually on the left side), the paralyzed cord remains in the cadaveric position (D), while its fellow moves in and out during phonation and respiration, even crossing the middle line during phonation (adduction).

The foregoing description refers to total paralysis of all the muscles supplied by one (or both) recurrent laryngeal nerves. Although all of these muscles are innervated by the same nerve, yet either the abductors or the adductors may be alone affected, paralysis of the abductors occurring first and most frequently.

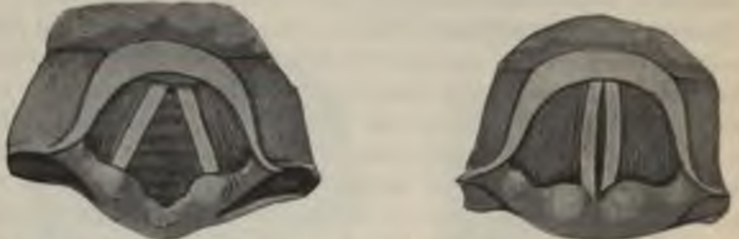
(3) In *abductor paralysis* (affecting the posterior crico-arytenoid muscles) one or both sides may be involved. If unilateral, the paralyzed cord stands in the median line and does not move outward during inspiration (E); if bilateral, the cords lie together as during normal phonation, but fail to move outward during inspiration (F).

(4) In *adductor paralysis* (affecting the lateral crico-arytenoid muscles), if bilateral, the cords lie apart, as they do during normal inspiration (B), but fail to come together (or make a sudden and



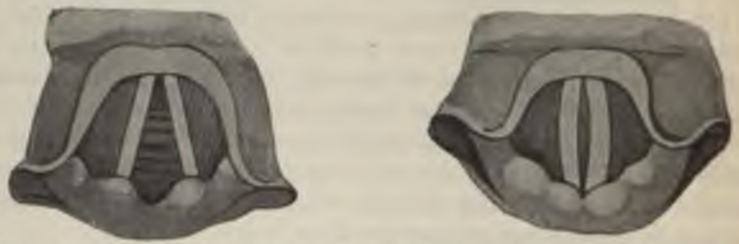
A. Normal larynx during phonation.

E. Right-side (abductor) paralysis of posterior crico-arytenoid muscles.



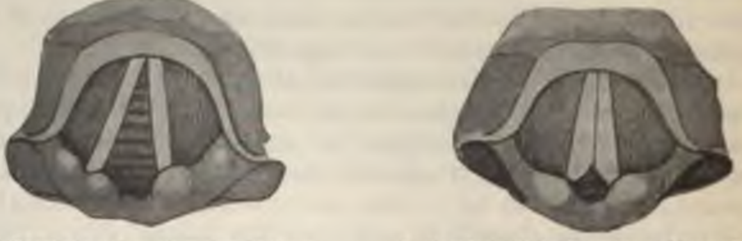
B. Normal larynx during deep inspiration; also position in adductor paralysis (lateral crico-arytenoid muscles).

F. Bilateral paralysis of the posterior crico-arytenoid muscles.



C. Bilateral paralysis of the recurrent nerves.

G. Paralysis of both internal thyro-arytenoid muscles.



D. Paralysis of the left recurrent nerve.

H. Paralysis of the interarytenoid muscle.

Fig. 60.—Showing paralyses of the vocal cords (after Wesener).

unsuccessful attempt to do so) when phonation is attempted. If the paralysis is unilateral (rare), one cord fails to approach its fellow in the median line during phonation.

(5) If the internal thyro-arytenoid muscles (adductors) alone are paralyzed, the cords endeavour to approach the median line during phonation, but the movement is only partly accomplished, thus leaving an elliptical opening between them (G).

(6) If the anterior $\frac{2}{3}$ or $\frac{3}{4}$ of the cords come together during phonation, leaving a triangular cleft or space posteriorly (H), there is paralysis of the transverse adductor, the interarytenoid muscle.

Causes and Diagnostic Value of Laryngeal Paralysis.—Paralysis of the muscles of the larynx is rarely if ever due to cortical disease, but is caused by lesions affecting the nuclei or origin of the vagus in the medulla or the nerve itself at some part of its course. Thus it may be implicated at its deep origin by the lesions of bulbar paralysis, syringomyelia, locomotor ataxia, or multiple sclerosis; or an injury, tumour, or disease of the base of the brain may involve the nerve near its superficial origin. The causes of paralysis most frequently encountered are those which affect the pneumogastric or its laryngeal branches in the neck or the thorax—viz., in the *neck*, enlarged cervical glands, enlarged thyroid gland, tumours of the neck or pharynx, and caries of the cervical vertebræ; in the *thorax*, aneurism of the aorta, abscess, tumour, or enlarged glands in the mediastinum, esophageal cancer, and, rarely, compression resulting from inflammation of the pericardium or pleura and pulmonary tuberculosis. The nerve and its branches may become inflamed (neuritis) or the laryngeal muscles themselves undergo pathological changes terminating in paralysis as an effect of the toxines of a number of the acute specific infections, notably diphtheria; or chronic poisoning, especially from lead and alcohol.

Total bilateral paralysis is most commonly indicative of aneurism of the thoracic aorta, cancer of the esophagus, or great thyroid enlargement.

Abductor paralysis is usually due to organic disease, is commonly unilateral, and is the most frequent variety of laryngeal palsy.

Adductor paralysis, if bilateral, is as a rule of functional origin (especially hysteria); if unilateral, it is due to hysteria or lead poisoning.

Paralysis of the *interarytenoid* muscle (adductor) may be the result of catarrhal laryngitis, but is most apt to be a symptom of hysteria.

Paralysis of the *internal thyro-arytenoid* muscles (adductors) is a not uncommon result of overuse of the voice and laryngeal inflammation.

The following table, from Gowers' work, is frequently quoted in this connection (see also Voice and Speech, in the following section):

SYMPTOMS	SIGNS	LESION
No voice; no cough; stridor only on deep inspiration.	Both cords moderately abducted and motionless.	Total bilateral palsy.
Voice low pitched and hoarse; no cough; stridor absent or slight on deep breathing.	One cord moderately abducted and motionless, the other moving freely, and even beyond the middle line, in phonation.	Total unilateral palsy.
Voice little changed; cough normal; inspiration difficult and long, with loud stridor.	Both cords near together, and during inspiration not separated, but even drawn nearer together.	Total abductor palsy.
Symptoms inconclusive; little affection of voice or cough.	One cord near the middle line not moving during inspiration, the other normal.	Unilateral abductor palsy.
No voice; perfect cough; no stridor or dyspnœa.	Cords normal in position and moving normally in respiration, but not brought together on an attempt at phonation.	Adductor palsy.

SECTION XXII

VOICE AND SPEECH

THERE are certain alterations in the voice sounds, in the manner of speech, and in the ability to produce or understand written or spoken words or thoughts which are of much importance in diagnosis. The significant symptoms relating to the voice and speech are:

I. **Aphonia and Hoarseness.**—Loss of voice or whispering voice (aphonia), and coarse or harsh quality of the voice (hoarseness or dysphonia), are both due to some interference with the function of the vocal cords. The imperfect or abolished action of the cords may be due to local and inflammatory disease of the larynx, or disease of or pressure upon the recurrent laryngeal nerve. Aphonia may come suddenly or be preceded by hoarseness, or the latter alone may be present. The cough and respiration may be modified in sound by the existence of hoarseness or aphonia.

Laryngeal Causes of Aphonia.—In the majority of cases aphonia and hoarseness are due to some form of laryngitis, acute or chronic, simple or specific, as in the ordinary catarrhal variety and that due to measles, variola, diphtheria, syphilis, or tuberculosis. Œdema of the glottis and retropharyngeal abscess are fortunately rare causes. Excessive use of the voice, by inducing congestion of the cords, is a common cause of hoarseness or aphonia. Tumour of the larynx, impacted foreign bodies, and cicatricial stenosis of the larynx may be found responsible. The laryngeal muscles may be involved, and thereby stiffened, in trichiniasis.

Aphonia from Involvement of the Laryngeal Nerves.—The larynx receives its nervous supply from the superior laryngeal branch of the pneumogastric and the inferior or recurrent branch of the same nerve. These nerves may be involved in consequence of lesions affecting their nuclei, or in their course by inflammation (neuritis) or by pressure. Interference with their functions gives rise to anæsthesia of the larynx or paralysis of the laryngeal muscles, manifested by aphonia, hoarseness, and, if the abductors are affected, dyspnœa. An examination of the action of the vocal cords during respiration and phonation (page 256) is required to determine which muscles are affected.

If there is a deep, hoarse voice and a brassy cough, with a tendency for particles of food to enter the larynx, there is probably interference with the superior laryngeal nerve, giving rise to anæsthesia of the upper portion of the larynx and paralysis of the *crico-thyroid* muscles. This interference may be due to bulbar paralysis, diphtheritic neuritis, or to the pressure of a goitre or carotid aneurism.

If there is aphonia without cough or dyspnœa, it may be due to paralysis of *all the laryngeal muscles* caused by bulbar paralysis, tumours of the medulla, diphtheria, or pressure upon both recurrent nerves; or to complete paralysis of the *adductors*, which is usually of hysterical origin, but may occur from overuse of the voice, or laryngitis.

Hoarseness, with easy fatigue upon slight use of the voice, is indicative of *unilateral abductor* paralysis, of which the most common cause is an aneurism of the thoracic aorta, less frequently a mediastinal tumour pressing upon one recurrent nerve. If right-sided, it may be due to a much thickened pleura. Bronchocele, cancer of the upper part of the esophagus, and innominate or subclavian aneurism are other possible causes.

A weak, rough, low-pitched voice may be noted as a result of *total unilateral* paralysis due to involvement of one recurrent nerve by the same causes as in the preceding variety.

It should be remembered that normal voice and phonation may coexist with a dangerous form of laryngeal paralysis—*bilateral palsy of the abductors*—but in this case there is inspiratory dyspnœa with loud stridor. It may be due to laryngitis, rarely to hysteria, and is a symptom of locomotor ataxia, bulbar palsy and pressure from aneurism or tumour.

A curious rattling, rough voice (the so-called ventricular voice) is due to the occasional vicarious vibration of the false vocal cords or ventricular bands, in cases where the true cords are paralyzed or destroyed by ulceration. Other peculiar abnormalities of the voice consist in a double or triple splitting of the sounds. In the first variety the two sounds, although simultaneous, differ in pitch (diplophonia, diphthongia); it is due to a small tumour on the edge of one vocal cord, or to unilateral paralysis of the cords. The triple voice is a very uncommon finding, and is caused by a pedunculated tumour, which has its attachment below the vocal cords. During the early portion of phonatory expiration (which is clear) the growth rises, during the middle part (which is hoarse) it lies between the cords, and during the latter part (which is again clear) the tumour has been protruded to a point above the glottis.

II. **Nasal Voice.**—The peculiar quality of voice which goes by this name is sometimes a matter of habit. It is of service to recognise two varieties of the nasal voice. The first, the *open nasal*, is caused by non-closure of the naso-pharyngeal opening by the soft palate (e. g., diphtheritic paralysis), and may be imitated by speaking without opening the mouth; the second, the closed or *stopped nasal*, is due to nasal stenosis, resembling the tone imparted to the voice by speaking while the nose is pinched between the thumb and forefinger.

The open nasal tone is indicative of paralysis of the soft palate, destruction of the soft palate by ulceration, usually syphilitic, or a congenital cleft of the palate. The closed nasal voice, when present, is often suggestive of coryza, hay asthma, hypertrophic rhinitis, nasal polypus, or postnasal adenoids. It is present also with enlarged faucial tonsils, suppurative tonsilitis, acute pharyngitis, and retropharyngeal abscess. A slight nasal intonation may be noted in the early stage of scarlet fever, pharyngeal diphtheria, variola, and typhus fever.

III. **Alterations in the Manner of Speech.**—(1) Dumbness or mutism—inability or unwillingness to speak—may be present in hysteria, idiocy, melancholia, and dementia. Feigned inability to speak is sometimes resorted to by malingerers, and a real inability, due to cerebral fatigue, may succeed a severe and exhausting illness, particularly typhoid fever. Great swelling of the tongue may absolutely prevent articulation. Mutism exists in those who are congenitally deaf, or in children who become totally deaf before the power of speech is permanently acquired. Furthermore, the power of speech is almost entirely lost in some forms of aphasia.

(2) Anarthria, indistinct or imperfect speech, the impairment varying in extent, may be an evidence of paralysis of the tongue, soft palate, and facial muscles, paralysis of the latter affecting particularly the labials *p*, *b*, *f*, *m*, *v*, *w*, and the vowels *o* and *u*. Another variety of anarthria, a difficulty in pronouncing the dentilinguals *d*, *t*, *th*, *s*, *z*, *n*, *l*, and *r*, with an indistinct mumbling speech, is suggestive of bulbar or pseudo-bulbar paralysis, and may be noted more rarely in amyotrophic lateral sclerosis. If the lips are involved, the labials also are affected and speech becomes almost impossible. The speech may be very indistinct as well as weak and whispering in the typhoid status and conditions of exhaustion. Glossitis, parotitis, and the absence of teeth may be responsible for a mumbling manner of talking.

(3) A slow, interrupted manner of speech, the words being slurred over, somewhat as if the patient were intoxicated, with tremulousness of the tongue and lips, is observed in general paresis.

(4) A piping, querulous voice, with manifest hesitation in beginning a sentence, the words then being rapidly spoken, is quite characteristic of paralysis agitans.

(5) Scanning speech, in which the words are spoken slowly, each syllable accented as if reading verse, is an important symptom of disseminated (insular) sclerosis, but is also found in Friedreich's (hereditary) ataxia.

(6) Hesitant or embarrassed speech, amounting in severe cases to a confused and incoherent verbal tangle, may be due to chorea.

(7) Interrupted speech, two or three words at a time being gaspingly uttered, is observed in those who are suffering from marked dyspnœa as well as during the incidence of a chill or rigour. It is to some extent an index of the severity of these symptoms.

IV. **Aphasia.**—The term aphasia embraces a variety of defects in the use or the comprehension of language, either spoken or written. In order to understand and interpret this symptom and its several forms one must first have a conception of the normal manner and mechanism of the faculty of language. The subject is in reality extremely complex, and exceptional cases are accumulating year by year. So far as possible the statements made here are those the truth of which is either admitted or considered as probable by good authority.

NORMAL SPEECH MECHANISM.—The normal exercise of the faculty of language depends upon the existence and integrity of certain *cerebral* centres: (1) Psychical centres of intelligent perception, (2) sensory receptive centres, (3) emissive or motor centres, (4) the association tracts. Finally, there are the basal ganglia for the special senses and the nuclei in the medulla and spinal cord actuating the muscles employed in articulate speech, which constitute respectively the sensory and motor *peripheral* apparatus.

(1) **The Psychical Centres.**—An intelligent perception of an object, whatever the latter may be, depends upon the evidence of its character derived from the senses of sight, sound, taste, smell, touch, and muscular sense. The memories—i. e., the retention and recall of these various impressions—constitute the *mental image* of the object. The mental images thus formed are stored up in either the right or left hemisphere, although the exact localization of these centres in the hemisphere has not yet been determined.

If, in addition to the evidence thus obtained, the name of the object has been heard and spoken, read and written, further memories of the sound of its name, the muscular actions in speaking its name, the appearance of the name as written or printed, and the muscular actions in writing it, constituting the *word image*, are added to the mental image, and a complete and intelligent *conception* of the object is secured.

As thought precedes and may be independent of language, the mental image of an object as it presents itself to the various senses is to be distinguished from the word image which results from education. Words are merely the symbols by which we receive or communicate ideas. In *receiving* ideas through the medium of language one *hears* the spoken word, *sees* the written or printed word, and in some cases sees expressive gestures (e. g., lip reading and sign language of the deaf). In *communicating* ideas by means of language, one *speaks* the word, *writes* the word or makes significant gestures. The faculty of language has therefore a receptive or sensory side, and an emissive or motor side, for both of which special cortical centres have been found to exist in the left hemisphere in right-handed, the right hemisphere in left-handed, individuals. A considerable number of speech centres may ultimately be demonstrated. At the present time the existence of three such centres is proved, while a fourth is accepted by some and denied by others.

(2) **Sensory or Receptive Speech Centres.**—Two sensory cortical speech centres have been accurately localized, of which one is for the perception and memory of the *sound* of spoken words. It is situated in the posterior part of the first and corresponding upper portion of the second temporal convolutions (*C*, Fig. 61); while the other, for the memory of the *visual appearance* of written or printed words, lies in the angular and inferior parietal convolutions (*D*).

(3) **Motor or Emissive Speech Centres.**—In conveying our thoughts to others we speak or write. Just as in learning the sounds or appearance of words constant repetition stores the sensory speech centres with auditory or visual memories, so there are centres which by steady practice become familiar with the complicated series of muscular actions involved in uttering or writing words. The centre for memories of articulate or motor speech is in the posterior portion of the third frontal convolution (Broca's, *B*). The existence of a special centre for the memories of writing movements is not definitely proved, but is considered probable by good authority. It is assigned to the posterior portion of the second frontal convolution (*A*).

(4) **Association Tracts.**—The various motor and sensory speech centres are believed to be connected by association tracts or fibres with each other and with the hypothetical psychical and ideation centres. These in turn are put in communication with the special-sense basal ganglia and the motor nuclei in the medulla and spinal cord by means of the projection fibres. The fibres which unite the speech centres are also called *transcortical*, those passing to and from the lower ganglia and nuclei, *subcortical* fibres. It is conjectured with some show of probability that the island of Reil (insula) may contain a centre of ideation to which converge fibres from the several speech centres. Fig. 64 shows the various association tracts between the speech centres.

In the use of language the motor, auditory, and visual memories are interdependent. The word image as a whole is made up of the memory of its sound as heard, its appearance as written, and the effort made in uttering or writing it, and it is by means of the association fibres that these various elements are harmonized and correlated in hearing, reading, and speaking.

A break in the nervous mechanism described produces various disturbances in the language faculty, the nature and severity of such

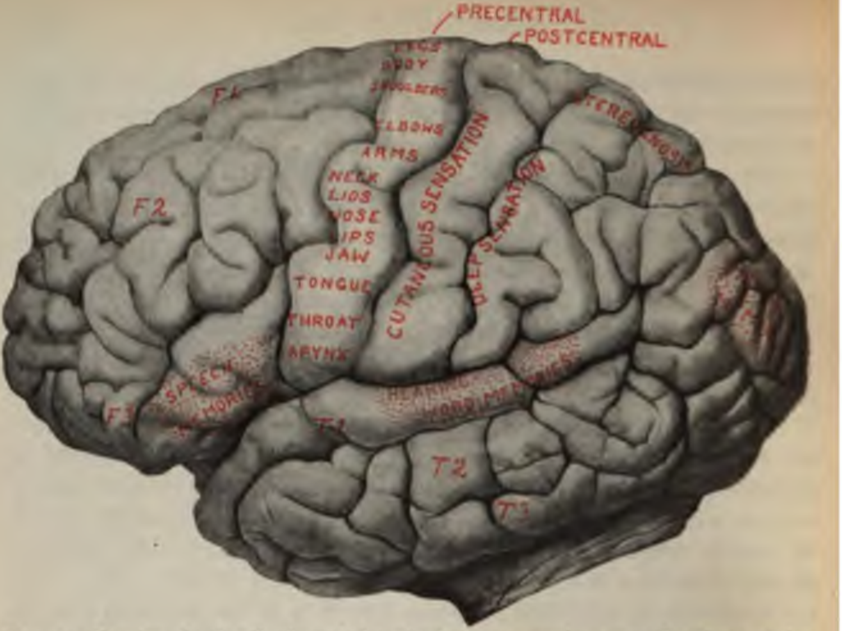


FIG. 61.—Showing the localization of the principal motor and sensory functions of the brain upon the outer surface of the left hemisphere. Red lettering = motor; black lettering = sensory. Compare with Figs. 62 and 63. Brain itself redrawn after a photograph from Dana.

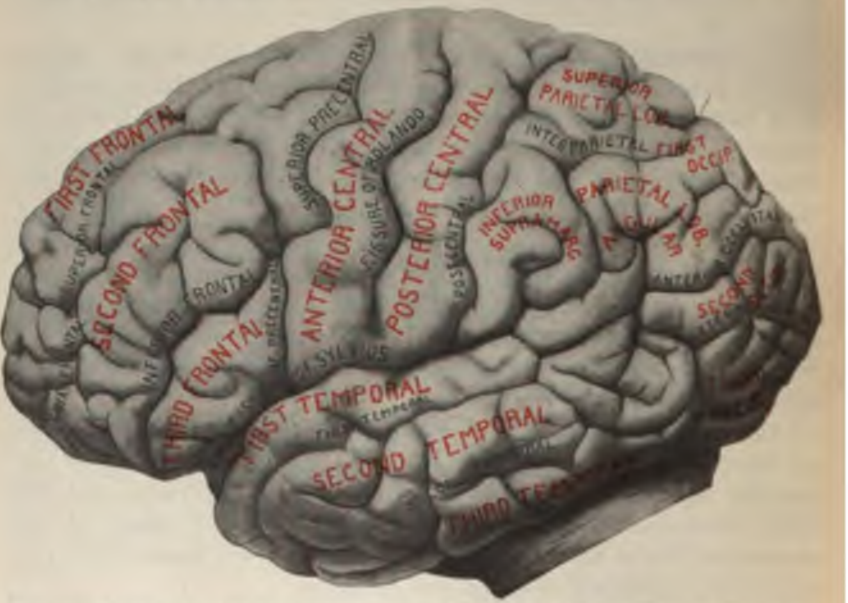


FIG. 62.—Showing the principal convolutions and fissures of the outer hemisphere of the brain. Compare with Figs. 61 and 63. Red lettering = convolutions; black lettering = fissures. Same brain as in Fig. 61 above.

disturbances depending upon the locality and extent of the causal lesion. In order to produce symptoms referable to but one of the centres a small and strictly delimited lesion must exist. More extensive and diffuse lesions of the same centre will affect the combined actions of the other centres with a corresponding increase in the variety of speech disorders.

THE SYMPTOMATOLOGY OF THE LANGUAGE FACULTY.—(1) **Apraxia.**—If an individual has to a greater or less extent lost the power of recognising or understanding the nature and uses of objects or, possibly, the identity of persons, the condition is termed *apraxia*. The varieties of apraxia correspond in number and character to the varieties of sensation. An object, a pencil, for example, may be seen and even handled, but evidently without any conception of its use—*mind blindness* (visual amnesia). A sound—e. g., the ringing of a telephone bell—may be heard, but it awakens no thought of answering it—*mind deafness* (auditory amnesia). The odour of gas in a room does not suggest a leaky burner—*mind anosmia*. A taste is not recognised as belonging to a certain edible—*mind ageusia*. The usually characteristic feel of a fabric gives no clew to its composition—*mind atactilia*.

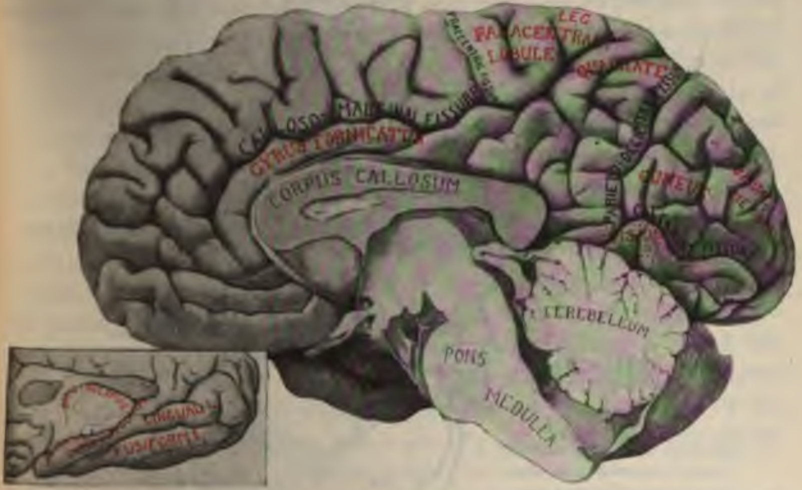


FIG. 63.—Showing the functions, convolutions, and fissures of the median aspect of the right hemisphere. Compare with Figs. 61 and 62. Red lettering = functions and convolutions; black lettering = fissures. Brain redrawn from Dana.

Apraxia is closely allied to, and is usually although not invariably found in connection with, the various forms of *aphasia*—i. e., an inability to produce or understand spoken or written speech. Indeed, one form of aphasia, word blindness or alexia, may be co ered to be a limited mind blindness. So also word deafness m classed under mind deafness.

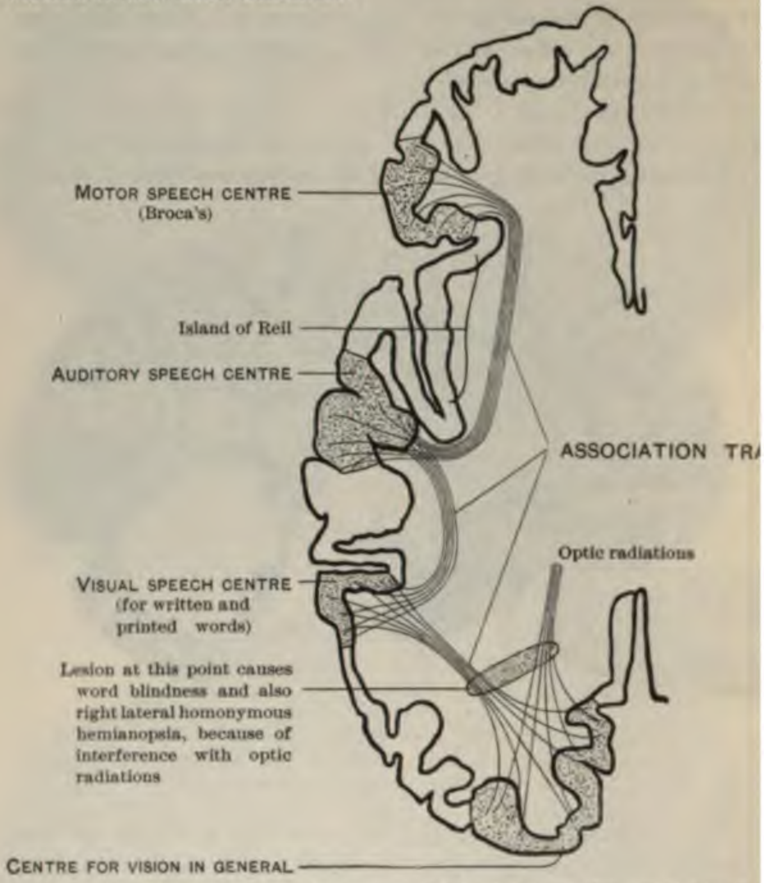

MOTOR SPEECH CENTRE
(Broca's)
Island of Reil
AUDITORY SPEECH CENTRE
ASSOCIATION TR
Optic radiations
VISUAL SPEECH CENTRE
(for written and
printed words)
Lesion at this point causes word blindness and also right lateral homonymous hemianopsia, because of interference with optic radiations
CENTRE FOR VISION IN GENERAL

FIG. 64.—Showing the association tracts of the left hemisphere of the brain.

(2) **Aphasia and its Varieties.**—The principal varieties of aph are:

Motor aphasia...... { Aphemia.
Agraphia.
Sensory aphasia. ... { Visual.
Auditory.
Conduction aphasia.

As aphasia implies a loss of power to produce or to unders spoken or written speech, it is evident that we may have a *m aphasia*, inability to express thought; or *sensory aphasia*, inabilit perceive and interpret thought which has been clothed in language or other means of expression. It is necessary to discriminate aphasia from *anarthria*. The latter (page 263) is due to a partial or complete paralysis of the muscles of articulation arising from disease of the peripheral nerves (or their nuclei) which innervate these muscles, their cortical centres being intact. On the other hand, in aphasia the centres in the cortex are involved, while the peripheral muscular and nervous articulating mechanism is unimpaired.

(*a*) *Motor or Ataxic Aphasia*.—This embraces as its most important division *aphemia*, the loss of power to utter words, although the patient knows what he desires to say, because he can not revive the motor memories for articulating them; and *agraphia*, inability to write words for lack of power to recall the motor memories of the muscular actions involved in writing. Subsidiary motor defects of expression are: Motor *amimia*, loss of ability to convey ideas by gestures; *motor amusia*, loss of power of musical expression—e. g., singing; *musical agraphia*, loss of the ability to write music.

(*b*) *Sensory Aphasia*.—The principal varieties of sensory aphasia are the auditory and visual. *Auditory* aphasia or word deafness is, as its name indicates, an inability to recognise words when spoken. The patient *hears*, but does not understand, as if listening to a foreign language. *Visual* aphasia or alexia is a condition in which the patient *sees*, but does not recognise or understand written or printed words. Subsidiary sensory defects are: *sensory amimia*, loss of the ability to understand gestures; and *sensory amusia*, loss of the ability to recognise musical sounds when heard.

Two other symptoms of some importance are: *paraphasia*, in which the subject employs the wrong words, repeats words, or talks with an unintelligible confusion of language; and *paragraphia*, a similar mixture or confounding of words on attempting to express himself in writing.

(3) **Examination for Apraxia and Aphasia.**—With reference to the examination of patients for the purpose of determining the presence of these symptoms some very elaborate schedules have been devised (Eskridge, Starr), better fitted for the specialist in neurology than for the practitioner of inner medicine. For the latter, Elder's 12 questions probably embrace all that is required in the great majority of cases, and they are here given, with some additions and explanatory comment.

To Determine the Presence of Word Deafness (*Auditory Aphasia*)

1. Can he hear sounds? This determines the presence or absence of ordinary deafness.

2. Can he hear spoken words? If so, then

3. Can he understand the words spoken? This may be ascertained by asking him to put out his tongue, to open and close his hand or take up some article. If he does not comply with these requests, and evidently can not attach any meaning to them, he has word deafness, the principal symptom of auditory aphasia.

To Determine the Presence of Word Blindness (*Alexia, Visual Aphasia*)

4. Can he see objects? This determines the presence or absence of ordinary vision.

5. Can he see words written or printed and read them silently?

6. Can he understand written or printed words—i. e., can he read intelligently? This may be ascertained by writing out a question—e. g., "How long have you been ill?" or a direction, "Close your eyes." If he fails to respond, there is alexia or word blindness, the principal symptom of visual aphasia.

To Determine the Presence of Motor Aphasia (*Aphemia*)

7. Can he speak voluntarily?

8. Can he repeat words?

9. Can he read aloud? If he is unable to do these things he has motor aphasia or aphemia.

To Determine the Presence of Agraphia

10. Can he write voluntarily?

11. Can he write to dictation?

12. Can he copy? If he is unable to do these things there is agraphia, a symptom which may be found in all forms of aphasia. If he can not write voluntarily because of inability to remember words or their appearance, but can write to dictation, it is sensory agraphia. If he can not write either voluntarily or to dictation it is a motor agraphia.

To these may be added the following:

To Determine the Presence of Paraphasia and Paragraphia

13. When speaking, does he commit errors in the use of words? That is, does he use one word for another, so that the result is more or less unintelligible, amounting in some cases to an empty babble? If so, there is paraphasia.

14. When writing, does he commit errors in the use of words? If the written speech is similar in character to the spoken speech as described under 13, preceding, there is paragraphia.

Paraphasia and paragraphia are the symptoms of conduction aphasia (lesion of the association tracts), but this form of aphasia is usually combined with the auditory or visual form.

To Determine the Presence of Mind Blindness

15. Does he recognise ordinary objects seen and their uses? Put before the patient pen, ink and paper, a hand mirror, a match (any two or three other familiar objects will answer), and request him to write or ignite the match. If he can not pick out the proper article he is the subject of mind blindness, provided that he is not unable to understand the request because of the presence of word deafness.

To Determine the Presence of Mind Deafness

16. Does he recognise ordinary sounds—i. e., does the ring of a bell, the noise of an opening door, the raking of a fire, and similar sounds, awaken a recognition of their import? If not, the patient has mind deafness.

It must be remembered that there are variations in the completeness of all speech defects which have been described. There may be only a partial loss of any one of the normal powers of expression, depending mainly upon the extent of the productive lesion.

Aphasic Symptoms with Reference to Localization of Lesions.—Having determined and noted the departures from the normal use of the language faculty—i. e., the apraxic and aphasic symptoms present—by means of the examination as detailed, it remains to draw an inference as to the seat of the causative lesion. Following are the symptom groups of the aphasias which possess a localizing value:

(1) **Motor Aphasia.**—If the patient *can* read silently, write voluntarily, write to dictation, copy, and hear and understand spoken words, but *can not* speak voluntarily, repeat words, or read aloud (motor aphasia, aphemia), there is a small lesion of Broca's convolution, third frontal (*B*, Fig. 61).

If the patient *can* hear and understand spoken words, read and understand written or printed words, and copy, but *can not* speak voluntarily, repeat words, read aloud, write voluntarily or to dictation (motor aphasia, aphemia, *plus* agraphia), there is a marked lesion of the third frontal convolution. This is the most frequent form of aphasia. In the severest cases there is a lessened power of understanding written or spoken speech.

(2) **Visual Aphasia.**—If the patient *can* speak voluntarily, and understand spoken words, but *can not* understand written or printed words, write voluntarily, write to dictation, or copy (visual aphasia

plus agraphia), there is a lesion involving the cortex of the angular gyrus and supramarginal lobule (*D*, Fig. 61).

If the patient *can not* understand written or printed words o copy, but *can* write, the lesion involves the subcortical substance o the angular gyrus. Visual aphasia is often associated with hem anopia, and some hemiataxia and hemianæsthesia.

(3) **Auditory Aphasia.**—If the patient *can* speak voluntaril read intelligently, and write voluntarily, but *can not* understan spoken words, repeat words, or write to dictation (auditory aphasia there is a small subcortical lesion of the first and second tempor convolutions (*C*).

If the patient *can* speak voluntarily, but *can not* understan spoken words, read intelligently, read aloud, repeat words, write dictation, or copy (auditory aphasia *plus* visual aphasic and agraph symptoms), there is an extensive lesion of the first and second tem poral convolutions.

(4) **Conduction Aphasia.**—If the patient uses the wrong wor or talks jargon (*paraphasia*), and makes similar mistakes in writin (*paragraphia*), *but* can speak voluntarily, understand spoken word read, and write, it is indicative of an interruption of the associatio tracts between the various centres, and the lesion is ordinarily in th island of Reil or the convolutions about the fissure of Sylvius. should be remembered that unmixed conduction aphasia is not all frequent. It generally coexists with auditory or visual aphasia.

(5) **Mind Blindness.**—If the patient can not understand the us and nature of seen objects, there is mind blindness, which is indic tive of a considerable lesion (cortical or subcortical) involving th angular gyrus and supramarginal lobule, essentially the same lesio as that of visual aphasia (word blindness).

THE DISEASES WHICH CAUSE APHASIA.—In the majority cases aphasia is one of the symptoms of organic focal cerebral di ease, occurring in the left hemisphere in the right-handed and vi *versa*. It may be found in cerebral hemorrhage, thrombosis, emb lism, abscess, tumour, or gumma and depressed fracture of the sku The diagnosis between these various lesions must be made from th associated symptoms.

More rarely it has been noted in hysteria and neurasthenia, an has followed an epileptic convulsion. It is an occasional sympto in migraine, and may be present during convalescence from exhaus ing fevers, particularly typhoid. Sudden fright has been considere as causative in certain cases.

SECTION XXIII

COUGH

In the majority of cases cough is a symptom of disease of the larynx, trachea, bronchial tubes, lungs, or pleura. Less frequently it is due to disease of the nasopharynx, and still more infrequently it is a result of hysteria and disease or irritation in organs not forming a part of the respiratory apparatus. If not caused by disease of the respiratory apparatus, it is frequently spoken of as "reflex" cough, which is a misnomer, cough being always a reflex action. "Transferred" would be a better term, and "extra-respiratory" corresponds more nearly to the facts.

Cough may be considered with reference to the indications from its character, and also with reference to its possible causes.

I. **Diagnostic Indications from the Character of Cough.**—(*a*) *Dry Cough.*—Cough without expectoration or with the occasional expulsion of a small pellet of mucus is caused by the first stage of acute bronchitis, pulmonary phthisis, bronchial asthma, pertussis, epidemic influenza, and acute pneumonia; pleurisy; diseases of the nasopharynx and larynx; inhalation of irritating fumes or dust; elongated uvula or enlarged lingual tonsil; foreign bodies; and "reflex" or extra-respiratory conditions. A single, slight, dry cough, frequently repeated, is the "hacking" cough premonitory of pulmonary phthisis.

(*b*) *Loose Cough.*—Cough with expectoration occurs in the later stages of acute bronchitis, pertussis, pneumonia, pulmonary phthisis, bronchial asthma, bronchiectasis, and pulmonary gangrene.

(*c*) *Paroxysmal Cough.*—Cough coming in fits or paroxysms is most characteristically seen in pertussis. It also occurs in conditions attended with increased secretion, continuing until the bronchial tubes are cleared, as in the second stage of acute bronchitis and the softening stage of pulmonary phthisis. A paroxysmal cough with a considerable interval between the seizures may be due to abscess of the lung, bronchiectasis, and phthisical or gangrenous cavities. Under such circumstances large quantities are expectorated in a short time, the cough ceasing when the cavity is emptied and recurring when it refills. A somewhat significant fact in these cases is that the coughing fit is precipitated by change of position. The inference is that a cavity exists, the contents of which are permitted by the changed posture to flow into the bronchial tubes and thus initiate the paroxysm. Enlargement of the bronchial glands, mediastinal tumour, and other extra-respiratory lesions will produce a paroxysmal but dry cough. A coughing fit terminating in vomiting is of some importance as a symptom in pertussis and pulmonary phthisis.

(*d*) *Laryngeal Cough.*—A dry cough, variously described as "croupy," hoarse, ringing, brassy, or metallic in character, is that caused by laryngeal irritation, either direct or transferred. A certain amount of clinical experience is necessary to enable one to appreciate the fine shades of difference between the designated qualities; but the distinctions are real, although elusive in description.

The conditions which may be indicated by a laryngeal cough are spasm of the larynx, laryngitis, tuberculous or syphilitic ulceration of the larynx, inhalation of dust particles in certain occupations, impacted foreign body in larynx, food particles entering the larynx in pharyngeal paralysis, elongated uvula, or enlarged tonsils. The monotonous, croaking, nervous cough of hysteria and the barking cough of puberty are laryngeal in character. A brassy, metallic cough arises from irritation of the recurrent laryngeal branch of the pneumogastric by the pressure of thoracic aneurism, cancer of the esophagus, enlarged bronchial glands, and mediastinal tumour.

(*e*) *Suppressed Cough.*—A voluntary effort to suppress a cough is usually a sign that the act of coughing is particularly painful or exhausting, as in pleural inflammations (especially diaphragmatic pleurisy), pneumonia, pleurodynia, acute peritonitis, and abdominal rheumatism. A child with developed pertussis will vainly endeavour to restrain the paroxysm because of the discomfort experienced. Patients with acute coryza and bronchitis often complain that coughing is painful, either because of substernal soreness in the early stages, or intensification of pain in the inflamed accessory nasal sinuses during the latter part of the disease.

(*f*) *Inability to Cough.*—If the diaphragm is paralyzed, either from disease of the nervous system or because of overstretching by ascites or abdominal growths, cough becomes difficult or impossible. Sinking in of the epigastrium during inspiration is, in the absence of laryngeal obstruction, indicative of diaphragmatic paralysis.

An oncoming inability to cough, if it occurs in the later stages of pulmonary disease attended with profuse secretion, is of bad omen, especially if the retained material can be heard rattling in the tubes. It is indicative of extreme prostration, and is found with pulmonary phthisis, lobar and broncho-pneumonia, chronic bronchitis, and pulmonary œdema.

(*g*) *Winter Cough.*—A cough which disappears in the summer and returns with the advent of cold weather is usually due to chronic bronchitis, but may be significant of a very chronic form of pulmonary phthisis.

II. **Summary of the Causes of Cough.**—From the diagnostic point of view the causes of cough may be divided into two classes: (*a*) Direct or respiratory causes, embracing the diseases of the larynx, bronchi, lungs, and pleura, to which the majority of coughs will be found due; and (*b*) indirect, reflex, transferred or non-respiratory causes, including all others. In general, although with some exceptions, non-respiratory coughs are persistent, spasmodic, dry, afebrile, without pulmonary physical signs and without impairment of the general health.

(*a*) **Direct Causes.**—It is unnecessary to recapitulate here the diseases (including new growths) of the larynx, bronchi, lungs, and pleura, all of which must be excluded by means of the history and a careful physical and laryngoscopic examination before searching elsewhere for a possible cause.

(*b*) **Indirect and Unusual Causes.**—1. *Nasal Cavities.*—The various forms of rhinitis (hypertrophic, atrophic, and vasomotor), spurs and deviations of the septum, nasal polypi, and the irritation of dry crusts may be responsible for cough. In such cases sensitive areas are found by the probe, irritation of which produces cough, and the application of cocaine to the same areas will relieve it.

2. *Pharynx.*—Postnasal adenoid or lymphoid growths and the collection of thick mucus in the same locality, acute or chronic pharyngitis (follicular, atrophic, or hypertrophic), leptothrix, papillomata, elongated uvula, and paralysis of the palate or pharynx may give rise to cough. In children a cough coming on at night during recumbency may be due to the trickling of a free mucous secretion into the larynx from the postnasal space, or to enlarged tonsils or long uvula touching the pharyngeal walls.

3. *Tongue.*—Enlargement of the lingual tonsil, which lies in the space between the root of the tongue and the epiglottis (the glosso-epiglottic fossa), is the cause of a number of reported cases of obstinate cough, the cough disappearing when the swelling was reduced.

4. *Ear.*—By the auriculo-temporal branch of the fifth nerve an irritation may be conveyed which will cause cough, as in that which results from probing or syringing of the ear, or from the presence of impacted wax or foreign bodies.

5. *Miscellaneous Causes.*—Cough may signify pressure effects from enlarged bronchial glands, mediastinal tumour or abscess, thoracic aneurism, or caries of the dorsal vertebræ. It is a not infrequent symptom of cardiac disease, such as dilatation, hypertrophy, and valvular lesions. "Stomach cough" is found at times in the subjects of chronic gastric catarrh, due most probably to the low grade of accompanying pharyngitis.

"Liver cough" may occur as a symptom of enlarged liver in consequence of pressure against the diaphragm, or in connection with hepatic abscess, hydatids, gallstones, perihepatitis, and subphrenic abscess, presumably from irritation or involvement of the diaphragmatic pleura. In a case of multiple hepatic abscesses observed by me, and which came to autopsy, each chill heralding the formation of an abscess was accompanied by a violent paroxysm of cough. An enlarged or inflamed spleen may for similar reasons give rise to cough.

Habitual cough may be incident to occupations which involve the inhalation of dust or irritating fumes, and is not infrequent in excessive tobacco smokers. The loud "barking" cough of puberty occurs mainly in boys of neurotic family affiliations (CLARK). Dentition in infants and an inflamed tooth in older persons will, it is said, produce sufficient transferred irritation to cause a cough. It has been stated that the cough of hysteria sounds as if it was intended to attract attention. Aspiration of a pleural exudate will often cause a somewhat violent fit of coughing. Finally, cough has been attributed, perhaps upon inadequate evidence, to irritation from disease of the uterus, ovaries, mammary glands, or testicles.

SECTION XXIV

SPUTUM (INCLUDING HÆMOPTYSIS) AND ITS GROSS CHARACTERS

THE naked-eye examination of the sputum is made with reference to its quantity, consistence and apparent composition, colour, and odour. The microscopic examination, which with reference to parasitic diseases is more important than the macroscopic, is dealt with elsewhere (Index—Sputum, microscopic examination of).

Scanty or Absent Sputum.—A small amount of sputum is expectorated in the first stage of acute bronchitis and asthma, and in laryngitis and pleurisy. In children under the age of seven years the sputum is usually swallowed and does not appear.

Abundant Sputum.—Almost the only important diagnostic evidence from the quantity of sputum relates to the cases in which a large amount, particularly if it be purulent, is expectorated in a brief time. In such cases a suspicion may be entertained of the existence of phthisical, gangrenous, or actinomycotic cavities, or a bronchiectasis; or the rupture into a bronchus of an empyema, abscess of the lung, liver, or kidney, or a subphrenic abscess.

Watery or serous sputum, which may be blood-stained, occurs in pulmonary œdema and the catarrhal form of influenza. A thin and watery expectoration, partly regurgitated and partly hawked up, sometimes in considerable quantity, is an occasional symptom in the gastric disorders of neurotic elderly people.

Viscid Sputum.—A thick, viscid, gelatinous sputum, adhering to the container even if the latter is inverted, is somewhat characteristic of lobar pneumonia, but may occur also in phthisis, pertussis, and broncho-pneumonia.

Mucous Sputum.—A diffluent, clear sputum, resembling egg albumen and composed mainly of mucus, is observed in the early stages of pneumonia, bronchitis, and phthisis pulmonalis, at the termination of an asthmatic attack, in developed pertussis, and pharyngitis, laryngitis, measles, influenza, and emphysema.

Muco-purulent Sputum.—A mixture of opaque yellowish streaks of pus with mucus constitutes this variety of sputum. It is noted toward the end of measles and pertussis, in resolving pneumonia, phthisis, and subacute or chronic bronchitis.

Purulent Sputum.—Sputum composed purely of pus is of infrequent occurrence, and when found is indicative of the rupture into a bronchus of an abscess of the lung, liver, or kidney, or a subphrenic abscess, or purulent pleurisy. An opaque yellow sputum, consisting very largely of pus, is found with bronchiectasis, phthisical cavities, broncho-pneumonia, and in chronic or the later stages of acute bronchitis.

Frothy Sputum.—Sputum containing numerous small air bubbles may be noted in bronchitis, broncho-pneumonia, and emphysema, but its most important association is with pulmonary œdema, in which case it resembles water made frothy with soap.

Nummular Sputa.—If upon being deposited in water certain portions of the sputum sink to the bottom, because airless, and assume a button-shaped or coinlike form, it is said to indicate chronic bronchitis, or bronchiectatic or phthisical cavities.

Rusty Sputum.—A viscid, rusty sputum is generally indicative of a lobar pneumonia, but it may also be found in acute tuberculo-pneumonic phthisis, and in some forms of pyæmia.

Prune-juice Sputum.—Expectoration of material resembling prune juice, the colour being due to altered blood, is witnessed late in the adynamic and septic forms of lobar pneumonia, and in gangrene or cancer of the lung.

Currant-jelly Sputum.—Sputum of this character is found in cancer of the lung, and has been reported as one of the manifestations of hysteria.

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Black Sputum.—Sputum of a black or black-speckled appearance is found in persons who have inhaled smoke or coal dust for long periods, and is sometimes present in gangrene of the lung.

Yellow or Green Sputum.—This may be caused by an abscess of the liver which has ruptured into a bronchus (bile pigment), or in some cases of pneumonia (altered hæmoglobin).

Anchovy Sauce.—A dark, brownish sputum is indicative of a ruptured amœbic abscess of the liver, and the parasites may be found by a microscopic examination.

Fetid Sputum.—An offensive, bad-smelling expectoration usually means bronchiectases, gangrene of the lung, phthisical cavities containing decomposed material, pulmonary actinomycosis, or an empyema or subphrenic abscess discharging into a bronchus.

Sputum containing shreds or casts may be noted in plastic bronchitis, diphtheria (especially of the larynx), and rarely in pneumonia. Casts, unless large and branching, are more apt to be found during the microscopic examination. Suspicious masses should be floated in water and unravelled with needles.

Blood-streaked Sputum.—Sputum more or less streaked with blood may be due to the arts of the malingerer (wounding gums or cheeks), to violent vomiting or coughing, spongy gums, eroded tonsils, and slow leakage from an aortic aneurism through a small opening in a bronchus. It may be present as a sequel of hæmoptysis and in cancer or abscess of the lung, acute broncho-pneumonia, emphysema, and plastic bronchitis. If the blood is dark it may be due to pulmonary infarcts. Most commonly it is found, lasting for days or weeks at a time, in the middle stage of pulmonary phthisis.

Hæmoptysis.—In order to constitute hæmoptysis an appreciable amount of pure or nearly pure blood must be spat out, not merely sputum streaked or stained with blood. The amount may be small and continue for several days or, as with the rupture of an aneurism, sufficiently large to cause death in a short time.

Large and rapidly fatal hæmoptysis, fortunately a rare occurrence, is almost invariably due either to a ruptured aneurism or to the erosion of a vessel of considerable size in a large phthisical cavity. Lesser hemorrhages are caused for the most part, in the order of frequency, by pulmonary tuberculosis, disease of the heart, and disease of the blood vessels.

There may be premonitory symptoms, such as dyspnœa, cough, and substernal soreness or oppression, but in many cases it occurs without warning. A tickling sensation is felt in the larynx, and, with a slight cough, warm, salty blood fills the mouth. After the hemorrhage has ceased, sputum, blood-stained or containing small dark clots, may be expectorated for several days. If the amount of blood is small, the immediate symptoms are simply those due to the mental shock and perturbation which usually attend the attack; if large, the evidences of acute internal hemorrhage (*q. v.*) are present.

It is necessary to exclude the nose, pharynx, larynx, and buccal cavity as the sources of the blood by a careful examination for varicosities, erosions, and ulcerations, as well as spongy, bleeding gums. Hæmoptysis has been caused by bleeding varicose veins of the lingual tonsil (KINNICUTT).

The gastric origin of the hemorrhage may be eliminated by noting that the blood is coughed up, is bright red, frothy, and alkaline. On auscultation of the chest bubbling râles may be detected, and the sputa are tinged with blood, while subsequently tarry stools are exceptional. The patient is usually able to state whether the blood is coughed up or vomited.

The *causes of hæmoptysis* are as follows:

(1) *Pulmonary Disease.*—In the majority of cases hæmoptysis is due to pulmonary tuberculosis. It may be the first obvious symptom, in which case it comes from a congested area of the bronchial mucous membrane, and the amount lost may be very considerable, but practically never lethal. In cases which subsequently develop unmistakable signs of the disease there can be no doubt that tubercles were present at the time of the hemorrhage. If the hæmoptysis comes on at an advanced period, especially if cavities exist, it may be due to the erosion of a vessel of considerable size, and prove fatal. Small recurring pulmonary bleedings may be present for considerable periods in cases of quiescent or arrested tuberculosis. Small hæmoptyses may occur in the initial stage of lobar pneumonia: and either small or large in bronchiectasis and abscess, gangrene, or cancer of the lung. It not infrequently attends the expulsion of the casts in plastic (fibrinous) bronchitis, or may be a preliminary symptom of the attack. It has also occurred with emphysema.

(2) *Cardiac Disease.*—A not uncommon cause of hæmoptysis, usually small and recurring, is pulmonary venous obstruction arising from valvular disease of the heart. As a rule it occurs late in the course of the disease, except with mitral stenosis, in which it may be an early symptom. It is due to the rupture of small veins or leakage from an engorged mucous membrane. It may be due to cardiac hypertrophy, especially that associated with chronic nephritis and arteriosclerosis involving the branches of the pulmonary artery. Small hæmoptyses may also be caused by pulmonary infarction occurring with valvular disease and arising from embolism or thrombosis. If the infarcted area is large and in the usual site, consolidation and

bronchial breathing may be found in one of the lower lobes. If the embolism is septic, abscess or gangrene may follow.

(3) *Vascular Disease.*—A large and almost immediately fatal hæmoptysis may be due to the rupture of a thoracic or innominate aneurism. On the other hand, slight, continued spitting of blood may be the result of a small opening from an aneurism, or the oozing of blood through the fibrinous layers of the aneurismal sac into a bronchus. Hæmoptysis also occurs in gouty persons over fifty years of age as a result of endarteritis affecting the branches of the pulmonary artery (CLARK).

(4) *Diseases and Conditions of the Blood.*—The diseases of the blood which are liable to cause hemorrhages elsewhere may also be responsible for bleeding from the lungs—i. e., hæmophilia, purpura, scurvy, leucæmia, and the severe anæmias, although this is rarely the case with the last. Cholæmia (jaundice) has been known to produce it. The blood dyscrasiæ of the severe infections may give rise to broncho-pulmonary bleeding, as in typhus fever, typhoid fever, the hemorrhagic type of variola, and the exanthemata in general.

(5) *Miscellaneous Causes.*—There are cases of hæmoptysis occurring in young persons without obvious cause and not followed by pulmonary or other disease. The third leg of diagnosis, i. e., time, must necessarily decide the question as to its innocuous nature in a given case. In hysteria there may be spitting of a bloody fluid of a pale-red colour, not pure blood, which comes in all probability from the mouth or pharynx, and its occurrence may lead to an incorrect diagnosis of phthisis pulmonalis. On microscopical examination cylindrical or ciliated epithelial cells are absent. There are unquestionable cases, although of rare occurrence, in which the menstrual flow is replaced by periodical hæmoptyses—vicarious menstruation. Mediastinal tumours by pressure or extension may have blood-spitting as a symptom. In China and Japan there is an endemic hæmoptysis due to the presence of the distomum in the bronchial tubes.

SECTION XXV

THE NECK

1. **Shape.**—A short, thick neck is traditionally associated with the apoplectic habit. A long, scrawny neck, with a projecting larynx, frequently coexists with the phthisinoid chest, and is not rarely significant of the tuberculous diathesis.

2. **Rigid Neck.**—Rigidity of the neck, slight or marked, may be due to disease of the cervical vertebræ, as in caries or rheumatoid arthritis affecting the cervical vertebræ, and to rheumatism of the cervical muscles. Inflammatory disease of the throat accompanied by painful and swollen cervical glands may interfere with the free movement of the neck. Boils or carbuncles (perhaps diabetic) are other causes of inability or disinclination to move the neck. (See also Head, abnormal fixity of.)

3. **Prominence of Sterno-mastoids.**—If both sterno-mastoids stand out in unusually bold relief, search should be made for some cause of frequent or long-continued dyspnœa, such as bronchial asthma, emphysema, chronic bronchitis, or cardiac disease. If one muscle is prominent, it may be due to tonic wryneck, congenital thickening or tumour of the muscle, a cyst, or a tuberculous abscess.

4. **Clavicle.**—Localized enlargements upon the clavicle, resembling the callus resulting from fracture, if occurring in adults in the absence of trauma, are probably syphilitic nodes.

Swelling or tumefaction above both clavicles is most commonly due to emphysema, the enlarged apex rising abnormally high, and to myxœdema, or mediastinal tumours; above left clavicle to pressure of aneurism on left innominate vein. An inflammatory, brawny swelling may indicate perforation of the esophagus (malignant disease or ulceration).

5. **Thyroid Gland.**—*Enlargement.*—A tumour in close relation with the trachea, and moving with it during the act of swallowing, is an enlarged thyroid gland. The enlargement is usually greater on one side. The presence or absence of induration, fluctuation, pulsation, or systolic thrill (and murmur) must be determined.

If fluctuation is found, it is in all probability a fibrocyst (simple goitre), or very rarely an abscess of the gland. If pulsation, thrill, and murmur exist and the enlargement is unequal and varying, the associated symptoms usually will be found to declare it a case of exophthalmic goitre (*q. v.*). If the pulsating enlargement is on the right side, the possibility of its being either an innominate aneurism or a dynamic (neurotic) pulsation should be remembered. A solid, hard enlargement is usually a simple goitre, but may be due to adenoma, cancer, tuberculosis, or gumma of the gland.

The thyroid sometimes enlarges during menstruation, returning to the normal size during the interval. Tumours or aneurism in the root of the neck and behind the sternum may cause thyroid swelling, pressure hindering the return circulation from the gland.

Atrophied Thyroid.—An unusual depression in the location of the thyroid gland, owing to its atrophy, is found in myxœdema and cretinism (congenital or sporadic).

6. **Trachea and Larynx.**—*Inspiratory descent* of the larynx is seen in all conditions which give rise to laryngeal stenosis and in extensive pulmonary collapse or consolidation, bronchial asthma, and laboured breathing in general. Occurring in patients who are critically ill it is usually of bad omen.

Displacement of the trachea to one side or the other may be due to thoracic aneurism, mediastinal tumours, or chronic fibroid phthisis. In the latter case it is drawn to the affected side by the shrinking lung. Aneurism of the innominate pushes it to the left.

Tracheal tugging is considered a valuable (not pathognomonic) sign in suspected thoracic aneurism. It depends upon the fact that the arch of the aorta curves back over the left bronchus, and if an aneurismal dilatation of the arch is present each systolic distention of the vessel presses down upon the bronchus, thereby pulling the trachea downward. To determine its presence, stand behind the patient. Let him close his mouth and raise his chin. The tips of the forefingers are then placed firmly upon either side of the cricoid, pressing upward, when, if a downward tug is present, it will be detected.

7. **Enlarged Lymphatic Glands of Neck.**—If the lymph, as it passes through the lymph glands, contains toxic or infective material, either from simple inflammation, specific bacterial infection, or malignant disease of the parts from which it comes, the glands enlarge. The glands of the neck, because of the manifold varieties of disease which may occur in their tributary regions, are frequently found to be enlarged, especially in children. In the large majority of cases the enlargement is an acute simple inflammation terminating in resolution, sometimes in suppuration. Next most frequent is tuberculous or syphilitic enlargement. Finally, the increase in size may be due to the hyperplasia (lymphoma) of leucæmia and Hodgkin's disease, or to a neoplasm (especially cancer) secondary to a focus elsewhere, or lymphosarcoma.

For diagnostic purposes it is necessary to note the location of the enlarged glands, whether the enlargement is acute or of long standing, their consistence (hard or fluctuating), and if they are well defined or matted together. According to the location of the enlarged nodes various regions should be examined for the presence of a causative disease (see Fig. 65).

(*a*) *The upper deep cervical* glands, behind the ramus and at the angle of the jaw, become acutely swollen and painful to a varying degree in diphtheria, follicular tonsilitis, scarlet fever, measles, rötheln, varicella, roseola and variola, as well as in erysipelas, glanders, pertussis, suppurative tonsilitis, and retropharyngeal abscess.

Chronic enlargement of the deep cervical glands, if they are matted together and tend to suppurate, may be tuberculous; if small and non-suppurating, syphilitic; if large, separate, non-suppurating, and forming a part of similar extensive changes elsewhere, Hodgkin's disease; rarely leucæmic; perhaps a secondary malignant growth, particularly a sequence of gastric cancer and sometimes the only palpable evidence of the existence of the latter. Septic ear disease and carious teeth may also cause some enlargements here.

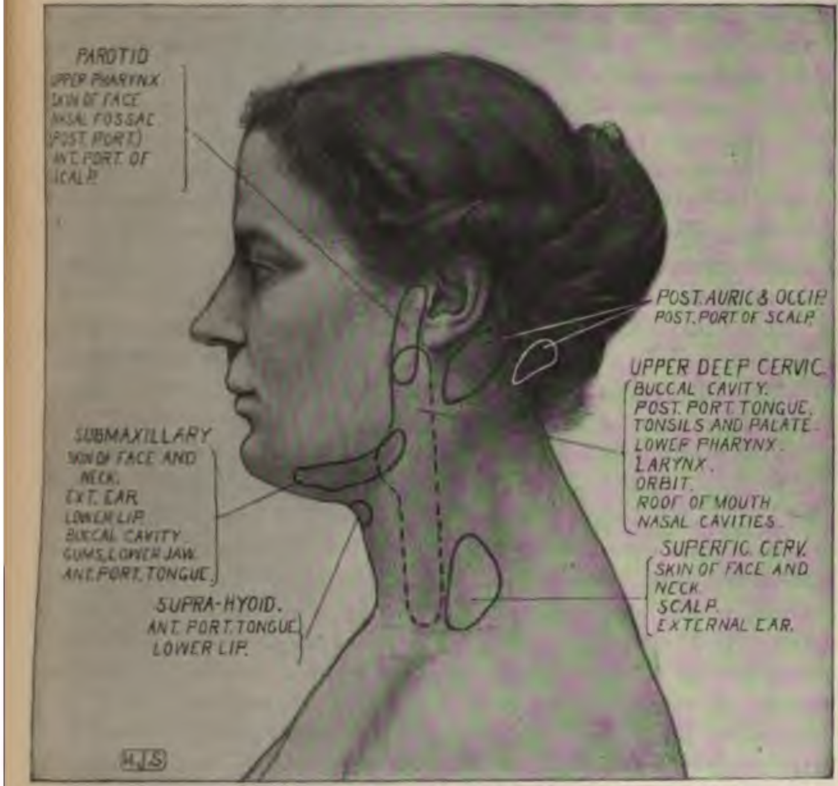

Fig. 65.—Diagram showing the various groups of glands in the head and neck and the regions which should be examined to discover a cause for enlargement of particular groups.

(*b*) The *parotid lymph nodes*, overlying and in the substance of the parotid gland in front of the ear, are enlarged in inflammatory or other disease of the upper pharynx and skin of the face.

(*c*) The *posterior auricular* glands, particularly those lying under the upper extremity of the trapezius (occipital), may become enlarged as a result of German measles, or syphilis, or cutaneous disease of the posterior scalp. The superficial cervical group lying above the clavicle, between the sterno-mastoid and trapezius, becomes swollen in cutaneous disease of the face, neck, and external ear.

(*d*) The *submaxillary group*, and sometimes the *suprahyoid*, are enlarged, because of carious teeth, stomatitis, syphilis, mumps, roseola, diphtheria, and cancer of the lower lip or anterior portion of the tongue.

8. **Condition of the Arteries in the Neck.**—(*a*) *Pulsation*, abnormally visible or violent, of the *carotids* may be due to a variety of causes. It occurs as the result of exertion or excitement. It may be most violent in anæmia and large hemorrhages. It is seen in the atheromatous vessels of elderly people, in aneurism and in the extremely rare cases of obliteration of the descending aorta. It may be a symptom of left ventricular hypertrophy and aortic regurgitation. It is an occasional concomitant of fevers, particularly sunstroke and the early stage of variola. It is not infrequent in the cerebral apoplexies. Finally, it may be a manifestation of exophthalmic goitre or be of neurotic origin, the vessel walls apparently possessing an unnatural elasticity (dynamic pulsation).

In the majority of cases excessive carotid pulsation may be attributed to one of four causes—atheroma, anæmia, aortic regurgitation, or a general neurosis (neurasthenia, hysteria) attended by other evidences of vasomotor instability.

(*b*) *Pulsation in the episternal notch* is sometimes found as a normal condition in old people. Like excessive throbbing in the carotids and abdominal aorta, it is not infrequently of anæmic or neurotic origin, in this case affecting the innominate. More rarely an aneurism of the same vessel may be responsible for it. Provided it is well marked, it may be due to an aneurism of the transverse arch of the aorta. As a rarity it is caused by an abnormal origin of the right subclavian to the left of the median line or an unusually large thyroidea ima artery. The subclavian artery sometimes pulsates with unusual visibility when the lung of the same side is retracted or extensively consolidated.

9. **Condition of the Veins in the Neck.**—The jugular veins are sufficiently large, superficial, and near the heart, to afford particular advantages in determining the condition of the circulation through the right heart. The right jugulars are best fitted for examination.

It is desirable to observe whether the jugular veins are collapsed, distended, or pulsating. If distended, the external jugular can always, and under ordinary circumstances usually, be seen traversing the surface of the sterno-mastoid. Under normal conditions neither the internal jugular nor its bulb—i. e., its junction with the subclavian vein—can be seen. The bulb lies directly behind the lower end of the sterno-mastoid between the sternal and clavicular attachments. If distended, it becomes apparent as a small rounded mass in this locality and, if greatly engorged, rises half an inch above the upper border of the sterno-clavicular articulation. Venous swelling or pulsation is most marked when the patient is recumbent.

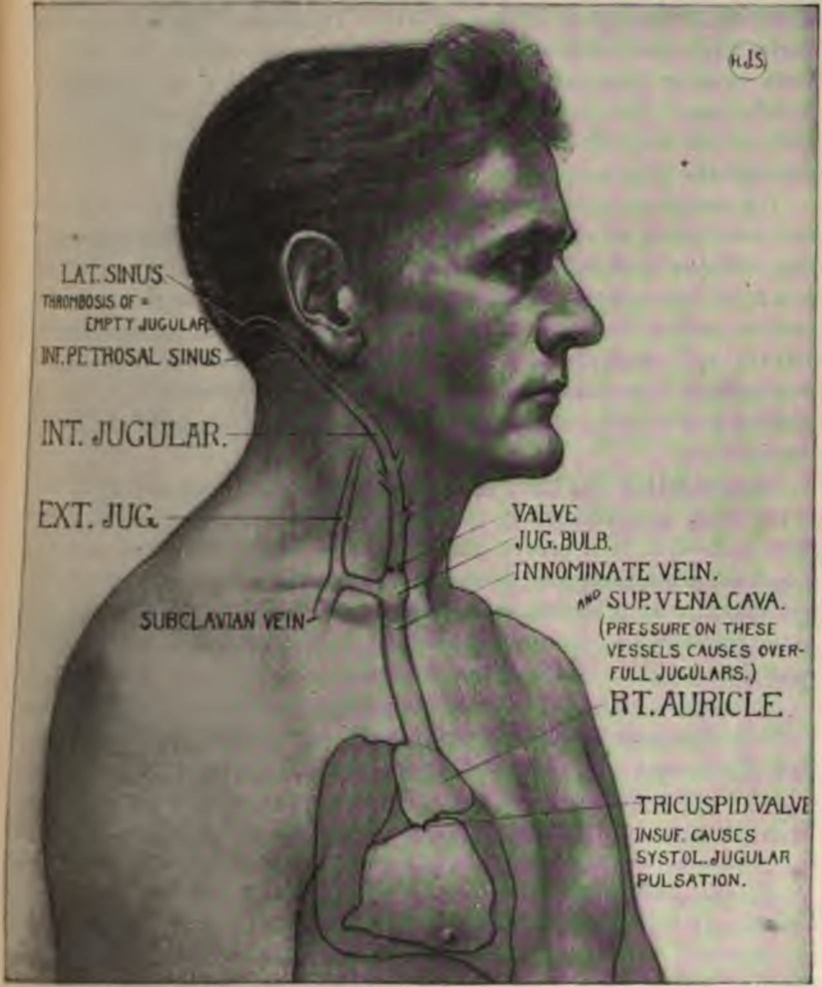

Fig. 66.—Diagram of the right external and internal jugulars, showing the mechanism of jugular collapse, distention, and pulsation.

(*a*) A *collapsed jugular*, the vein remaining permanently more or less empty, even if pressure is made upon it just above the clavicle, is significant of thrombosis of the lateral sinus (Fig. 66). Sudden diastolic collapse is seen in extensive adhesive pericarditis (see (*c*) below).

(*b*) *Distended or engorged jugulars*, without abnormal pulsation, are seen during the act of coughing, or when the glottis is closed to facilitate straining or lifting efforts. Aneurism, especially if intrapericardial, and mediastinal tumour, by compressing the superior vena cava or innominate vein, may be responsible for permanent overfulness. The various causes of obstructed pulmonary circulation, which may ultimately lead to tricuspid regurgitation, tend to engorge the jugulars.

(*c*) *Respiratory swelling and collapse* of the jugulars is under normal conditions so slight as not to be appreciable. But if engorged, they collapse noticeably during inspiration because of the aspirating power of the chest cavity, and swell during expiration when the suction action is least. This phenomenon is seen particularly in asthma and emphysema. It is inversed in indurative mediastinopericarditis, because of the obstruction to the venous flow due to pulling and bending of the adherent vena cava superior by the act of inspiration.

(*d*) **Pulsating Jugulars.**—If pulsation of the jugulars (Fig. 66) is observed, it is absolutely requisite to determine its relation to the apex beat—i. e., is it presystolic or systolic—and whether or not it is a communicated impulse from the carotid artery, which may be present when the vein is distended or the artery throbbing violently. The patient should be requested to breathe very quietly in order to eliminate the respiratory oscillation, if present.

Presystolic pulsation is *normal* and is seen in many healthy individuals, especially when recumbent, as an undulation or flicker at the root of the neck. It is caused by the auricular systole giving rise to a back wave which stops at the valve situated just above the jugular bulb. It may be unusually marked in anæmia and in the rare condition of tricuspid stenosis if the patient is in the upright position.

Systolic pulsation is synchronous with the apex beat and is *pathological.* It is almost invariably due to regurgitation through an incompetent tricuspid valve. If the valve in the internal jugular remains competent the pulsation is seen only in the jugular bulb, but under these circumstances the vein is usually sufficiently distended to render this valve unable to close and the systolic pulsation extends upward into the neck.

Sphygmographic tracings from the jugular, both in health and disease, exhibit certain undulations and variations for which a perfectly satisfactory explanation has not yet been reached. The diagram (Fig. 67) shows all that is required for clinical purposes—viz., the normal presystolic impulse due to the auricular systole, which, if tricuspid regurgitation exists, does not, as in health, terminate with the ventricular systole, but is immediately followed by a greater systolic impulse due to the latter.

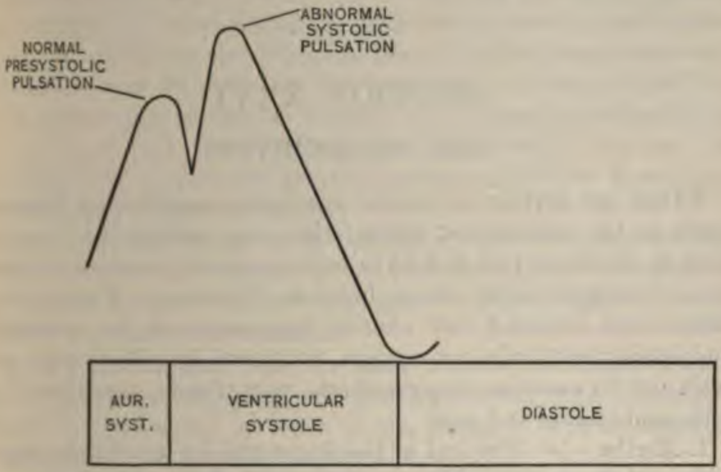

FIG. 67.—Diagram showing the presystolic and systolic jugular pulse.

To determine that the venous pulsation is systolic the finger should be placed upon the vein at the root of the neck and run upward so as to empty the vessel. If there is no regurgitation the vein refills slowly, the blood running into it from the small collaterals; but if the tricuspid and jugular valves are incompetent, successive systolic impulses are seen rising from below, and the vein is very shortly again distended and pulsating. If there is any question as to the pulsation being transmitted from the neighbouring carotid, the finger may be laid upon the vein midway between ear and clavicle, which, if the pulsation is communicated, will cause the peripheral portion above to become more distended and to pulsate more strongly, while the lower portion collapses and pulsation ceases. But if the pulsation is in the vein it will continue as before.

Although, as stated, a systolic venous pulse is usually indicative of tricuspid insufficiency (relative or absolute), it may, as a clinical curiosity, be due to a patent foramen ovale coexisting with mitral insufficiency, the regurgitant current passing from the left to the right auricle and thence to the superior cava and jugulars; or to the still rarer case of a thoracic aneurism communicating with the superior cava.

An additional rare phenomenon, which simulates systolic venous pulse, is a sudden diastolic collapse of the jugulars occurring in certain cases with extensive pericardial adhesions, the walls of the heart contracting with difficulty because of the adhesions, and springing suddenly into diastolic expansion, thus aspirating the venous contents.

SECTION XXVI

THE EXTREMITIES

THERE are certain signs and symptoms manifesting themselves largely in the extremities, which, with some exceptions, have been grouped elsewhere (see Index) in order to avoid unnecessary repetition and promote better comprehension. These are: Tremor, spasm, contractures, athetoid and choreic movements of the extremities, hemiplegia, paraplegia, monoplegia, wrist and foot drop, pain, anæsthesia and its varieties, hyperæsthesia, paræsthesia, condition of the joints, and station and gait.

1. **Nails.**—(*a*) The bed of the finger nail by its *tint* shows excellently well the presence of general cyanosis, less perfectly the existence of anæmia. It is said that if pressure on the finger tip completely exsanguinates the nail bed not more than 50 per cent of hæmoglobin remains in the blood.

(*b*) The *subungual pulse*, an alternate paling and reddening of the nail bed corresponding to the cardiac systole and diastole (capillary pulse), is sometimes easily visible, but often requires slight pressure on the nail tip to develop it, the pale area produced by the pressure reddening with each pulse beat. It is seen in aortic regurgitation, chlorosis, anæmia, severe hemorrhage, and other conditions in which the peripheral arteries are quickly filled and as quickly emptied. The capillary pulse may also be seen by pressing a bit of glass on the inner surface of the lip, or in the red line caused by scratching the skin.

(*c*) A *transverse groove* in the nails, unless due to traumatism, usually indicates a recent acute illness. The growth of the nail from matrix to end requires about 6 months, and a rough estimate as to the date of the illness may be made from the position of the groove.

(*d*) Hard, brittle, and longitudinally striated nails are found in gouty individuals.

(*e*) Malformation, fragility, and dryness or cracking of the nails may be due to injury or syphilis, or may represent trophic defects resulting from nerve injuries, neuritis, syringomyelia, pulmonary osteo-arthropathy, Raynaud's disease, and scleroderma affecting the fingers. Arrested growth of the nails may be present in hemiplegia and acute infantile paralysis. It may be demonstrated by spotting a nail on each hand with nitric acid and comparing the rate of growth.

(*f*) A great hypertrophy of the nails, mainly in the lateral dimension, without defective structure, has been described (*megalonychosis*, Keyes), which may be mistaken for acromegaly, or the clubbed fingers of pulmonary or cardiac disease, or pulmonary osteo-arthropathy; but they lack the brittleness and striations of the last, the bone changes of acromegaly are not present, and there is no long-standing disease of heart or lungs.

(*h*) Ecchymoses and ulcers at the bases of the nails may be present in victims of the chloral habit. An unhealthy ulceration of long standing around the nail (*onychia*) in a child may be due to syphilis or the scrofulous diathesis.

(*i*) An indolent sore near the nail, whether indurated or not, with enlargement of the lymphatic gland above the inner condyle, if occurring in a physician or other person liable to have been in contact with syphilis, is probably an initial lesion.

2. **Hand and Fingers.**—(*a*) The *spade hand*, large, coarse, thick-fingered, with broad nails, is seen as an evidence of myxœdema, in which the enlargement affects mainly the soft parts; and of acromegaly, in which the enlargement affects the bones (Figs. 68 and 69).

(*b*) The *claw hand*, also called ape hand and *main-en-griffe* (griffin hand), is a deformity which occurs in consequence of paralysis and atrophy of the interossei and lumbrical muscles. Paralysis of these muscles leads to a dorsal extension of the proximal phalanges with flexion of the others, and when atrophy takes place the claw hand results (Fig. 70). This abnormality suggests the existence of a neuritis of the median and ulnar nerves, particularly the latter, or progressive muscular atrophy. Similar deformities may be due to amyotrophic lateral sclerosis (the spastic form of progressive muscular atrophy), syringomyelia, and, very rarely, the adult type of chronic anterior poliomyelitis.

(*c*) *Atrophy of the muscles* of the hand and forearm occurs not only in the diseases just mentioned, but also to a slighter degree in cerebral paralysis, and, in consequence of disuse, from rheumatic or gouty affections of the hand.

(*d*) *Coldness of the hands and feet*, with or without a tendency to sweating, if persisting for weeks or months, is most commonly due to

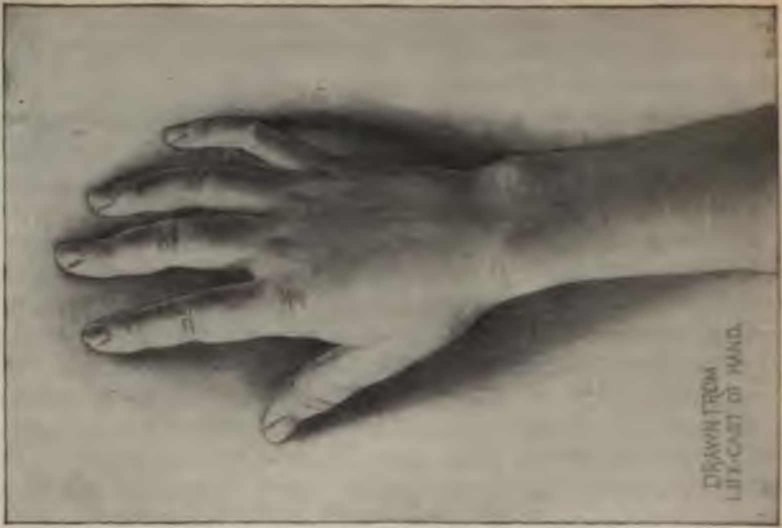

Fig. 68.—Cast drawing of normal hand for purposes of comparison.

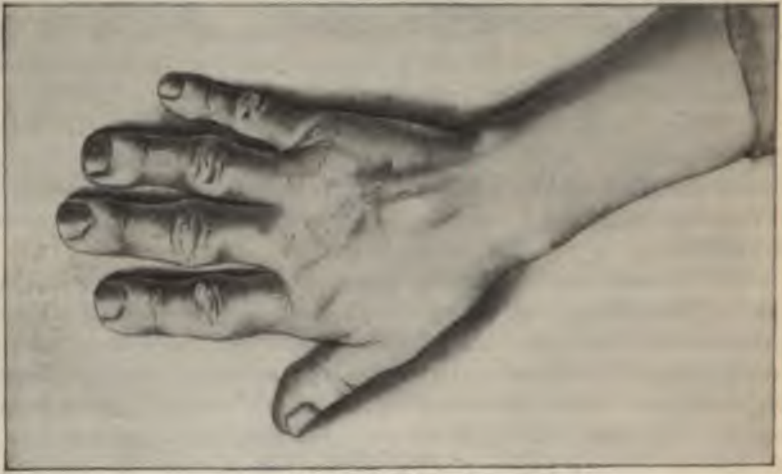

Fig. 69.—Spade hand.

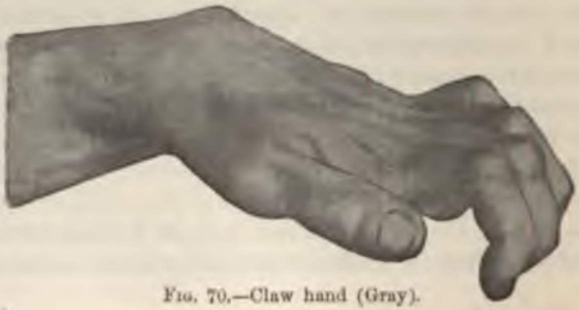

Fig. 70.—Claw hand (Gray).

neurasthenic conditions, anæmia, chronic digestive disorders, rheumatic or gouty affections, and cardiac or pulmonary diseases interfering with the circulation. Sudden or transient coldness of the extremities is observed in many persons, especially those of a nervous temperament, under excitement or anxiety, as well as in shock, collapse, anginose attacks, hemorrhages, and the premonitory chilliness of rising fever. It is necessary to discriminate between actual lowering of the temperature palpable to the observer and a subjective sensation of coldness. The latter is a paræsthesia.

(*e*) *Excessive sweating* of the hands may be due to hysteria, progressive muscular atrophy, bromidosis, and excessive leucorrhœa.

(*f*) *Glossy skin*, smooth, close fitting and hairless, is an evidence of nutritive disturbance caused by injury to a nerve, neuritis, or affections of the trophic centres.

(*g*) A red or waxy fugitive swelling which does not pit upon pressure is probably an example of angioneurotic œdema.

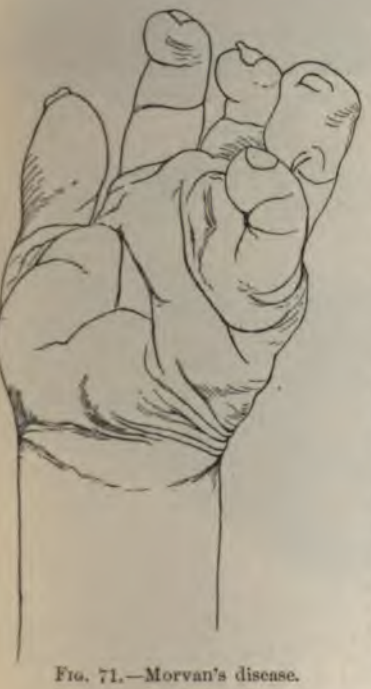

FIG. 71.—Morvan's disease.

(*h*) A waxy or dusky blue colour of the fingers, followed perhaps by areas of dry gangrene, may be caused by Raynaud's disease, alcoholic neuritis, leprosy, or frostbite. If painless whitlows form (Fig. 71), it will come under the head of Morvan's disease, which is sometimes neuritis, in other cases the neuritic form of syringomyelia.

(*i*) *Clubbed fingers*, the finger ends becoming bulbous and the nails much curved longitudinally and laterally, are seen in conditions which cause long-continued congestion of the peripheral veins, such as congenital malformation or acquired valvular disease of the heart in children. Prolonged dyspnœa, chronic bronchitis, emphysema, chronic pulmonary phthisis, old and extensive pleuritic adhesions, may also be responsible for this change in shape. A similar clubbing is present in pulmonary osteo-arthropathy (Figs. 72 and 73), but with this there are in addition enlargements of the ends of the bones of both arms and legs.

(*j*) *Distorted fingers*, not merely abnormal positions, but notable irregular changes in their shape, are due to gout, arthritis deformans, and, less frequently, to chronic rheumatism. In gout the deformity is largely due to deposits of sodium urate in and about the phalangeal and metacarpo-phalangeal joints, and tophi (chalky, uratic masses) are frequently seen. In arthritis deformans there are extensive

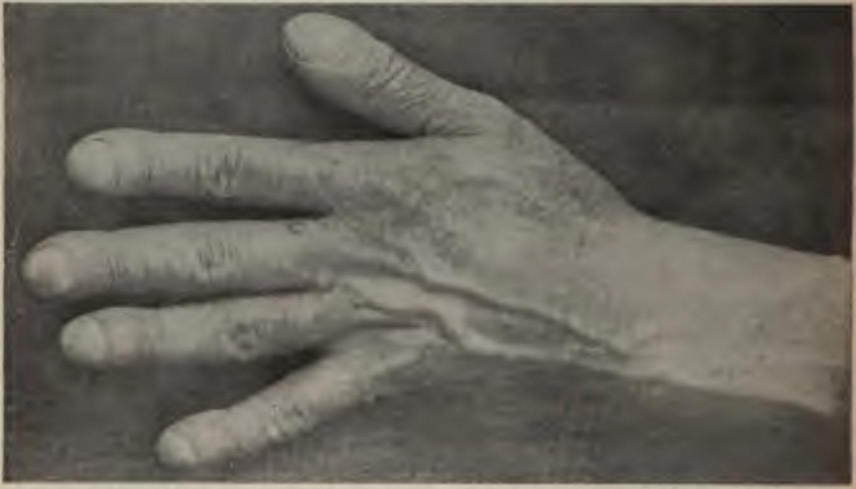

FIG. 72.—Hand of pulmonary osteo-arthropathy, due to chronic bronchitis of thirty years' standing.

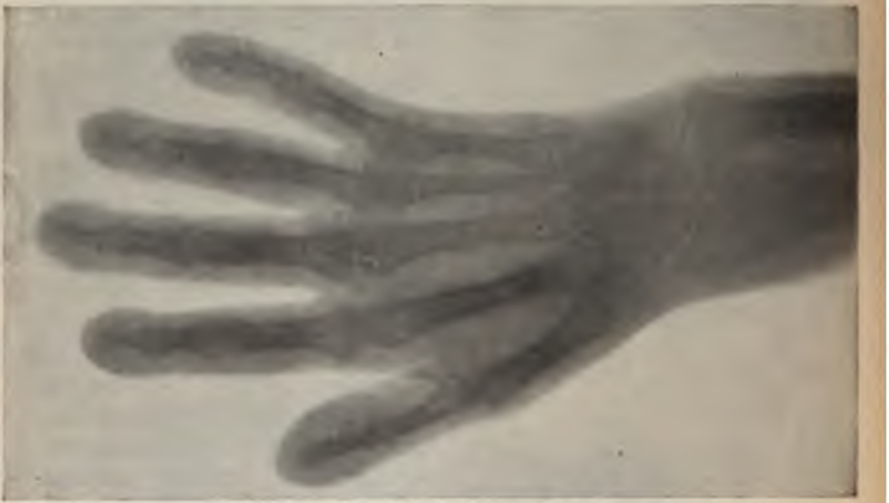

FIG. 73.—Skiagraph of hand in Fig. 72, above.

changes in the articular extremities of the phalanges (Fig. 74), leading in part to absorption of bone, in part to the development of exostoses. Trophic changes and wasting of muscles may be present to such an extent as to give a close resemblance to the hand of progressive muscular atrophy.

In both gout and arthritis deformans the fingers may be deflected toward the ulnar side of the hand, and the joints are anchylosed in varying degrees. The deformities resulting from one, may closely resemble those of the other so that a differential diagnosis may be difficult. If the excrescences found are easily recognised as tophi, or proved to be such by their superficial seat, or by ulceration or incision giving exit to a chalky material, the gouty nature of the deformity is settled. Otherwise the discrimination is to be made by the history, and the presence of associated symptoms and joint changes, or tophi elsewhere—e. g., ear and great toe.

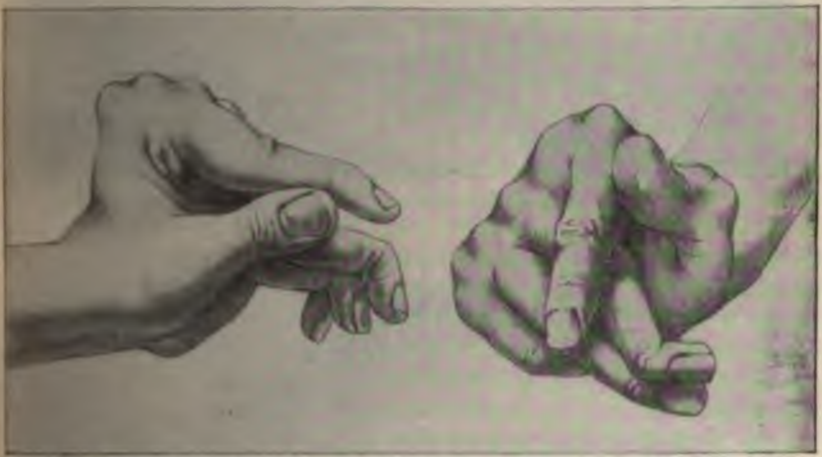

FIG. 74.—Hands of arthritis deformans. Drawing made by permission of Dr. G. R. Hall.

Heberden's nodes, knobby enlargements of the proximal ends of the terminal phalanges (Figs. 75 and 76), may also be due either to gout or arthritis deformans. Small vesicles, "crab-eye" cysts, may form over the nodes.

In determining the existence of enlarged joints it must not be forgotten that atrophy or wasting of the soft parts may cause an apparent increase in the size of the joints.

(*k*) A *flexed finger* may be due to the contraction of a scar resulting from a burn or a felon, but if a dense ridge is found passing from the palm to the finger, not of traumatic origin, it is a case of Dupuytren's contraction (of the palmar fascia). The patient is usually under the erroneous impression that it is a contracted tendon. The contraction may be of gouty origin, although there is some basis for considering it to be a neurogenic condition. (See also Contractures.)

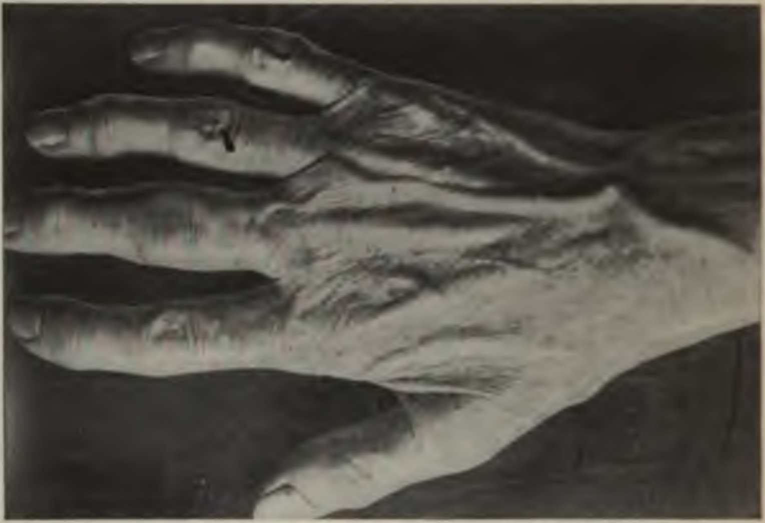

FIG. 75.—Hand showing Heberden's nodes.

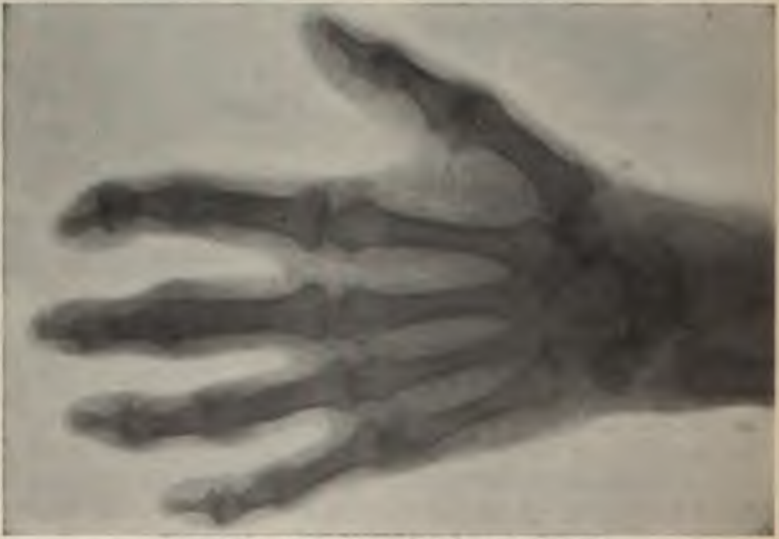

FIG. 76.—Skiagraph of hand in Fig. 75, above.

(*l*) *Scleroderma.*—Here may be mentioned the hand of scleroderma, in which the skin becomes thickened and firmly fused to the subcutaneous tissues. The skin is glossy, dry, and smooth, and because of its tense, unyielding character the fingers and hands become immovable. The disease (probably a trophic neurosis) may affect localized portions of the body or, less commonly, be diffused over the entire surface.

(*m*) *Handwriting.*—Total inability to write may be due to aphasia (*q. v.*), mental defects, and lack of education.

Indistinct or illegible writing may indicate carelessness, gouty or rheumatic hands, writer's cramp, or the tremor of old age, disseminated sclerosis, alcoholism, general paralysis, or coarse cerebral lesions. "Mirror writing," in which words are written from right to left and reversed, as if seen in a mirror, may be a result of some cerebral lesions, deficient mental development, and perhaps hysteria.

3. **Arm.**—(*a*) Œdema of *one arm and hand* may be the result of a tumour of the mediastinum or lung, aneurism of the arch of the aorta, innominate (right arm) or axillary artery, thrombosis of the axillary vein, enlargement of the axillary glands, or trichiniasis.

(*b*) A slow enlargement of the *lower ends of both radial bones* in a weak or poorly fed child is probably due to rachitis, and confirmatory evidence may be found in other regions.

(*c*) Circumscribed bony swellings of long duration on the subcutaneous surface of the ulna are syphilitic nodes. Recent, round, and superficial indurated swellings in the same locality, with a mottled or bruised appearance of the skin, are the lesions of erythema nodosum, which, however, occurs more commonly over the tibia. Painless, non-inflammatory, circumscribed indurations about the *elbow* are probably gummata.

(*d*) Pain and tenderness over the head of the *radius*, with restriction in the movement of the joint, is sometimes observed after overuse of the arm.

(*e*) Stiffness and pain in the *shoulder joint*, increased by damp weather, and occurring as a rule in an elderly person, is a rheumatic inflammation of the ligaments; but, if accompanied with an inability to elevate the arm, it may be due to neuritis of the circumflex nerve.

(*f*) Enlargement of the *humerus*, *radius*, or *ulna* of a chronic nature, accompanied by constant deep-seated pain which increases at night, may be due to syphilitic periostitis.

(*g*) If the *humerus* becomes acutely swollen or painful during the course of typhoid fever or scarlatina, it is an acute periostitis which may eventuate in necrosis and abscess.

4. **Foot and Leg.**—(*a*) Œdema (*q. v.*) of both lower extremities arising from general conditions is described elsewhere, but œdema of one leg only may be due to thrombosis of the femoral vein or varicose veins of the leg. Unilateral œdema may accompany rheumatic or gouty inflammation of the foot.

(*b*) *Enlarged glands in the groin* occur in two main groups—one along Poupart's ligament, the other over and around the saphenous opening. Their location and the areas drained by them are shown in Fig. 77.

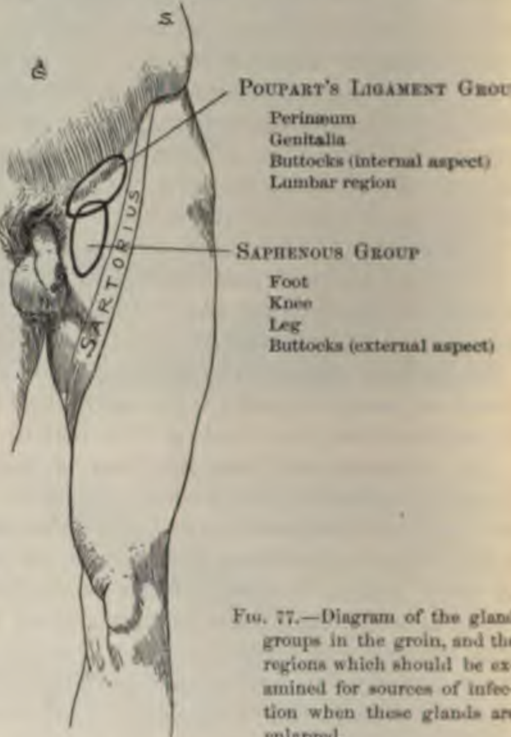

FIG. 77.—Diagram of the gland groups in the groin, and the regions which should be examined for sources of infection when these glands are enlarged.

Acute swelling (adenitis) results from the virus of chancroid, gonorrhœa, and syphilis. Enlargements due to the last-named disease are usually small, hard, and painless. There is a rare acute adenitis occurring in the course of acute articular rheumatism, which yields to the salicylates.

Chronic swellings may be tuberculous, secondary to tuberculous disease in the hip or knee, but a simple adenitis due to intertrigo, eczema, or varicose ulcer may become tuberculous. The enlarged glands may be a part of Hodgkin's disease, in which case similar changes will be found elsewhere. In tertiary bone or genital syphilis the inguinal glands may undergo gummatous changes, but this is extremely infrequent. Malignant disease of the genitalia, especially epithelioma and carcinoma, may secondarily invade these glands, and they are not infrequently affected in gastric cancer. Very seldom they may enlarge because of the presence of parasites, echinococcus, cysticercus, and *Filaria sanguinis hominum*. Aside from glandular disease, swellings in the groin may be due to femoral aneurism, retained testicle, and a small incarcerated hernia, the latter sometimes of great importance as a possible explanation of abdominal pain or the symptoms of intestinal obstruction. A fluctuating swelling at the apex of Scarpa's triangle, internal to the femoral vessels, may be a psoas abscess.

(*c*) The *femoral artery* in elderly emaciated individuals may be thickened and rigid. Under these circumstances it may pulsate so visibly as to simulate aneurism. On the other hand pulsation may be absent because of closure of its lumen by the arteritis which is present, gangrene occurring as a sequel.

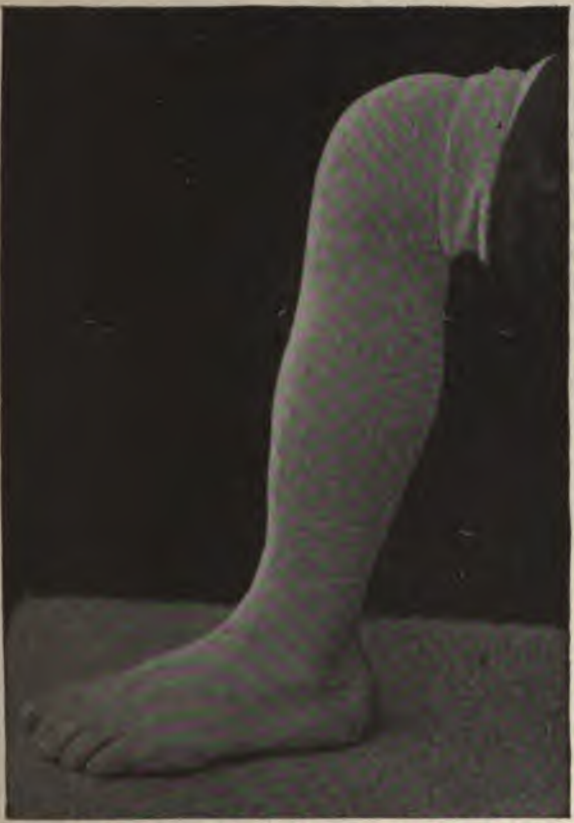

Fig. 78.—Syphilitic disease of the tibia, showing the sabre-like deformity, in a boy nine years old (Holt).

(*d*) Chronic painful enlargement of the tibia, the pain being worse at night, with a somewhat characteristic deformity (Fig. 78), is a *syphilitic periostitis*, late hereditary if occurring in a child, tertiary in the adult.

(*e*) Redness, heat, and œdema of thigh or anterior and inner aspect of the leg, occurring with a rise of temperature in the course of the acute specific fevers, may be an *acute periostitis* of femur or tibia, with sequent suppuration and necrosis.

(*f*) *Curvatures of the leg bones* are most commonly due to rachitis, but also occur in osteitis deformans, mollities ossium, and cretinism. Shortening of one leg may be the result of tuberculous disease of the hip or an old infantile paralysis.

(*g*) *Nodes*, small circumscribed bony growths situated upon the tibia, are tertiary syphilitic manifestations. Reddened or bruised-looking patches, having a nodelike feeling on palpation, are probably the manifestations of erythema nodosum, but scurvy and the various forms of purpura should not be overlooked as a possible explanation. Painless, non-inflammatory indurations scattered over the leg may be gummata, some of which may subsequently ulcerate. Multiple annular ulcers, nearer the knee than the ankle, are probably tertiary syphilitic lesions.

(*h*) An abnormal increase in the size of the calves in a child, combined with difficulty in walking and going upstairs, owing to weakness of the apparently hypertrophied muscles, is noted in *pseudo-hypertrophic muscular paralysis*.

(*i*) Atrophy of the anterior and outer muscles below the knee is a symptom of the rather uncommon peroneal type of *progressive muscular atrophy*.

(*j*) *Varicose veins* of the leg may be significant of excessive standing, fæcal accumulation, abdominal tumours, pregnancy, or other conditions interfering with the return circulation. A species of lineæ albicantes or welt-like scars may appear over the front of the knees in rapidly growing adolescents during typhoid fever.

(*k*) A painful œdema and congestion of the leg and foot, with a knotty condition of the veins behind the internal malleolus, in a gouty individual, may be due to *phlebitis* of the deep veins of the leg.

(*l*) *Perforating ulcer* of the foot, occurring with locomotor ataxia, and rarely with diabetes, presents itself as a deep, circular lesion, usually under the great toe, and often leads to necrosis of the bones.

(*m*) Redness and swelling confined to the surface of the *metatarso-phalangeal joint of the great toe* may be of gouty origin, or a bursitis arising from shoe pressure upon a deformed joint. In the latter case the attack is not sudden, and there is no constitutional disturbance.

(*n*) *Gangrene of the foot*, beginning in the toes, is most frequently a consequence of diabetes or disease of the arteries in elderly persons (dry, senile gangrene). Less often it is a result of the embolism of cardiac disease and thrombosis due to the specific infections, especially enteric fever and scarlatina. Raynaud's disease, and very rarely exophthalmic goitre, may give rise to gangrene of the toes or localized patchy spots of necrosis. Ergotism, trauma, and frostbites are other causes which may be responsible for this condition.

(*o*) Burning pain in the sole of the foot, with a mottled, dusky redness, both of which are made worse by walking and relieved by elevating the limb, constitute *erythromelalgia*, or red neuralgia of the feet.

(*p*) Abnormal size, distortion, atrophy, clubbing, and contractures affecting the feet and toes, as well as changes in the toe nails, are, when found, due in the main to the same causes which produce similar changes in the upper extremities—viz., disease of joints, acromegaly, myxœdema, pulmonary osteo-arthropathy, hysteria, locomotor ataxia, Friedreich's ataxia, anterior poliomyelitis, cerebral paralysis, progressive muscular atrophy, and multiple neuritis.

(*q*) *Clubfoot and Flat Foot.*—These deformities are, as a rule, congenital, but may result from a previous infantile paralysis (anterior poliomyelitis). The history usually determines this point, but it may be borne in mind that if the deformity is paralytic the limb is usually atrophied, the muscles are flaccid, and the deformity may be temporarily reduced without much difficulty. The varieties are:

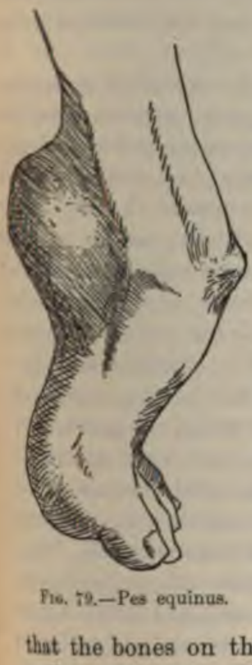

Fig. 79.—Pes equinus.

Pes (or talipes) equinus, in which there is a drawing up of the heel so that the patient walks upon the ball of the toes or even upon the dorsum of the foot (Fig. 79); or, as in a second variety with a nearly normal position of the heel, there is a sharp dropping of the front portion of the foot. This may occur in pseudo-hypertrophic paralysis. There is also a slight deformity, caused by contraction of the plantar fascia from arrested growth of the latter, as a remote result of anterior poliomyelitis. The tarsal arch is exaggerated, the distance between the ball of the foot and the heel is lessened, the proximal phalanges are extended, and the terminal phalanges flexed. The deformity, as a whole, bears some resemblance to the claw foot.

Pes varus is an inversion of the foot, so that the patient walks upon its outer border (Fig. 80).

Pes valgus is an eversion of the foot, so that the bones on the inner side of the knee and ankle are abnormally prominent, and the arch of the foot is lost. It is an exaggerated flat foot (pes planus). The latter is quite frequently responsible for a painful condition of the heel or sole of the foot for which gout, rheumatism, or other causes of painful feet may have been held responsible.

Pes calcaneus (usually with valgus), the foot being drawn up to the leg by contraction of the extensors and everted, the patient walking upon the inner side of the heel (Fig. 81), is one of the unusual deformities resulting from infantile paralysis, and may exist in connection with spina bifida.

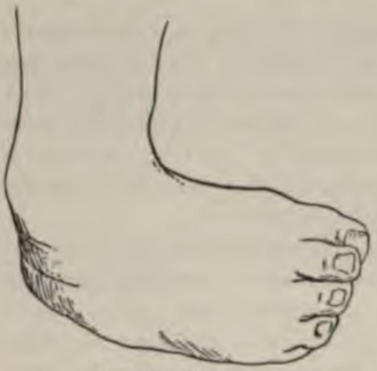

Fig. 80.—Pes varus.

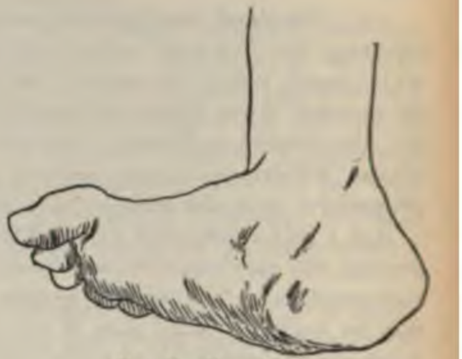

Fig. 81.—Pes calcaneus.

(*r*) *Kernig's Sign.*—In order to elicit this sign the thigh must be placed at a right angle to the body, the patient lying upon the side or, better, upon the back. The same object can be accomplished by having him sit upon the edge of the bed, thighs horizontal, legs hanging vertically downward. An attempt is then made to extend the leg, thus bringing it in a line with the thigh. If meningitis is present, it will be difficult or impossible to extend the leg because of the presence of a marked flexor contracture (of the hamstring muscles), and the procedure is often painful. In delirious patients gentle but persistent traction must be used before the muscles yield; but, if a true Kernig's contraction exists, the muscles do not give way even to a long-continued steady effort, and if the patient is in the dorsal position the pelvis may generally be lifted from the bed without causing the leg to extend.

It is necessary to exclude sciatica, old contractures, disease of the knee or hip joint, myositis, old age, prolonged recumbency, acute eye diseases, and lesions of the cerebellum or upper motor neurones. This sign is present in from 80 to 90 per cent of cases of meningitis, or cerebro-spinal fever. With the reservations mentioned it is a differential symptom of value.

SECTION XXVII

THE BACK

THE back should be examined for curvatures of the spinal column, prominence of spinous processes or scapulæ, swellings, and stiffness. (For pain and tenderness in the back, see Index.)

(*a*) **Kyphosis.**—A posterior curvature of the spine with the convexity directed backward (kyphosis) is seen in many elderly persons, especially in the dorsal region, and it forms a part of the changes which constitute an emphysematous chest. A kyphotic condition of the spine may be present in a rachitic child or in old or young persons who are weakened by acute or long-continued illness. These posterior curvatures are to be distinguished from the more or less sharply angled kyphosis of Pott's disease of the spine and mollities ossium localized in the cervical, dorsal, or lumbar region. In some patients, children in particular, there is a somewhat unusual but normal prominence of the 7th cervical (*vertebra prominens*) or 8th and 9th dorsal spinous processes.

(*b*) **Scoliosis.**—A lateral, in reality a rotary-lateral, curvature of the spine (scoliosis), with prominence of one scapula, is witnessed most frequently in girls from 8 to 15 years of age. It may indicate unequal length of the lower extremities; the habit of standing habitually on one leg or carrying weights on one arm; muscular weakness from fever or anæmia; and general debility, congenital or acquired. Its most common cause is rachitis. It may also result from the tilting of the pelvis in old sciatica, paralysis due to anterior poliomyelitis and cerebral paralysis, and mollities ossium. It is not infrequently encountered as a result of the contraction of one lung after a pleurisy or an empyema.

(*c*) **Lordosis.**—An unnatural curvature of the spine, with the convexity looking forward (*lordosis*), when found, is usually in the lumbar region, and is indeed an exaggeration of the normal curve in this locality. It may be due to the dragging weight of pregnancy, ascites, and all large abdominal tumours. It is apt to occur in the course of pseudo-muscular hypertrophy.

(*d*) **Prominent Scapulæ.**—If both scapulæ are abnormally projecting, it is probable that the patient has an alar or pterygoid chest. If one scapula projects, it indicates the existence of lateral curvature and its existing causative conditions; or paralysis of the serratus magnus on that side (Fig. 82). If the left scapula projects, it is possibly due to an aneurism of the arch of the aorta which has bulged out the left interscapular region.

(*e*) **Lumbar Bulging.**—A swelling in the posterior lumbar region on one or the other side of the spinal column, either solid, fluctuating, or edematous, may be due to malignant disease of the kidney, pyonephrosis or hydronephrosis, hydatids of the kidney, perinephritic abscess, or an abscess connected with caries of the spine. In the latter case there is usually a coexisting angular kyphosis.

(*f*) **Stiff Back.**—Stiffness and lack of mobility in flexion, extension, and lateral bending of the spine, with or without pain in so doing, may be caused by muscular rheumatism (lumbago) or strain of the back muscles, arthritis deformans, or a tonic spasm of the muscles, as in opisthotonus.

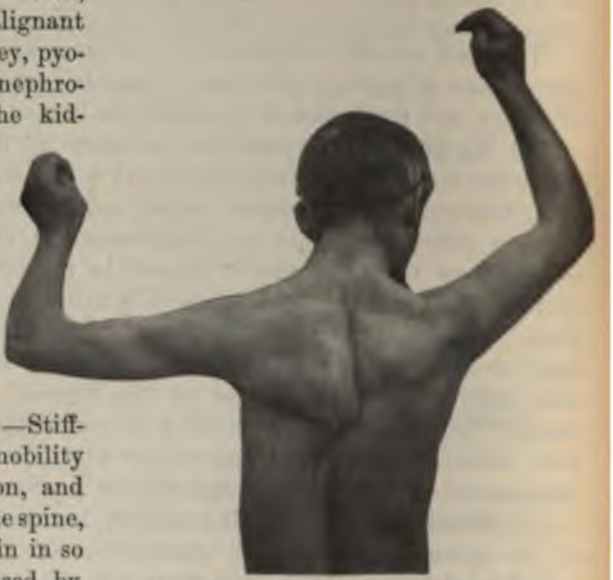
FIG. 82.—Paralysis of the serratus magnus (Leszynsky).

Chronic stiffness or anchylosis of the vertebral column without involvement of other joints, except, perhaps, those of the hip and shoulder, may be due to injury, Pott's disease, chronic or gonorrhœal rheumatism, paralysis agitans, arthritis deformans, or certain laborious occupations. Two other varieties of chronic spondylitis have been described, which are believed by some authors to be special types of disease, and dependent on other causes than those just mentioned.

The first is the so-called spondylitis rhizomelia (STRÜMPELL-MARIE). The disease, which attacks men only, at or beyond middle age, begins usually in the hip joints, which become anchylosed, the process subsequently extending to the spine and the shoulder joints, very rarely to the knee joints. The spine becomes rigid, and there is kyphosis (Fig. 83), usually not very marked. The dorsal and gluteal muscles are atrophied, and exostoses are found upon the vertebræ and sacral bones. There is but little pain attending the process.

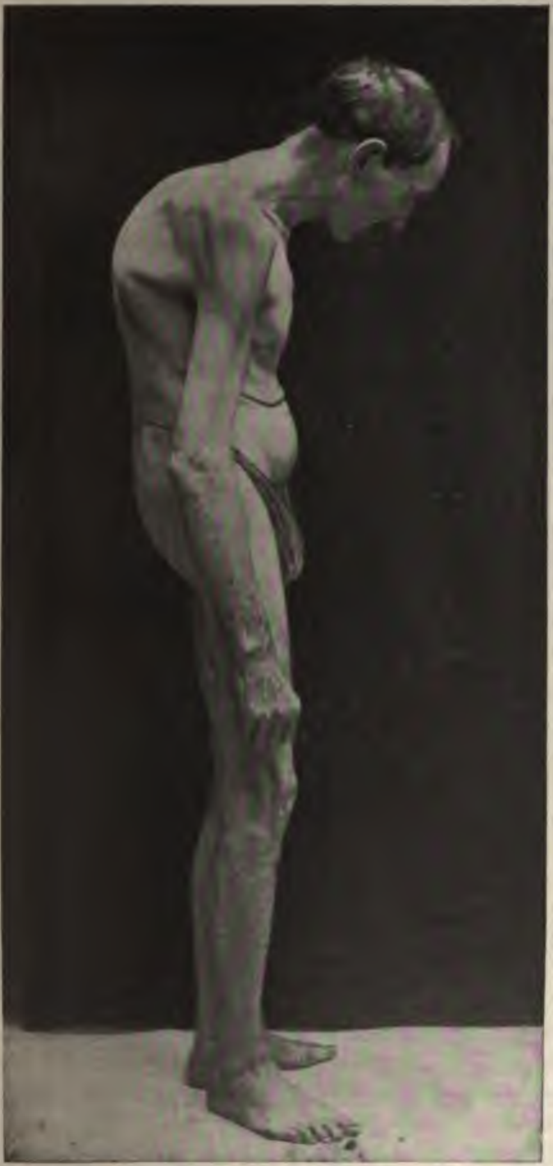

FIG. 83.—Rhizomelique spondylitis (dorsal spine) (Pearce).

In the second type (Bechterew-Marie) the trouble begins in the spine, which becomes anchylosed and kyphotic, the shoulders stoop, the head is lowered and carried forward, and there is much intercostal pain. The hip and shoulder joints are slightly if at all affected. The disease is often hereditary.

Dana, who has made a careful study of the subject, believes that these two conditions are not special maladies, but that the first is a form of arthritis deformans, and the second either arthritis deformans or syphilitic meningitis.

(*g*) **Swellings.**—A rounded congenital tumour in the middle line, especially if translucent and reducible, and associated with club-foot or hydrocephalus, is a spina bifida. Fluctuating swellings, cervical, dorsal, lumbar, or sacral, may be abscesses in connection with disease of the vertebræ or sacro-iliac joint. A fatty tumour (lipoma), unless lobulated, may simulate an abscess, and require aspiration for a differential diagnosis. An unusual case seen in consultation was that of a suppurating dermoid cyst anterior to the sacrum. The pus worked its way outward through the greater sacro-sciatic foramen, causing a typical sciatica, for which it was mistaken until a fluctuating swelling appeared in the buttock. Two radical operations resulted in recovery. A red, brawny swelling, usually in the cervical region, discharging pus by one or more openings, is a carbuncle, and when found requires an examination of the urine for sugar.

SECTION XXVIII

THEORY AND PRACTICE OF PALPATION, AUSCULTATION, AND PERCUSSION

I. PALPATION

Palpation, the use of the tactile sense of the hand and fingers, is an indispensable means of obtaining information. The remarkable extent to which the "educated touch" may be developed is exemplified in many physicians.

In the practice of palpation care should be taken to keep the nails short. The hands should always be warmed before touching the patient. Neglect of these points may seriously impair the usefulness of the practitioner. All rough or abrupt pressure should be avoided. The hand at first should be laid flat upon the part; later the fingers and their tips may be employed to determine and localize more accurately any discoverable abnormality. If palpating deep-seated organs, or if there is a large amount of overlying fat or fluid, the palpating hand may be usefully re-enforced by pressing upon it with the other, thus sparing the palpating hand much muscular exertion, and leaving unimpaired its power of tactile appreciation.

When palpating, the points to be determined with reference to the part or organ are:

Shape.—Rounded, ovoid, irregular, nodular.

Size.—Hazelnut, almond, English walnut, lemon, orange, fœtal head, etc.

Consistence.—Hard, soft, edematous, brawny.

Movable.—Freely, slightly, with or not with respiration.

Tenderness.—Slight, marked, deep-seated, superficial.

Fluctuation.—The undulation of an inclosed fluid produced by the pressure of the finger, and appreciated by another finger placed on the opposite side of the inclosing cavity.

Fremitus.—Vibrations originating in the larynx during phonation, and transmitted through the trachea, bronchi, and lung substance to the surface of the chest.

Pulsations.—Character and locality noted.

Thrill.—Vibrations originating in the heart or blood vessels, and transmitted to the surface.

II. PERCUSSION

(1) **Theory of Percussion.**—If any portion of the body is sharply struck by the finger, the part tapped either resounds to the impact or gives out a dead, non-resonant sound, like that resulting from percussion of a mass of moist clay. Resonance upon percussion shows that the part percussed is so constructed that it is able to vibrate with some regularity, whereas a non-resonant (dull) sound is due to an almost entire lack of such power. The term "clear" is employed as a synonym for "resonant." Resonance, therefore, has some of the characters of a musical tone, the latter consisting of a series of uniform and regular vibrations, while the dull sound, not arising from regular vibrations, is technically a noise. The structures of the body which resound when percussed are the bones and the air-containing organs. Bone tissue is sufficiently elastic to vibrate when struck. The air in the hollow or air-containing viscera vibrates rhythmically when percussed, and the vibrations are increased or diminished by the degree of tension of the containing walls.

Bone (or osteal) resonance has a character of its own—e. g., percussion of the sternum or cranium. As there are wide differences in the size, complexity, and tension of the air cavities in the organs containing air (stomach, intestines, lungs), the character of the percussion sound or note is often quite distinctive. The stomach cavity is large and simple, and emits a drumlike (tympanitic) sound, while the lung, with its complicated arrangement of multitudinous air cells and tubes, affords a percussion note (pulmonary resonance) the quality of which is easily recognisable by experience. There is little to be gained by the recognition of osteal resonance except to avoid confusing it with air resonance. Practically, percussion is employed to determine whether there is more or less than the normal amount of air in an air-containing organ, as, for instance, in emphysema or consolidation of the lung; or to delimit the borders of adjacent airless and air-containing viscera—e. g., liver, lung; or contiguous air-containing organs—e. g., lung, stomach. The line of contact between two contiguous airless organs can not be located unless by a very slight change in the quality of the sound, the dull percussion sound over one being continuous with that over the other—e. g., heart, liver. One also detects, by lack of resonance, the possible interposition of fluid or tissue between the percussed surface and an underlying air-containing organ, as in pleural effusion or thickening.

(2) **Practice of Percussion.**—(*a*) **Technic.**—The plessor or striker may be a small hammer, or, as is commonly the case, a finger may be used. The blow may be received upon the percussed surface with nothing interposed to modify the stroke (direct or immediate percussion), or a pleximeter may be employed (indirect or mediate percussion). Pleximeters are made in the shape of a pillar or plate of metal, glass, rubber, or celluloid.

With regard to the choice of methods it is probable that, as in other lines of work, each practitioner will by a process of selection evolve a procedure which fits best to his special manner of work. Personally the middle finger of the right hand is the best plessor, and the middle finger of the left hand the best pleximeter. The finger pleximeter adapts itself smoothly to the surface under examination and, as an additional and valuable advantage, is able to appreciate the resistance of the underlying structures. It is employed also in "threshold" percussion, an important aid in outlining the exact limits of the entire cardiac dulness—i. e., the size of the heart.

The pleximeter finger having been laid firmly and smoothly upon the surface to be percussed and adapted to its inequalities, the stroke is delivered upon the middle phalanx of the finger by the plessor finger, its tip striking vertically upon the pleximeter finger. The blow should be given by a quick, elastic movement of the wrist and finger joints, the hammer finger rebounding at once in order not to damp the vibrations which have been initiated. It is sometimes serviceable to place the 4 fingers of the left hand upon the surface and percuss from one to the other with successive single strokes. A change from dulness to resonance is often thus brought out with unusual clearness.

Three, or at most four, successive strokes in the same spot are desirable, as a longer series is apt, by fatiguing the ear, to interfere with a correct judgment of the character of the sounds. Nothing is gained by long-continued percussion over one point.

(*b*) **Judging the Sound.**—In estimating the character of the percussion sound the elements to be considered are *pitch* (high or low), *duration*, *volume*, and *quality*. The sound produced by percussion over a considerable body of air in a simple cavity is low in pitch, of decided duration and volume and, taking all the factors into consideration, is said to possess a *tympanitic* quality (e. g., stomach). At the other extreme is the percussion sound over a cavity containing fluid, which, so far as it can be said to have pitch, is high, short, of little volume, and is called *flat* (e. g., pleural effusion). The percussion sound over the lung has a distinctive quality, *normal pulmonary resonance*. Absence of resonance not amounting to flatness is called *dulness*. There are two percussion sounds of peculiar quality, the "cracked-pot" and amphoric or metallic, which will be described in connection with the examination of the lungs (*q. v.*).

Depending upon the fact that in many instances, as a surface is traversed, a percussion sound of one kind merges almost imperceptibly into another variety, or that a given sound can not always be classed with absolute definiteness, certain qualifying terms are employed to describe degrees of difference, their number in actual practice varying with the personal acuteness of the examiner. The following list, arranged in logical order, is believed to comprise as many grades as are desirable. Tympanitic resonance possesses the lowest pitch and greatest volume, flatness the highest pitch and least volume. The physician inquires:

First: Is this sound resonant or dull?
Tympanitic?
Almost tympanitic?
Dull tympanitic?
(Cracked-pot resonance?)
(Amphoric resonance?)
Hyperresonant?
↕
Second: Is it—Normal resonance?
↕
Diminished or impaired resonance?
Slight dulness?
Marked dulness?
Flatness?

Third: Is it normal for the area where it is found?

(*c*) **Muffling of the Sounds.**—The greater the thickness of the tissue (muscular, adipose, or other) which overlies an air-containing organ, the greater is the damping or muffling of the vibrations excited by percussion. The resulting sound is, therefore, more or less dull. An allowance, to be learned by practice, must be made for this fact when examining an obese person or one with edematous thoracic walls, or a deep-seated organ. The employment of an unusually strong percussion stroke will largely obviate this difficulty.

(*d*) **Strength of Stroke.**—The refinements of percussion are lost if a powerful thumping stroke is uniformly employed. Such a stroke will easily demonstrate the flatness of a chest full of fluid, but is entirely unsuitable for such work as accurately outlining the heart or the lower edge of the liver. The strength of the percussion stroke should, therefore, be modified in accordance with the considerations about to be stated.

A gentle stroke throws the underlying tissues into vibration to a certain depth, while by a more powerful blow vibrations are returned from structures situated still farther below the surface. The diagram (Fig. 84) will aid in elucidation. The small triangles represent the depth reached by gentle, the large triangles by strong, percussion strokes. In passing from *A* to *B*, from air-containing lung over solid liver to air-containing intestine, it is desired to determine, *first*, the upper border of the liver; *second*, the lower border of the lung; *third*, the lower border of the liver, these organs being so shaped and situated as to overlap or underlie one another. Beginning at the upper part of the right chest, one passes downward, percussing smartly, until the normal pulmonary resonance becomes diminished, because the stroke has been sufficiently strong to develop the dulling effect of the solid liver, although covered by the lung as at 1. If the stroke had been gentle, as at 2, nothing would have been elicited except the resonance of the lung tissue. As the next point is to determine the lower edge of the gradually thinning lappet of lung, the percussion stroke should be gentle in order to bring out lung resonance only, until it terminates abruptly in the unmistakable dulness of the uncovered liver (strokes 2 and 3). Finally, gentle percussion defines the lower thin edge of the liver (strokes 5 and 6), a forcible stroke as at 4 developing too soon the tympanitic resonance of the intestines, and defeating the object in view. Therefore, to formulate the conclusions:

(1) Use *forcible percussion* when the organ, tumour, consolidation, or fluid is deep-seated, or the covering walls are thick (see (*c*) preceding).

(2) Use *gentle percussion* when the edges of the organ are thin,

and the organ superficial, especially when it is desired to define its limits.

(*e*) **Auscultatory Percussion.**—If, instead of listening to the percussion sounds as transmitted through the air, the stethoscope is employed to convey the vibrations direct to the ear, it constitutes auscultatory percussion. It is a sufficiently useful method to deserve a much wider use than it has yet attained. Its particular application is to determine the outlines of either solid or air-containing organs.

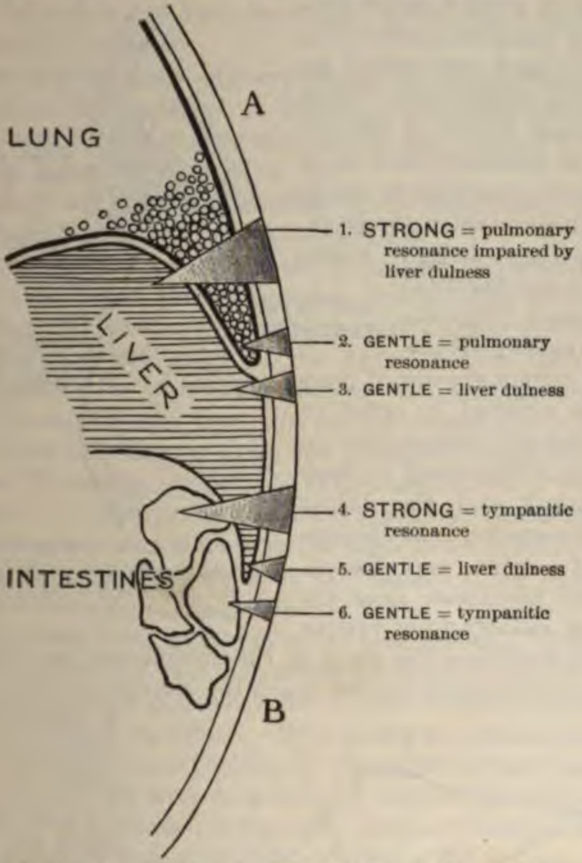
A
LUNG
LIVER
INTESTINES
B
1. STRONG = pulmonary resonance impaired by liver dulness
2. GENTLE = pulmonary resonance
3. GENTLE = liver dulness
4. STRONG = tympanitic resonance
5. GENTLE = liver dulness
6. GENTLE = tympanitic resonance

FIG. 84.—Diagram showing the *rationale* and utility of varying the force of the percussion stroke. A strong stroke as at 1 develops deep dulness and locates the upper border of the liver, while a gentle stroke in the same spot gives only pulmonary resonance. A gentle stroke at 2 gives pulmonary resonance and the next gentle stroke, 3, gives liver dulness, thus locating the lower edge of the lung, while a powerful stroke at 2 will give mainly liver dulness. Gentle stroke 5 is slightly dull, and gentle stroke 6 is unmistakably tympanitic, thus marking the boundary line between thin edge of liver and air-containing intestine, while strong stroke 4 elicits tympanitic resonance.

In practice the technic consists in placing the chest end of the binaural stethoscope upon the surface overlying the organ which is to be delimited, requesting the patient or an assistant to hold it in place. Using the fingers in the usual manner, percussion is then begun at some distance outside of the presumable limits of the organ and carried toward the stethoscope. When the outer border of the organ is attained there is a noticeable increase in intensity, an elevation in the pitch, and perhaps a slight alteration in the quality of the sound (Fig. 85). In percussing the thorax a certain amount of practice is required in order to discriminate between the superficial vibrations of its bony framework and the deeper tone emanating from the subjacent organs and tissues. Similar percussion lines are carried radially from various points encircling the organ to the stethoscope as a centre. If the exact spot on each line at which the alteration in the sound occurs is marked, and these marks subsequently joined by a continuous line, an outline of the organ will have been obtained (Fig. 86). The usual rules for regulating the strength of the percussion stroke should be followed. Directions for the employment of this method in examinations of special organs are given elsewhere (e.g., in Index—Heart, percussion of).

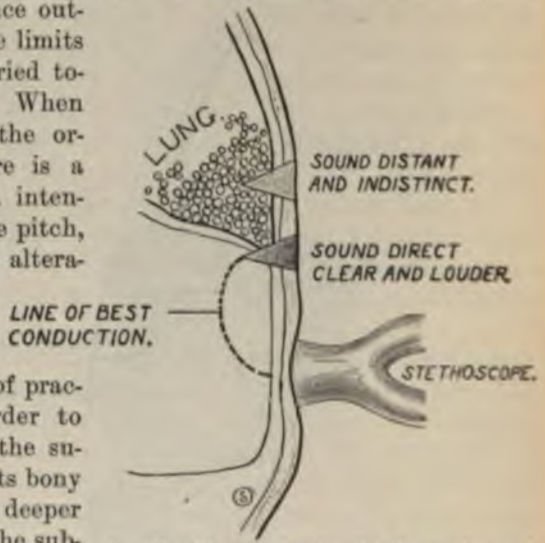

Fig. 85.—Diagram showing the theory of auscultatory percussion. The organ over which the stethoscope is placed may be either solid or hollow.

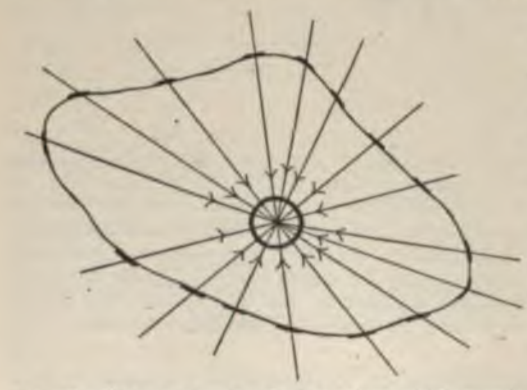

Fig. 86.—Diagram showing the lines along which auscultatory percussion should be carried in order to outline an organ. The central circle represents the chest piece of the stethoscope.

The use of the phonendoscope in auscultatory percussion has proved disappointing, as it gives no better results for this purpose than the ordinary stethoscope, and its employment in connection with stroking instead of percussion has proved futile. Since this opinion was formed it has been confirmed in published papers by Stengel, Grote, and others.

(*f*) **Sense of Resistance.**—An experienced pleximeter finger will detect differences in the degree of resistance felt while percussing. This sense of resistance is greater over fluid in the pleura or a fibroid lung than over hepatized lung tissue.

(*g*) **Palpatory and Direct Percussion.**—Like auscultatory percussion, these methods deserve a more extended use.

To perform *palpatory percussion*, the pleximeter finger of the left hand is laid firmly upon the surface and struck gently with three fingers of the right hand, which are only partly flexed and held in such a manner that the pulps of the fingers, rather than their extreme ends, touch the pleximeter finger. The distance through which the percussing fingers move should not exceed one inch, and it is essential that they remain upon the pleximeter finger for a few seconds before the stroke is repeated. The combination of pressure and percussion—i. e., a palpating stroke – is the essence of the method

Direct percussion is conducted in the same manner, except that the palpating blow is delivered upon the surface to be examined without the intervention of the pleximeter finger.

It is evident that these methods involve both the sense of hearing and the tactile sense, and that the sound produced is not of much intensity. Consequently one must listen very heedfully, with the ear close to the surface examined. By utilizing the blended impressions derived from touch and hearing, it is possible in many cases to confirm, extend, or make more accurate the results of ordinary percussion. Palpatory percussion is especially useful in connection with pleural and pulmonary disease and in determining the outlines of the liver and spleen. Because of its gentleness it may be employed over a tender or inflamed organ—e. g., in appendicitis—or over the lung in cases of pulmonary hemorrhage. See also "Threshold percussion."

III. AUSCULTATION

The restricted technical meaning of the term relates to sounds which may be perceived by direct application of the ear to the surface of the body or the use of an instrument of transmission. Such sounds are those produced in health and disease by the action of the respiratory, circulatory, and, to a limited extent, the digestive apparatuses.

Technic of Auscultation.—(1) **The Patient.**—If practicable, the patient should assume a comfortable and symmetrical posture, as a twisted or constrained attitude, either sitting or lying, will interfere with the respiratory movements and to some degree with the action of the heart. The surface to be auscultated should be bared, unless the ear is to be applied directly to the chest, in which case a handkerchief or thin, soft towel should be laid smoothly over the part. It is absurd to apply the stethoscope to a covered surface, and in the case of women patients a false sense of modesty may lead to a defective diagnosis. Women of the best social training exhibit the least false shame with reference to this point.

As a physical examination involves a certain amount of mental and physical fatigue to the patient, an excess of diagnostic zeal should be avoided in the very ill, after one thorough investigation has afforded satisfactory results.

(2) **The Methods.**—Auscultation may be direct (immediate), the ear being applied directly to the surface; or indirect (mediate), by the use of a stethoscope.

Auscultation without an instrument is useful in emergencies, and in getting a general idea of the sounds which are present. Some accomplished auscultators claim, moreover, that by it one may hear certain râles and cardiac murmurs more readily, and perceive an aneurismal bruit and thrill with greater distinctness, than with the stethoscope; and, furthermore, that it is easier by this method to determine the relative nearness or superficiality of friction sounds and râles. As a rule, however, it may be said that the direct method compares unfavourably with the stethoscopic examination in accuracy of localization, refinement of discrimination, and avoidance of unnecessary personal contact. The practical lesson from these differing opinions is that one should be trained to the use of both methods in order to avail one's self of either in a doubtful case.

In the United States the double (binaural) hollow stethoscope with flexible tubes is almost universally employed. It is highly desirable that the examiner should become accustomed to some one of the many patterns of the instrument, as better work is possible with a familiar and well-used tool. In choosing a stethoscope much care should be exercised, and a selection should be made by comparison between several kinds with reference to the following points:

First.—Its capacity for transmitting sounds. This is best done by laying a watch upon the thigh and placing the metacarpal portion of both hands, one over the other, palms down, upon the watch, and applying the chest piece of the instrument to the dorsal surface of the upper hand. It is usually easy to decide by listening to the ticking of the watch which instrument conducts sound to the best advantage.

Second.—The ear pieces must fit comfortably in the external meatus, and almost entirely exclude extraneous sounds.

Third.—Durability, simplicity, and convenience in carrying should be considered.

Fourth.—The chest piece for ordinary use should not exceed 1¼ inch in diameter. A larger rigid piece and one of soft rubber, the latter for use in much emaciated patients, should be added.

In applying the stethoscope to the surface its mouth should be held easily between the thumb and forefinger, and in some cases it may be advantageously steadied by resting the little finger of the same hand upon the surface of the body. The mouth of the instrument should be in firm contact at all points of its circumference in order to isolate the columns of air in the tubes, and to shut off outside noises. If the ribs are prominent and close contact can not be obtained, the skin may be gathered in a small mass by the fingers of the other hand, thus furnishing a sufficient amount of tissue to secure the desired apposition, or the soft-rubber chest piece may be used. In some cases the skin is so harsh and dry that confusing friction sounds occur with the respiratory or other movements of the surface examined. This may be readily obviated by wetting or, better, oiling the skin.

The *phonendoscope*, in personal use, has proved to have no advantages over the ordinary stethoscope in auscultation of the lungs, but it has certain points of usefulness in auscultation of the heart and vessels. By its power of intensifying sound one is able to detect very faint murmurs or bruits, slight clicking or harshness of the valve sounds, and the quality of an extremely weak first sound with greater facility than by the ordinary means. One practical advantage in the hurry of office work is the rapid determination of the presence of coarse cardiac murmurs and venous humming without removing the clothing, as such sounds, even if of moderate intensity, are readily heard with this instrument through several thicknesses of fabric.

The *differential stethoscope*, an instrument with two chest pieces and separate flexible tubes, one leading to the right, the other to the left ear, is very useful in comparing the time relations of two sounds heard at different portions of the chest. By placing the two chest pieces, one over each of the separate localities, the sounds from each area are heard simultaneously, and it is possible to determine very accurately which precedes the other, or to detect differences in their quality—e. g., two systolic murmurs at different orifices.

In view of the difficulty often experienced in auscultating the lungs posteriorly in those who are too ill to sit up or even to undergo the fatigue incident to being rolled over from one side to the other, a stethoscopic chest piece has been devised (Smith) which can be slipped under the back without disturbing the patient. It is shaped like a flattened disk with the tubes passing off from a point on its circumference.

SECTION XXIX

THE CHEST (THORAX)

Included here are certain points observed by inspection, partly also by palpation and mensuration, of the thorax. The importance of inspection and palpation of the thorax can not be too strongly emphasized. It is frequently the case that the examiner proceeds at once to auscultation and percussion of the chest, thereby neglecting to obtain valuable diagnostic evidence.

Method of Inspection.—The thorax should be bared, and the patient placed in a symmetrical and comfortable posture, whether sitting, standing, or lying. The light should, as a rule, fall directly on the surface which is to be examined. Altering the direction from which the light comes, either by moving the patient or the source of the illumination, so as to permit it to fall obliquely across the examined surface, is, by casting shadows, of much use in detecting slight pulsations or abnormalities of shape and movement. In inspecting the thorax the examiner should view its anterior, lateral, and posterior aspects. Finally, he should never omit to look down upon it from above and behind the patient. This view point gives practically the outline of a horizontal section of the chest, and is very useful in determining the amount and any inequality of expansion; or the presence of lateral curvature, projection of one or both scapulæ, or differences in the size of the lateral halves of the chest.

The Normal Thorax.—Absolute bilateral symmetry is extremely seldom found. In most cases there is a slight right lateral curvature, and the right side of the chest is usually a little the more capacious. A recognition of the normal shape of the chest depends upon the familiarity gained by clinical experience.

The bony cage of the thorax divested of the shoulder girdle is conical in shape, the smaller end upward, but in its clinical state, especially in muscular persons, the circumference at the level of the axilla is greater than at the lower end of the sternum, because of the presence of the structures mentioned. In the adult a horizontal section of the chest shows its transverse diameter to be greater than the antero-posterior, in the proportion of 10 to 7.5 (E. H. OTIS). In children it is more nearly circular.

In the *normal thorax*, in addition to its nearly symmetrical contour, it may be noted—

(1) That the clavicles may or may not be somewhat prominent, especially the right.

(2) That there may be slight depressions above and below the clavicles. The depression between the deltoid and greater pectoral muscles below the clavicle is Mohrenheim's fossa.

(3) That there is a depression just above the upper end of the sternum—the episternal notch—between the inner ends of the clavicles.

(4) That the line of junction between the first piece of the sternum (manubrium) and the second (gladiolus) forms a projection—the angle of Louis (angulus Ludovici), which may frequently be seen and almost invariably felt. It constitutes a valuable guide to the second rib, the cartilage of this rib articulating at the line of junction.

(5) That the true ribs are so shaped and attached to the sternum that the upper ribs run horizontally outward, the others passing downward and outward with increasing obliquity until the subcostal or epigastric angle formed by the divergence of the costal margins and the lower end of the sternum is usually 70° or 80°. The ribs, except in thin or ill-developed individuals, are only visible laterally and in the lower third of the thorax.

(6) That at the lower end of the sternum there is the infrasternal depression (*scrobiculus cordis*, pit of the stomach).

Thoracometry.—Measurement of the chest is done with reference to its circumference, semi-circumference, and main diameters, by means of the tape measure and the calipers.

(1) The *circumference* of the chest is usually measured at the line of the nipples, taking care that the tape passes around in a horizontal line. The average circumference in men is 34.3 inches (OTIS, HITCHCOCK); in women, 29.5 inches (MISS WOOD, MARY COTTON). An important use of the tape line is as a stethometer to determine the extent of respiratory expansion—the difference in the girth of the chest at the end of forced expiration and forced inspiration respectively. Special stethometers with graduated dials are unnecessary. Expansion varies normally from 1½ to 5 inches. The significance of variations in amount will be discussed in connection with the examination of the lungs (*q. v.*).

(2) The *semi-circumference* of the chest may be determined by making a vertical mark in the median line of the sternum, and another on the spinous process of the vertebræ at the level of the nipples. This may be done with a dermatographic pencil or a stick of dark grease paint (removed by wiping with a vaselined rag). The tape is passed from one mark to another, first around the right half, then around the left half of the chest. In right-handed persons the right side of the chest frequently exceeds the left in circumference by half an inch.

To measure the expansion of the right and left halves of the chest separately, it is convenient to have two tapes joined so that the beginning of each is in the middle of their combined length. The line of junction is placed over the median line of the spine at the proper level and steadied by the examiner's finger, while the tapes are brought around to the front and held by the patient with proper directions as to tension. The expansion of each side during inspiration can be then readily noted. It also serves to measure simultaneously the semi-circumferences of the chest.

(3) The *diameters of the chest* are measured at the level of the nipples by means of compass calipers with curved arms or by slide calipers, the use of which does not require special directions. The average *depth of the chest*—the antero-posterior diameter—is 8.0 inches in men (L. Brown), 6.9 inches in women (Wood). The average *breadth of the chest*—the transverse diameter—in men is 11 inches.

Cyrtometry.—The determination of the shape of the chest consists practically in obtaining an outline of its transverse section at any desired level, usually that of the nipples.

Several varieties of cyrtometers have been devised to attain this end. Some, as with those of Démény, of Paris, and Evans, of Brooklyn, are very accurate, but too elaborate and expensive for ordinary use. A practicable instrument consists of a compass with short arms to which are attached narrow strips of lead easily bent and yet able to retain their shape under ordinary circumstances. An indicator and set screw enables the arms to be fixed at any angle, opened, and set again at the same point. The arms are set to lie on either side of the spine and the metal strips brought forward from either side and moulded to the chest until the ends meet or cross over the sternum. The compass arms are then loosened, opened, the instrument removed and closed again to the same angle. It is then laid on a sheet of paper, the spinal and sternal points marked and an outline of the inner borders of the strips made with a soft pencil. For clinical purposes two bands of lead connected by rubber tubing will amply suffice. In many cases the size and shape of the chest may be ascertained after sufficient experience by inspection alone, but for accuracy, for purposes of record, and in order to detect slight differences the instrumental methods may be carried out.

Bilateral Deformities of the Thorax.—These deformities, both general and local, are as follows:

(1) **The Flat Chest.**—After many measurements Woods Hutchinson affirms that the normal chest is flat; that the apparently flat "tuberculous" chest is in reality rounder than the normal, i. e., the antero-posterior diameter, relatively to the transverse diameter, is increased, and that this form of chest precedes phthisis. Lawrason Brown, traversing the same subject, states that phthisis tends to reduce both diameters, and that the chest may become either flat, or deep and round.

(2) **The Pterygoid or Alar Chest.**—A long neck, prominent larynx, sloping shoulders, scapulæ projecting like wings (hence the name), great obliquity of the ribs, which dip sharply downward from the sternum and then bend sharply upward and backward to the spine, thus making a very acute subcostal (epigastric) angle, wide intercostal spaces, and a thorax, as a whole, vertically long and narrow, constitutes the chest variously termed phthisical, phthisinoid, or paralytic. It is often also a flat chest. It indicates a tendency to pulmonary phthisis, and if well marked is evidence of the actual presence of the disease. Extreme emaciation of the chest, without a real change of shape, such as may be found after a long fever, should not be mistaken for this form of chest.

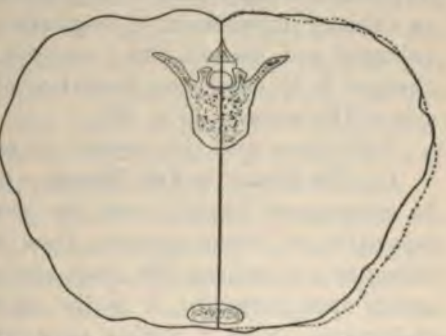

Fig. 87.—Cyrtometer curve of an emphysematous chest in a male, aged seventy-four. Dotted line shows a slight difference in the size of the right and left sides of the thorax. Redrawn from Evans.

(3) **The Emphysematous Chest.**—If the antero-posterior diameter of the chest is increased, if it appears broad and short, if the sternum is arched, if the ribs are thick and run horizontally outward, making the subcostal angle unusually wide, and the angulus L. is notably prominent, it is almost certain evidence of hypertrophic pulmonary emphysema (*q. v.*). The enlargement of the lungs in this disease causes a general expansion of the chest (Fig. 87), so that it presents a permanent inspiratory position. In many instances

the middle portion of the thorax bulges outward, giving rise to the "barrel-shaped" chest. Like phthisis and the phthisical chest, emphysema, particularly the atrophic form, may exist without causing a characteristic chest shape. It should be remembered that kyphosis may simulate an emphysematous chest.

(4) **The Rachitic Chest.**—In this deformity the ribs immediately external to the sternum are bent upward during early life, because of their softness, the lessening of the intrathoracic pressure during inspiration being sufficient to produce the change of shape without an existing impediment to respiration. The ends of the ribs are also enlarged and beaded, the "rachitic rosary." The result of these changes is to cause the formation of a vertical depression on either side of the sternum (Fig. 88).

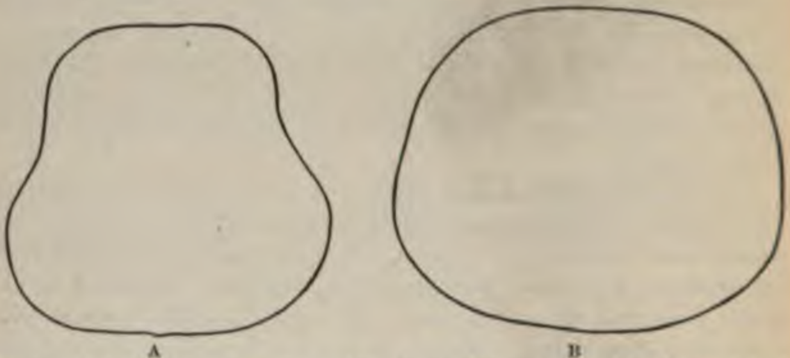

FIG. 88.—A, horizontal section of a rachitic chest, child two years old, showing lateral furrows; B, section of chest of healthy child of the same age (Holt).

Very often with this coexist (6) and (7), to be described.

(5) **The Pigeon or Keel Breast.**—In this form the chest appears to be compressed laterally and the sternum pressed sharply forward, especially its lower portion, thus increasing the antero-posterior diameter and making the cross section of the chest distinctly triangular, apex forward. It is the result of rickets plus respiratory obstruction (even if slight) from nasal catarrh, adenoids, enlarged faucial tonsils, and sometimes prolonged pertussis.

(6) **Harrison's Sulcus.**—This is a zonal constriction beginning at the sterno-xiphoid junction, extending outward and somewhat downward to the axillary line. It corresponds to the line of attachment of the diaphragm, and, as the ribs form the fixed point for the action of the diaphragm, if they are softened by rachitis they are bent inward along this line. The causes are the same as for pigeon breast (5), and the latter almost always presents Harrison's sulcus.

A flaring outward of the entire costal margin on both sides, causing the lower opening of the thorax to be expanded, is not abnormal in some individuals, but may occur acutely, especially in children, from the upward and outward pressure of great tympanites; or slowly from ascites and large abdominal tumours.

(7) **Funnel Chest.**—This is a more or less deep depression or foveation of the lower sternum (improperly termed funnel *breast*), which may extend upward as high as the third rib. It is usually congenital and of no importance, but if marked may interfere with respiration. It may be simply a stigma of degeneration. A similar depression of the ensiform appendix and lower end of the sternum may be due to the pressure of tools, especially in cobblers.

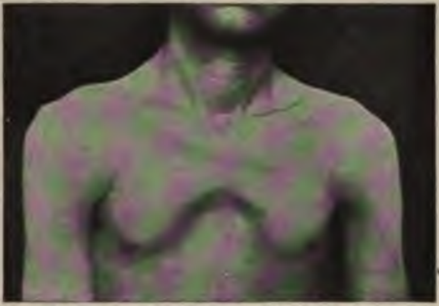

FIG. 89.—Funnel chest (M. E. H. case).

Unilateral and Local Thoracic Deformities.—(1) *Unilateral enlargement* or bulging of the thorax, as determined by inspection or measurement, is caused by gas or fluid in one pleural cavity, as in pyothorax, hæmothorax, pneumothorax, and pleurisy with effusion. The increased size may be due to compensatory emphysema caused by disease of the opposite lung—*e. g.*, fibroid changes. In this case the diseased side is smaller and the opposite side larger than normal, thus making the disparity more noticeable. A tumour of the lung or pleura may also be considered as a possible cause of one-sided enlargement. Bulging of the intercostal spaces is always present in enlargement of the chest, but may exist alone as evidence of the same conditions.

(2) *Local prominences* may be found in various portions of the thorax. Præcordial bulging is usually significant of an enlarged heart, large pericardial effusion, pneumopericardium, or a mediastinal tumour or aneurism pushing the heart bodily forward. As the existence and extent of this bulging depends upon the degree of plasticity of the chest wall, it is most marked in children and young adults. While it occurs, as a rule, gradually, there may be an acute bulging in young subjects from a rapid and large pericardial effusion. Bulging of the right hypochondrium may signify enlargement of the liver, hepatic abscess or hydatids, subphrenic abscess, or effusion into the right pleura. It may be present as part of a marked distention of the right side of the abdomen resulting from tumours on the same side of the median line—*e. g.*, sarcoma of the kidney.

Other causes of localized swellings or prominences of the chest are:

Aneurisms (*q. v.*); knuckling or irregular formation of one or more ribs or costal cartilages, of congenital or infantile origin, which by a careless examiner may be mistakenly referred to aneurism or cardiac disease; localized emphysema, or encysted fluid in the pleura, which, however, is apt to cause obliteration of the intercostal spaces over a limited space rather than a distinct prominence; general emphysema, which may give rise to prominence of the supraclavicular spaces; collections of pus due to disease of vertebræ, sternum, ribs, soft tissues of the chest wall, or actinomycosis, perforating empyema, mediastinal abscess, and subphrenic abscess; and hydatids or tumours of the lung, pleural growths, and, very rarely, hernia of the lung.

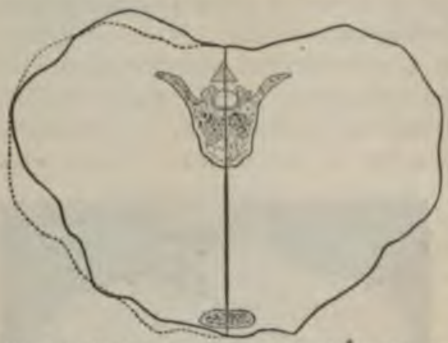

FIG. 90.—Cyrtometer curve of the thorax (at the level of the 4th rib) in tubercular infiltration of the upper and middle lobes of the right lung. The dotted line shows the difference in the size of the right and left sides of the thorax. Redrawn from Evans.

(3) *Unilateral contraction* of the thorax, one side of the chest being more or less evenly shrunken, owing to lessened size of the lung (Fig. 90), may be significant of chronic phthisis, interstitial pneumonia, extensive pleuritic adhesions, collapse of the lung from a foreign body in the bronchus, or occlusion of a bronchus by mediastinal tumour or abscess. It may follow long-continued pressure upon the lung itself by pleural effusions. The retracted side is obviously smaller than the other, the shoulder of the same side droops, the spine is curved with its concavity toward the diseased side, and the ribs approach each other or may nearly overlap. These changes, due to disease of the thoracic viscera, are somewhat similar to those of scoliosis (*q. v.*) arising from other causes (Fig. 91).

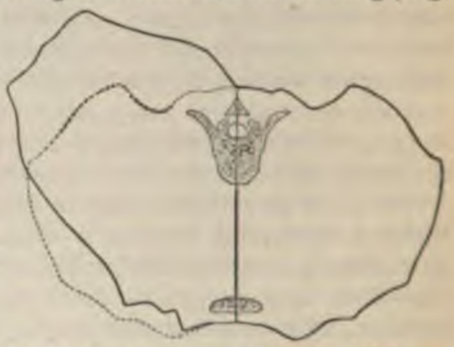

FIG. 91.—Cyrtometer curve of chest in lateral curvature of the spine (scoliosis). Dotted line shows the normal outline. Spine and sternum in normal relations. Redrawn from Evans.

(4) *Local depressions* of the thorax are seen above and below the clavicles, especially in the space between the deltoid and pectoral muscles, in phthisis. Flattenings or depressions in other parts of the chest may indicate the existence of bronchiectatic or phthisical cavities, localized pleuritic adhesions or old fracture of the ribs. Atrophy or removal of one breast or atrophy of one great pectoral muscle gives rise to flattening of the corresponding side.

Miscellaneous.—The *flexibility of the ribs* and their cartilages should always be tested by pressure upon the sternum. It is greatest in children and progressively decreases as age advances. The decrease is partly due to changes in the osseous tissue of the ribs, but largely also to partial calcification of the cartilages. Increased rigidity of the bony cage of the thorax renders it a better resonator and conductor of sound. Especially in thin persons, it may increase the intensity of the percussion sound, and in some cases give rise to a deceptive transference of auscultatory phenomena beyond the expected limits—e. g., the hearing of bronchial breathing for a short distance to the *left* of the spinal column, the sound being transmitted from a consolidated *right* lower lobe along the rigid ribs and vertebræ. On the other hand, the extreme flexibility of the ribs in infants is partly responsible for the cracked-pot percussion sound, and perhaps also for some of the obscurities in the differential diagnosis between pleural effusion and pulmonary consolidation in the very young.

Œdema of the thorax may be a part of general dropsy; or, if localized, a symptom of a deep-seated abscess of the chest walls or an empyema preparing to perforate.

Enlarged veins of the chest are seen in the neighbourhood of the mammary glands during lactation and are sometimes significant of malignant disease of the breast. In an unusually fair skin they may be extremely but normally conspicuous. They may result from the interference to the return of blood from the breast caused by the pressure of mediastinal tumours and thoracic aneurism. Portal obstruction, by compelling the blood to retrace its way through collateral paths, and right ventricular dilatation, by damming it back, will also give rise to abnormal distention. An arched line of dilated capillaries corresponding to the attachments of the diaphragm along the lower costal margin is not infrequently noted where the right ventricle is hard worked.

Respiratory movements of the chest (see Index—Respiration).

Pulsations in the thorax, visible or palpable (see Index—Thorax, pulsating areas of).

Pain in the chest (see Index).

SECTION XXX

ANATOMICAL LANDMARKS AND TOPOGRAPHICAL AREAS OF THE THORAX

For purposes of description, and to conduct a proper physical examination of the thoracic contents, it is necessary to be familiar with certain anatomical landmarks and arbitrarily fixed surface areas of this portion of the body.

Anatomical Landmarks of the Thorax.—(1) **Sternum.**—The average length of the sternum is 6 inches. The upper border of the sternum, the episternal notch, which can always be seen and felt, is on a level with the disk between the 2d and 3d dorsal vertebræ. The distance between the disk and the notch is 2 inches.

Running the finger downward from the episternal notch, a transverse ridge may be felt and often seen, the angle of Louis (Ludovici). It is better marked in the male thorax and is on a level with the lower border of the body of the 5th dorsal vertebra.

At the lower end of the sternum identify the ensiform (xiphoid) appendix and its junction with the gladiolus (body or corpus) of the sternum. This is often difficult because of the chondro-xiphoid ligaments which pass from the 7th cartilage to the appendix. The appendix varies in shape and size. Its point is often curved forward and its anterior surface hollowed out, forming a marked infrasternal fossa or depression. The sterno-xiphoid junction is on a level with the disk between the 9th and 10th vertebræ.

(2) **Ribs and Interspaces.**—Every physical examination of the chest requires identification of the ribs. The best routine method is to find the angle of Louis; then run the finger along its ridge to the right or left, when it will pass directly upon the 2d rib, from which the ribs may be counted in a line downward and outward, tracing those desired into the axilla and posteriorly, remembering that each rib runs upward as it passes to the spine. Furthermore, if the arm is raised outward to a horizontal line, the lower border of the great pectoral muscle corresponds to the 5th rib, and the highest digitation of the serratus magnus lies over the 6th rib, the next 2 below lying over the 7th and 8th ribs respectively.

Owing to the downward slope of the ribs from spine to sternum, the chondro-sternal articulation of each rib is considerably lower than its vertebral articulation. The articulation of the 1st rib in front is on a level with that of the 4th rib at the back. The corresponding relations of the 2d to the 7th ribs inclusive may be easily stated by adding 4 to the number of the rib in front—e. g., the 3d rib anteriorly is on a level with the 7th posteriorly, 6th with the 10th, etc.

Posteriorly the ribs may be counted upward, starting with the 12th, which can usually be felt, but, in fat persons, sometimes only with great difficulty. The tips of the spinous processes of the dorsal vertebræ may also act as guides to the ribs. Owing to their downward inclination, the dorsal spines do not all lie on a level with the same numbered rib. Thus the 2d dorsal spine corresponds to the 3d rib, the 3d spine to the 4th rib, and so on, down to and including the 9th spine, which corresponds to the 10th rib. The 10th spine lies midway between the 10th and 11th ribs. The 11th and 12th spines correspond to the 11th and 12th ribs.

The intercostal spaces have the same number as that of the ribs below which they lie. They are wider in front than behind, and the 3d is usually the widest, next the 2d, then the 1st. The identification of the ribs identifies the interspaces, but after some experience the examiner is often able to recognise at once the 1st and 2d interspaces. There is not infrequently a noticeable gap between the clavicle and 1st rib, which may be readily taken for the 1st interspace, and in a long thorax the 2d interspace may be surprisingly low down.

(3) **Nipple.**—In the male thorax this is usually between the 4th and 5th ribs, 4 inches from the median line of the sternum, but it may lie directly upon either of these ribs, or in some few cases in the 5th interspace. In the female chest its position is extremely variable, depending on the size and pendulousness of the mammary gland. One learns to recognise with some accuracy the point where it should typically be found.

(4) **The Mammary Gland.**—Vertically this gland extends from the 3d to the 6th (or 7th) ribs inclusive, and horizontally from the edge of the sternum to the anterior border of the axilla.

(5) **The Spine and Back.**—There usually is a median groove or furrow in the back, at the bottom of which lie the spinous processes. In thin persons and in many children the spinous line is quite prominent, largely replacing the groove. In order to palpate and identify the spines, the patient should be made to double over to the front, thus giving the spinal column a curve with its convexity to the rear, and making the spines more prominent. Or the spine may be briskly rubbed up and down, by which device the tip of each spinous process is capped by a spot of hyperæmic redness.

If searching for or determining a given spine, the following points are to be considered: Just under the occiput the spine of the axis may be felt. The spines of the 3d, 4th, and 5th cervical vertebræ are usually distinguishable only as a line or ridge. The 6th spine may frequently be felt, but the 7th cervical, as its name (prominens) indicates, is an unmistakable landmark and from it one may count downward. Or, if the number of a given rib is known the number of the spine on a level with it may be inferred, conversely as in (2).

In a patient sitting with the arms hanging easily at the sides the scapula covers the ribs from the 2d to the 7th (sometimes from the 3d to the 8th) inclusive. In the same position the inner end of the spine of the scapula is on a level with the 3d dorsal spine, and the inferior angle of the scapula corresponds to the 7th dorsal spine.

Topographical Areas of the Thorax.—Certain arbitrary lines, vertical and horizontal, are conceived to be drawn upon the front, side, and back of the thorax, thereby mapping it into regions or areas which are convenient for purposes of description or record (Figs. 92 and 93).

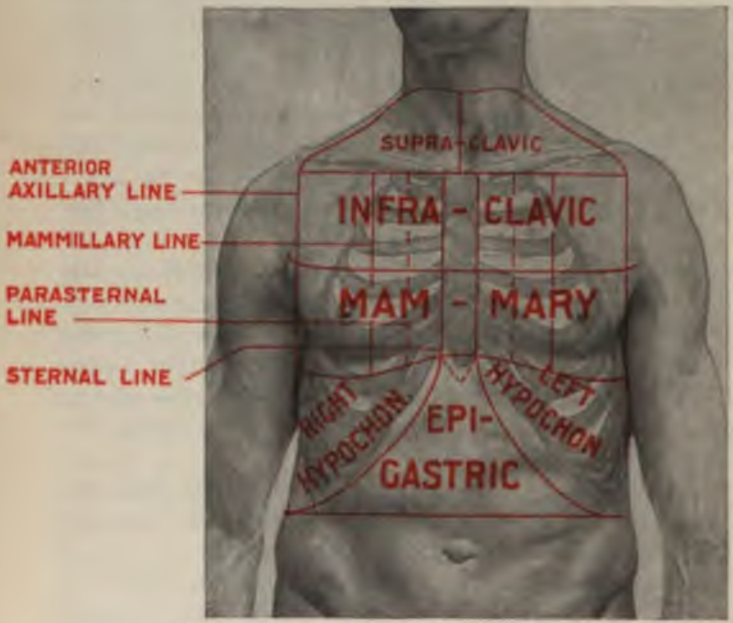

FIG. 92.—Showing the topographical areas of the thorax anteriorly.

The **vertical lines** are from front to back.

(1) The *midsternal line* and its prolongation upward.

(2) The *sternal line*, corresponding to the lateral border of the sternum.

(3) The *parasternal line*, midway between (2) and

(4) The *mammillary* (or nipple) *line*, which, even in the male thorax, does not always pass through the nipple, but may be more exactly defined as a vertical line dropped from the centre of the clavicle.

(5) The *anterior axillary line*, drawn through the point at which the great pectoral muscle leaves the chest when the arm is raised sidewise to a horizontal line.

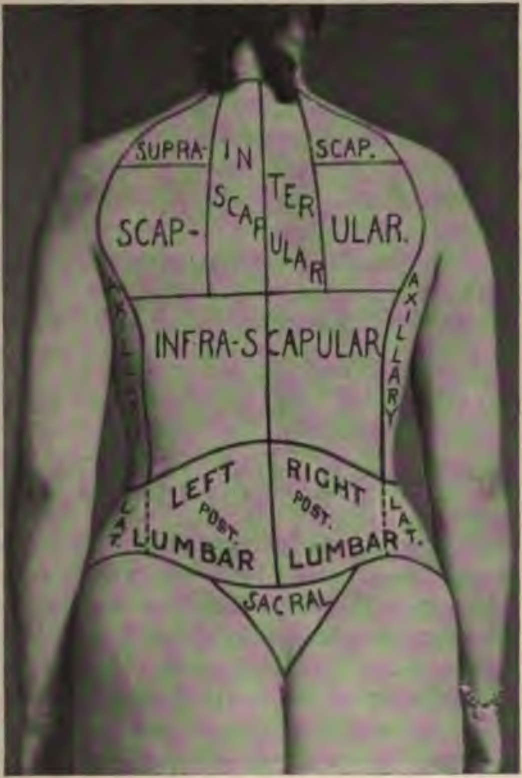

FIG. 93.—Showing the topographical areas of the trunk, posterior aspect.

(6) The *middle axillary line*, drawn through midway between (5) and

(7) The *posterior axillary line*, which is drawn through the point at which the latissimus dorsi leaves the chest, the arm being raised as in (5).

(8) The *scapular line*, drawn through the inferior angle of the scapula.

(9) The *midspinal line*.

The **horizontal** lines are, in front and at the side, from above downward:

(1) A line running from the cricoid cartilage to the outer end of the clavicle.

(2) The line of the clavicles.

(3) A line through the third chondro-sternal articulation.

(4) A line through the sixth chondro-sternal articulation. Posteriorly they are:

(5) A line through the spines of the scapulæ.

(6) A line through the inferior angles of the scapulæ.

(7) A line through the spine of the twelfth dorsal vertebra.

Describing the Site of Lesions in the Thorax.—(1) One method is simply to state the area in which a given sign or condition is found. This is of value in giving a somewhat general idea of locality.

(2) If it is desired to define an exact point, it is customary, *in front*, to state the number of the rib or interspace, and the distance, in inches or centimetres, either from the midsternal or lateral sternal line at which it lies; *laterally*, to give the number of the rib and the relation of the point to the anterior, middle, or posterior axillary lines; *posteriorly*, to record the rib or interspace, and the distance of the point from the midspinal line, *or*

(3) To state the number of the rib or interspace and the vertical line (e. g., parasternal, anterior axillary) at which the condition to be noted is found.

SECTION XXXI

EXAMINATION OF THE CIRCULATORY SYSTEM

Before proceeding to consider the evidences of disease which may be obtained by an examination of the heart and blood-vessels, it is desirable to outline the clinical physiology of these organs.

I. PHYSIOLOGY OF THE HEART

Myogenic Origin of the Heart Action.—The myogenic theory of heart activity can be regarded as resting upon a fairly firm foundation. Although there are some facts not yet fitted into place, this hypothesis has made possible many advances in cardiac pathology and diagnosis. It is as follows:

Properties of the Heart Muscle.—The heart muscle exhibits *five* distinct and inherent properties, each capable of varying, quite independently, in its degree of activity. These properties are:

First, the power of generating *automatic rhythmic stimuli*, which under normal circumstances originate in the venous ostia of the *right* heart, whence they pass downward to the ventricles, *via* the muscle-bundle of His, by virtue of

Second, the *conductivity* of the cardiac muscle. Thus the rhythmic stimulus travels from its point of origin, not by way of the cardiac nerves, but along the muscle cells and by reason of their continuity. Through the discovery of these facts the nervous apparatus of the heart has been relegated to a place of minor importance as a regulator, not an originator, of cardiac activity. The "auriculo-ventricular bundle" of His is a slender band of muscular tissue 18 mm. long, 2.5 mm. broad, and 1.5 mm. thick. It extends from the right side of the interauricular septum to the interventricular septum, and, splitting, is distributed on the right and left sides of the latter. It is this apparently insignificant muscle-band which constitutes the pathway of the contraction-stimuli.

Finally, the remaining three properties of the cardiac musculature are those common to all muscular tissue—*i. e., irritability, contractility*, and *tonicity*.

Modifications of these Properties.—Stimulus production, conductivity, and contractility are, normally, depressed during systole and increased during diastole. Stimuli may originate at other points than in the right auricle, but, except in disease, when the normal direction of conduction may even be reversed, they do not influence the rhythm of the heart. The rate of conduction varies in different parts of the heart, thus promoting a more effectual ventricular contraction. Conductivity is depressed by asphyxia. The presence of certain inorganic salts seems necessary for the production of rhythmic stimuli. The circulating blood may contain poisons which increase or depress the functions of the heart muscle. Stimulation of the pneumogastric depresses, of the accelerator nerve increases, the production of rhythmic stimuli, the rate of conduction, and the contractility.

General Scheme of the Action of the Heart.—During systole the blood is injected into the aorta and pulmonary arteries only, the auriculo-ventricular openings being closed each by a valve and the arterial valves open. When the ventricles relax and their cavities dilate the blood flows into them through the auricles only, the arterial valves, previously open, having closed; and the auriculo-ventricular valves previously closed, having opened.

During the greater part of the ventricular relaxation or diastole the auricles also are relaxed, and the heart as a whole is at rest. The blood is flowing quietly through the auricles into the ventricles, thus tending to fill and distend all four cavities.

Toward the termination of the ventricular repose or diastole the auricles contract (auricular systole), completing the full blood-charge of the ventricles. The auricular systole is brief, and terminates immediately before the ventricular systole commences. Thus the auricular diastole begins with and continues through the ventricular systole, and nearly to the end of the ventricular diastole. During this relatively long period blood is entering the auricles from the veins.

The Cardiac Cycle.—The "cardiac cycle" (CURTIS) comprises all events, auricular and ventricular, which occur during one complete auricular cycle (systole and diastole), as seen in Fig. 94, embracing the two essential facts of heart activity and heart repose.

The duration of the cardiac cycle, when the pulse rate is at the normal average of 72 per minute, is 0.8 second. The auricular systole lasts 0.1 second, the ventricular systole 0.3 second, and the period of repose 0.4 second. Clinically it should be borne in mind that with a rapid pulse the diastole is greatly shortened, rather than the systole.

Action of the Valves.—During the diastole of the ventricles the curtains of the auriculo-ventricular (mitral and tricuspid) valves hang free in their respective cavities, probably not closely touching the walls. As the blood flows quietly in from the auricle the ventricle fills, and currents pass between the valve-curtains and the ventricular walls, floating the curtains upward until their edges and a part of their surfaces are almost in contact. The auricle then contracts, completely filling the ventricle, and the systole of the latter immediately follows, causing an instantaneous increase in the ventricular blood-pressure. The increased pressure at once brings the edges and a part of the surfaces of the valve-curtains (which are already in close proximity) so suddenly and neatly together that no regurgitation is permitted. The apposed free edges of the curtains are thin and delicate, but when in contact are supported by the opposite blood-pressure. That portion of the segment between the free and the attached edge is prevented from bulging into the ventricle by the fine but strong chordæ tendineæ which spring from the papillary muscles and act as guys to the whole surface of the segments.

The segments of the *semilunar* (aortic and pulmonary) valves also have thin and flexible edges which, with a portion of their ventricular surfaces (lunulæ), are held in apposition by the blood-pressure in the artery at the close of ventricular systole. The remainder of the segment is thick and strong, and is partly supported when closed by the top of the thick ventricular walls. Finally, the presence of the Corpora Arantii at the central point of the junction completes the barrier to the back-flow of blood.

The Normal Heart Sounds.—Upon auscultation each cardiac cycle is found to be attended by two sounds or tones, first and second, differing in character and relation to the events of the cycle.

The *first sound* begins with the ventricular systole (Fig. 94), but while of somewhat prolonged duration does not last throughout the systole, nor does it terminate very abruptly. For convenience' sake it may be said to resemble the syllable "lubb." The first sound, with reference to its mode of production, is unquestionably composed of two elements, valvular and muscular. The valvular element is the short flapping closure of the mitral and tricuspid valve segments. The muscular element is the sound, more prolonged and booming in quality, which is produced by the contraction of muscle fibres and bundles. In fevers, when the heart muscle is weakened, the first sound loses its booming quality and becomes short and flapping in character, very like the second sound to be presently described. Indeed, this change in the first sound is one of the best clinical evidences of the weakened heart.

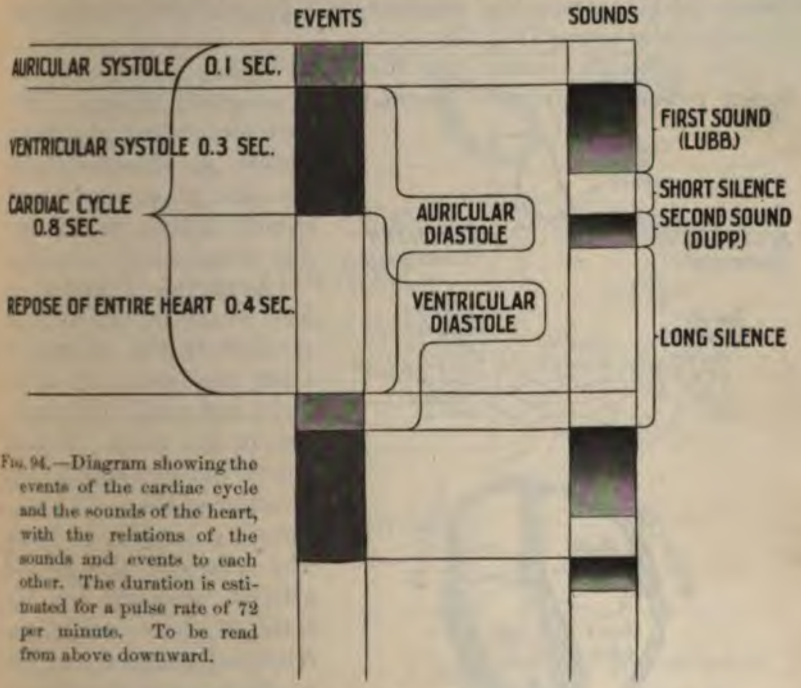

Fig. 94.—Diagram showing the events of the cardiac cycle and the sounds of the heart, with the relations of the sounds and events to each other. The duration is estimated for a pulse rate of 72 per minute. To be read from above downward.

After the first sound there is a very brief interval, the *first*, or *short*, *silence*, followed by the second sound.

The *second sound* coincides with the end of the ventricular systole, is purely valvular in character, and is due to the sudden closure of the aortic and pulmonary valve segments. It is short, sharp, and terminates abruptly by comparison with the first sound. Conventionally it is represented by the syllable "dupp."

The second sound is followed by the *second*, or *long*, *silence*, which continues until the first sound is again heard—e. g., lubb-dupp—lubb-dupp—etc.

Comparing the relative intensity (accentuation) of the two sounds under normal conditions, it will be found that when the heart is auscultated at the apex beat, the first sound is the nearer and more intense (lúbb-dupp), while, if listened to in the second interspace on either side of the sternum, the second sound is accented (lubb-dúpp).

Innervation of the Heart.—The nerve supply of the heart consists of (1) intracardiac ganglion cells, (2) inhibitory nerves, and (3) accelerator nerves (Fig. 95).

It is well-nigh certain that the heart muscle possesses an inherent power of rhythmic contractility (myogenic theory). The part played by the nervous system is, therefore, simply that of a regulator of stimuli arising in the cardiac muscle.

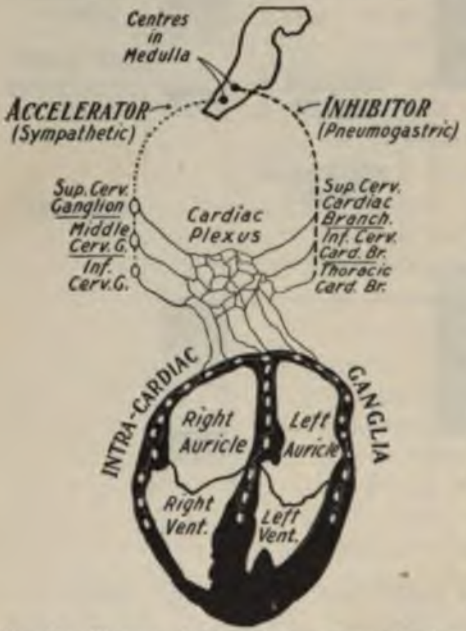

FIG. 95.—Diagram showing the nervous mechanism of the heart. Section of heart after Gegenbauer.

Arterial Tension.—The blood is under high pressure in the arteries, a lower pressure in the capillaries, and a still lower pressure in the veins. It thus moves continuously in the direction of the lowest pressure—i. e., arteries to veins. The causes of the normal arterial pressure are the force of the ventricle, the frictional resistance of the capillaries, and the elasticity of the arteries.

Thus connecting the cardiac pump—the force—with the capillaries—the resistance—is a system of tubes, the arteries, which are elastic and distensible (Fig. 96).

The *arterial tension* or pressure, therefore, is the resultant of the intermittent pumping of fluid into an elastic tube which at its further end is split into many fine tubes, offering a steady resistance to the outflow. The normal degree of pressure is maintained when the amount of blood accommodated by the yielding of the arterial walls during each systole of the ventricle is equal to the amount passed into the capillaries during the following diastole of the ventricle.

Changes in the arterial pressure are largely dependent either upon variation in the strength of the heart beat, or in the resistance, or in both. If the heart is weak, or the resistance lessened by dilatation or loss of tone in the arterioles, the pressure falls; while if the heart contracts with great vigour, or the vessels are contracted, it will rise.

Innervation of the Blood Vessels.—While the blood is kept in motion by the heart, its distribution to various portions of the body is regulated by the blood vessels. This regulation of the blood supply to various areas depends upon the existence of circular muscular fibres in the middle coat of the arteries, particularly those of small size (arterioles), and a nervous mechanism (the vasomotor apparatus) which, by its action upon the muscular coat, controls the calibre of the supplying vessels, and in consequence the amount of blood permitted to pass through them in a given space of time. The vasomotor apparatus (Fig. 97) consists of vaso-constrictor and vaso-dilator nerves, with centres in the cord and in the medulla. The *vaso-constrictor nerves*, when stimulated, cause contraction of the vessels to which they are distributed; if cut, the vessels dilate. The *vaso-dilator nerves*, if stimulated, give rise to enlargement or dilatation, but section of these nerves does not cause vascular constriction. The dilators are more easily excited than the constrictors. It is to be borne in mind that both dilator and constrictor fibres may run side by side in the same anatomical nerve—e. g., the sciatic.

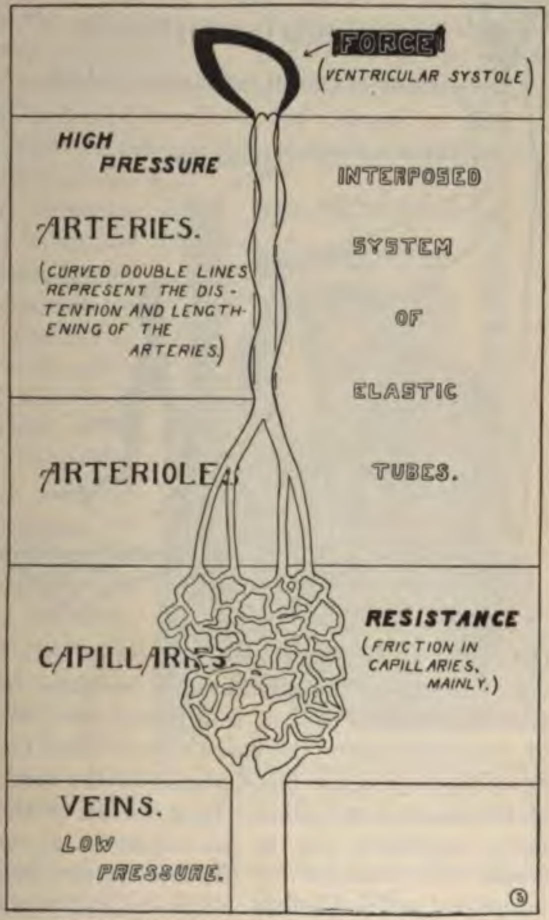
FORCE
(VENTRICULAR SYSTOLE)
HIGH PRESSURE
ARTERIES.
(CURVED DOUBLE LINES REPRESENT THE DISTENTION AND LENGTHENING OF THE ARTERIES)
INTERPOSED SYSTEM OF ELASTIC TUBES.
ARTERIOLES
RESISTANCE
(FRICTION IN CAPILLARIES, MAINLY.)
CAPILLARIES
VEINS.
LOW PRESSURE.

FIG. 96.—Diagram explanatory of arterial tension.

The state of contraction or dilatation of the vessels is, therefore, dependent upon the interaction of the constricting and dilating portions of the mechanism, by which more or less blood is admitted to special areas or to large portions of the body. In some areas the vessels may be dilated; in others, at the same moment, contracted. Much of this necessary variability in blood supply is dependent upon reflex influences, the stimulus or irritation coming either from the blood vessels themselves or from the end organs of sensory nerves in general. The constriction or dilatation appears usually in the vascular area from which the stimulus arises—e. g., the redness of a sinapism; or in a part functionally associated with the part stimulated—e. g., the hyperæmia attending an increased secretion from the submaxillary as the result of acid placed upon the tongue. The utility of counterirritation is based upon these facts. It is also to be noted that when the blood vessels of the skin are constricted, those of the interior are dilated (e. g., chill), or the contrary. The vasomotor apparatus may be an important factor in producing an increase or a diminution in the arterial pressure. Contraction of the peripheral arteries increases friction and, therefore, the resistance to the flow of blood from arteries into capillaries, and the pressure rises. Conversely, if the arteries dilate, the pressure falls.

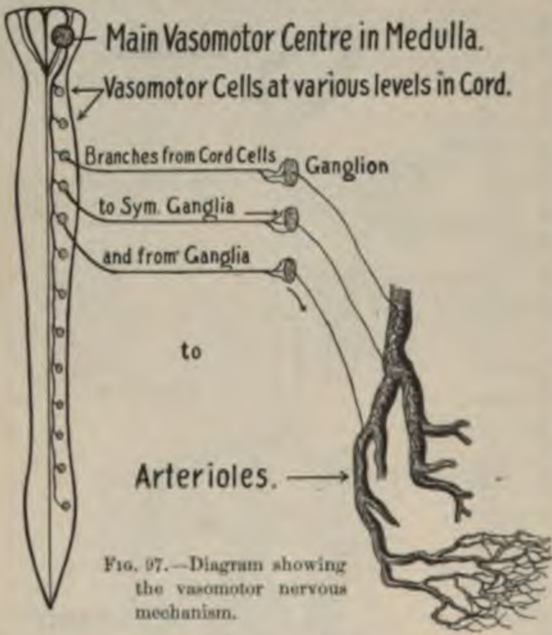

FIG. 97.—Diagram showing the vasomotor nervous mechanism.

II. PATHOLOGICAL PHYSIOLOGY OF VALVULAR DEFECTS

Direct Effect upon the Heart.—Before proceeding to study the evidences of cardiac and vascular disease, it will be of service to deal briefly with some of the disturbances of the normal cardiac functions which result from defective valves.

Normally the blood flows always in the same direction, because the valves close easily and accurately; and it passes in proper amount, because the openings into and out of the ventricles are sufficiently large to permit its free entrance and exit. Defects at the valvular openings are therefore of two kinds: either the valve openings are narrowed (*stenosis*), or the valves, because of shrinkage, do not close effectually (*incompetency, regurgitation*). The valves may be normal and yet incompetent, if the ring or opening to the margin of which the valve is attached becomes stretched and dilated to such an extent that the valve segments can not meet (*relative insufficiency*).

Whatever the nature of the defect, the final result is to hinder the flow of blood along its normal channels by causing stagnation or stasis in one of the chambers of the heart (Fig. 98). The cavity of the heart, which, with reference to the course of the blood, lies behind the narrowed or incompetent valve, is habitually overfilled; it can not properly empty itself, and because of its constant overdistention becomes dilated. In course of time its muscular walls, having an excessive amount of work to perform, increase in thickness and strength, i.e., undergo hypertrophy up to a certain degree, which is determined by the gravity of the lesion and the nutritional capability of the cardiac muscle.

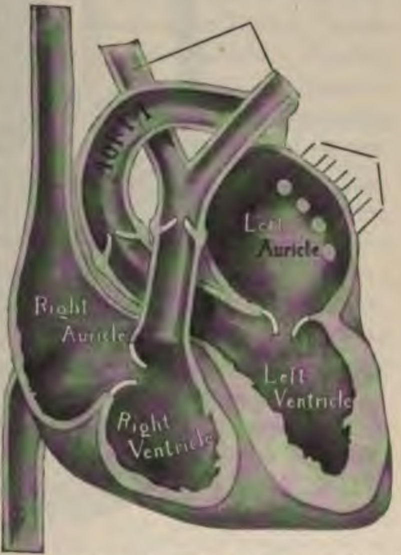

FIG. 98.—Semidiagrammatic representation of the chambers, valves, and vessels of the heart. After Page (redrawn and modified).

According to the particular valve affected and the nature of the lesion, the resulting changes vary in detail (Fig. 99, 100).

(1) In *aortic stenosis* the valve segments are most commonly thickened, rigid, and adherent by their edges so that during the systole of the left ventricle they refuse to open out against the aortic walls, and the exit of blood from the ventricle is hindered (Fig. 99). Consequently the latter hypertrophies, generally a simple hypertrophy, with little dilatation until the disease is well advanced, when there may be relative mitral insufficiency and its results. (See (3) following.)

(2) In *aortic incompetency* the valve segments are shrunken and

their edges curled, so that they fail of exact apposition. As a result, after the left ventricle has delivered its charge of blood into the aorta, the elastic pressure of the latter drives a portion of the blood which it contains back through the defective valve into the ventricle.

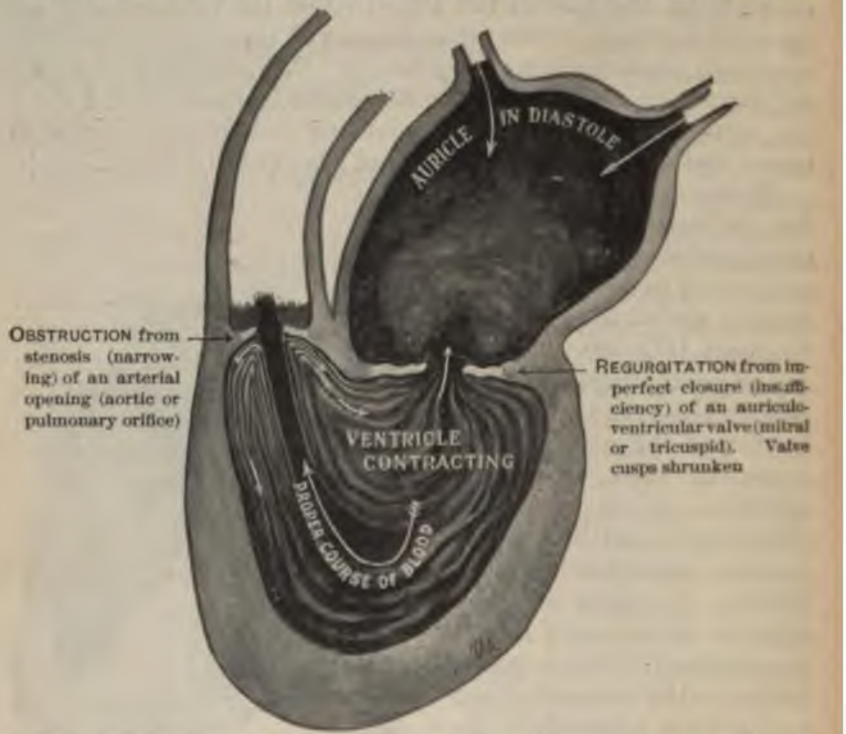
AURICLE IN DIASTOLE
OBSTRUCTION from stenosis (narrowing) of an arterial opening (aortic or pulmonary orifice)
REGURGITATION from imperfect closure (insufficiency) of an auriculo-ventricular valve (mitral or tricuspid). Valve cusps shrunken
VENTRICLE CONTRACTING
PROPER COURSE OF BLOOD

FIG. 99.—Diagram representing insufficiency and stenosis. Large arrows = normal course of blood; small arrows = abnormal currents.

During its diastole, therefore, the left ventricle is receiving blood from both the auricle and the aorta. Under this increased pressure its walls primarily dilate and secondarily become hypertrophied. Very frequently there is also relative mitral insufficiency. (See (3) following.)

(3) *Mitral incompetency* results either from shrinking and puckering of the cusps, or from dilatation of the left ventricle so that the segments can not fill in the enlarged opening (relative insufficiency), or from weakening of the muscular tissue of the heart, so that the papillary muscles do not act with accuracy, nor does the mitral orifice contract as in health during systole.

In consequence of the imperfect closure of the mitral valve during the systole of the left ventricle, a portion of the blood which should be expelled into the aorta regurgitates into the left auricle (Fig. 99). The auricle thus receives blood during its diastole from two sources, the left ventricle and the pulmonary veins. It therefore becomes overdistended and dilates, and, as it must do extra work in discharging an unusual amount of blood into the ventricle, undergoes hypertrophy. The left ventricle, receiving this unusual amount of blood just previous to its systole, also dilates and hypertrophies. The increased pressure in the left auricle dams back the blood successively in the pulmonary veins, capillaries, artery, and right ventricle. The right ventricle dilates and hypertrophies, because of the distention and increased work caused by the obstruction in the pulmonary circuit. The tricuspid valve may become relatively insufficient, and regurgitation take place into the right auricle. Finally, the venæ cavæ, and through them the venous side of the systemic circulation, may enter upon a condition of permanent engorgement.

(4) *Mitral stenosis* may be due in varying proportions to narrowing of the ring, adhesion of the valve cusps by their edges, or contraction of the chordæ tendineæ. In consequence of the lessened size of the mitral orifice the auricle has difficulty in expelling its contents into the left ventricle. It dilates and to a marked extent undergoes hypertrophy. As with mitral insufficiency, the impediment to the blood which comes through the pulmonary veins into the left auricle causes dilatation and hypertrophy of the right ventricle and auricle, and final systemic venous congestion.

(5) *Tricuspid incompetence* is usually relative and secondary to valvular lesions of the left side of the heart. The right ventricle and auricle dilate and undergo hypertrophy for the same reasons as the left chambers in mitral insufficiency, and the systemic veins become overfilled.

(6) *Tricuspid stenosis* is usually secondary to left-side lesions, the increased work and blood pressure imposed by the latter giving rise to sclerotic changes. It may be of congenital origin. By a similar mechanism to that of mitral stenosis, the right auricle dilates and hypertrophies. The systemic veins are engorged.

(7) *Pulmonary stenosis*, a rare congenital defect, causes hypertrophy and dilatation of the right ventricle, and subsequent similar changes in the right auricle.

(8) *Pulmonary insufficiency*, another rarity, produces hypertrophy and dilatation of the right ventricle and auricle.

Compensation and its Failure.—By compensation is understood the effort made by the heart muscle through hypertrophy to meet the increased work thrown upon it. So long as the increased strength of the cardiac walls serves to propel the blood in sufficient amount and with sufficient thoroughness to prevent marked stagnation in any portion of the blood stream, there are no subjective symptoms, and the patient is to all intents and purposes well, although during this period dilatation and hypertrophy are progressing.

If, however, for any reason the strength of the heart muscle fails slowly or abruptly, the compensation is said to be "broken" or "ruptured." The point of principal clinical interest with reference to compensation is that its completeness depends almost entirely upon the condition of the cardiac muscle. The most important question to be answered during the examination of a case of valvular defect is with reference to this point. Aside from the signs discerned in the heart itself—the *direct* effects of the lesion (dilatation, hypertrophy, etc.)—this question must be answered by the presence or absence of certain *indirect* or peripheral effects or symptoms.

Indirect Effects of Valvular Lesions.—With few exceptions the peripheral symptoms are due to passive (venous) congestion of various organs sequent to the damming back of the blood by various valvular defects. The congestion, for obvious reasons, affects the pulmonary circuit and the lungs primarily, and later in the disease the organs and parts drained by the systemic veins. The organs and parts of the body and the symptoms of more or less complete compensation which they offer are as follows (see Fig. 100):

(1) **Lungs.**—Dyspnœa, cough, hæmoptysis, or pulmonary œdema demand an examination of the heart. So also do frequent severe or protracted attacks of bronchitis. Hydrothorax is a direct result of valvular disease. The long-continued pulmonary congestion and high vascular pressure may lead to atheromatous changes in the vessels and brown induration of the lungs.

(2) **Liver.**—Back pressure in the inferior vena cava may show itself by congestion and great swelling of the liver and, if there is tricuspid insufficiency, pulsation of the liver as well. Long-continued passive hyperæmia produces the nutmeg liver.

(3) **Stomach, Spleen, Intestines.**—As these organs must drain through the portal vein, the congested hepatic capillary system furnishes, as compared with other viscera, an additional barrier to the emptying of their veins. Consequently, the *stomach* offers the symptoms of a catarrhal gastritis, the *spleen* becomes somewhat enlarged, and the *intestines* become the seat of a chronic catarrh. Diarrhœal attacks are controlled with difficulty or alternate with constipation. In advanced cases the congestion is so great that there is a serous exudate into the peritoneal cavity (ascites).

(4) **Kidney.**—There is renal congestion; the urine is scanty, albuminous, and contains tube casts.

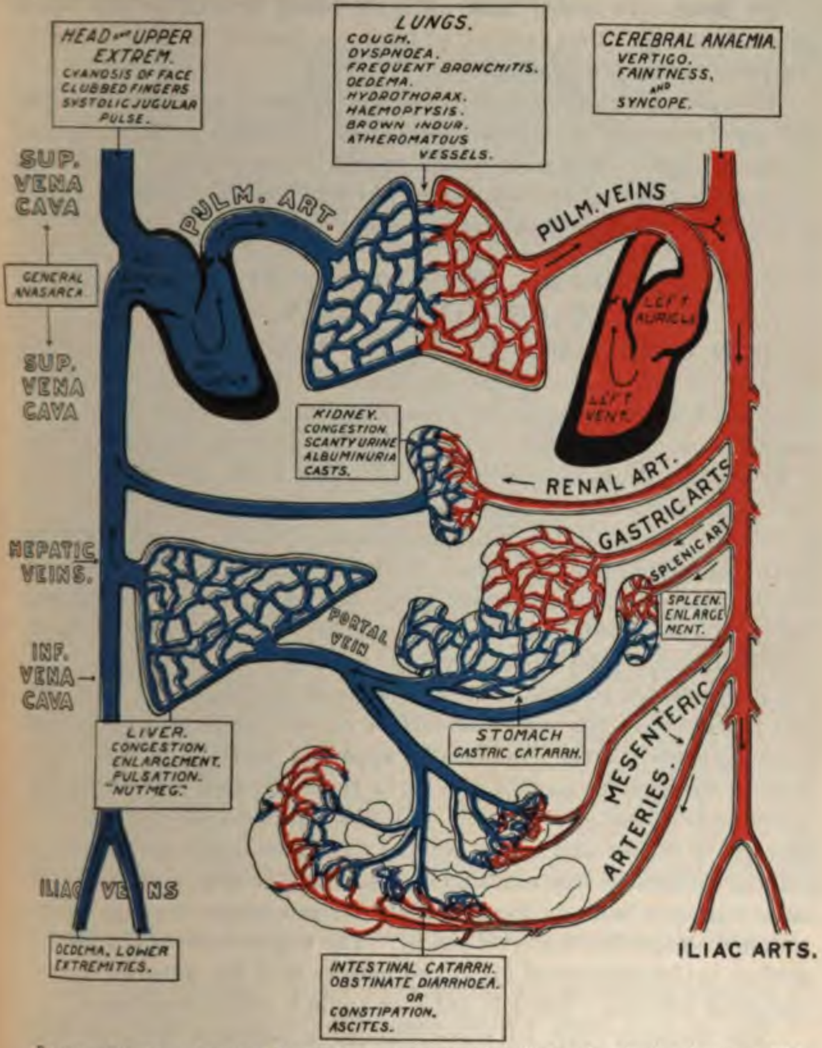

FIG. 100.—Diagram showing the indirect (peripheral) effects of valvular lesions. This diagram is also of service in tracing the direct (cardiac) changes due to valvular defects. The indirect results are catalogued in the sign squares.

(5) **Venæ Cavæ.**—If the back pressure in the superior cava is continuously high, there is cyanosis of the lips and face, clubbing of the fingers, and, with tricuspid regurgitation, a systolic jugular pulse. As a result of a similar condition in the inferior cava, there is œdema

of the lower extremities, and, if the pressure is extremely high in both cavæ, general anasarca.

(6) **Brain.**—In some cases so little blood is sent to the brain (arterial anæmia) that vertigo, or faintness, partial or amounting to complete syncope, may occur.

The foregoing signs and symptoms, if present, demand on the one hand a search for cardiac disease; on the other hand, if cardiac disease is found, these phenomena should be inquired for in order to make an estimate of the manner in which the heart is doing its work —i. e., the condition of the heart muscle.

III. TOPOGRAPHICAL ANATOMY OF THE HEART AND ITS VALVES

Shape of the Heart.—An excellent description of the surface anatomy of the heart is that of Keiller (American Journal of the Medical Sciences, April, 1898), based upon the models of His and his own gelatin-injected specimens. The present purpose will be fully subserved by describing the heart as a whole and the anterior surface in particular.

The heart is an irregular, four-sided pyramid, its base resting upon the diaphragm. Its apex is truncated, thus offering a place for the roots of the upspringing great vessels. It therefore possesses five surfaces, anterior, posterior, right, left, and inferior (the base), with well-defined borders separating them.

The anterior surface is triangular in shape, slightly curved, and lies parallel with the posterior surface of the sternum. It includes from right to left the whole right appendix and a part of the right auricle, the greater part of the right ventricle, and portions of the left appendix and left ventricle. It is separated from the right surface by the convex and nearly vertical right anterior border; from the inferior surface or true anatomical base, by the sharp, almost straight antero-inferior border; from the left surface by the convex, slightly rounded oblique left anterior border. The upper angle of the anterior surface is the *anatomical* apex, and merges into the anterior walls of the great arteries. The left anterior angle is the *clinical* apex.

The diagram (Fig. 101) is drawn in accordance with the facts just rehearsed, except that the right lower angle of the heart is rounded. Owing to the normal anatomical and functional variations in the shape of the heart, and the changes in its position resulting from respiratory action, bodily posture, and the shape of the thorax, no one diagram can tell the whole story.

Relation of Heart to Chest Walls.—The projection of the heart upon the anterior chest wall, an outline corresponding to the shadow of the organ which would be cast by parallel rays of light passing from back to front, is obtained approximately—provided that the chest is well formed—as follows (see Fig. 102):

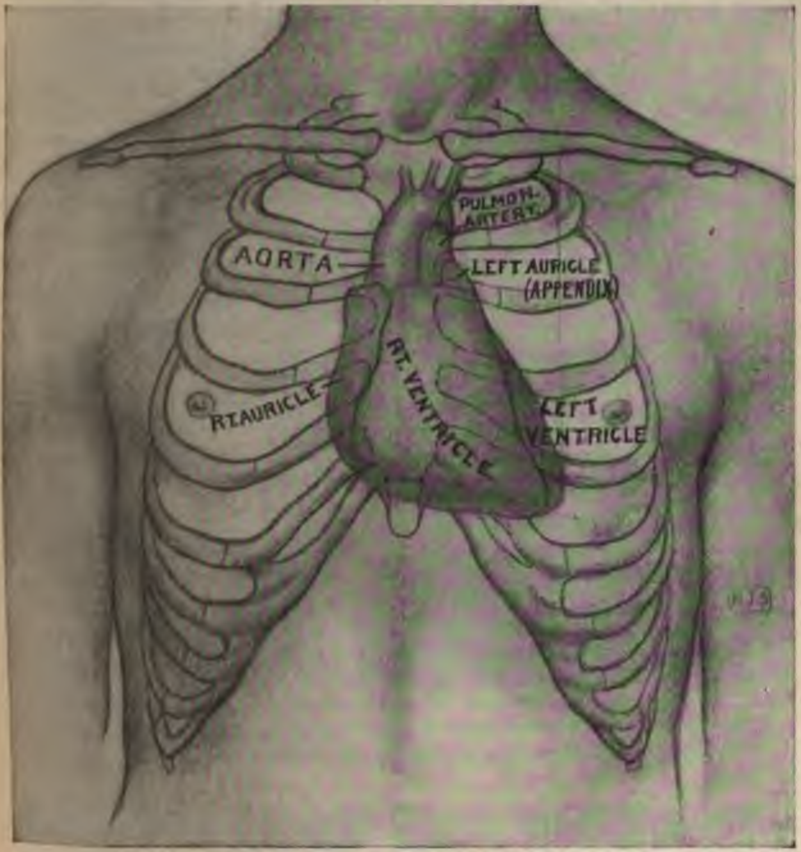

Fig. 101.—The anterior aspect of the normal heart and great vessels, showing their relations to the anatomical landmarks (ribs, sternum, clavicles) of the front of the thorax.

(1) *Upper Border.*—Draw a horizontal line across the sternum at the level of the 3d costal cartilages, from a point A, $\frac{1}{2}$ an inch to the right of the right edge of the sternum, to a point B, 1 inch to the left of the left sternal edge. This is the dividing line between the heart and the great vessels, and is the clinical base of the heart.

(2) *Lower Border.*—Mark a point D, vertically below the nipple, in the 5th intercostal space between the 5th and 6th left ribs, just at the upper edge of the latter. The apex beat (which is not made by the extreme apex of the heart) is usually felt in the middle of the

5th space, and $\frac{1}{2}$ to 1 inch inside the mammillary line. Mark also a point, *C*, at the junction of the upper and middle 3ds of the *ensiform cartilage*. Point *C* is usually $\frac{1}{2}$ an inch higher than point *D*.

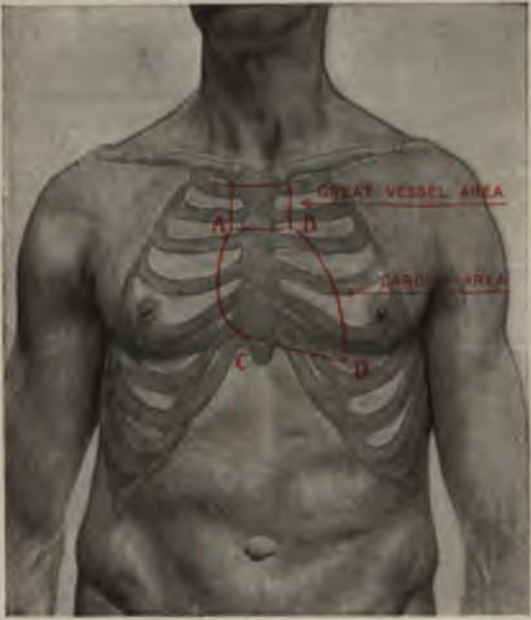

FIG. 102.—Points upon the chest by which the normal boundaries of the heart and great vessels may be determined. Nipples in this figure are too far away from the median line.

The statements here made with reference to the position of the lower border of the heart do not agree with those commonly accepted, which place this border at a point a little above the lower end of the sternum. The ordinary descriptions are correct for the cadaver (SIBSON), not for the living subject, owing to the ascent of the diaphragm and heart which takes place *in articulo mortis*.

Draw a line connecting these points.

(3) *Right Border*.—From point *A* draw a line downward, curving a little to the right, until it reaches the middle of the 4th interspace, then curving to the left until it passes over the cartilages of the 6th and 7th ribs, a little before they join the sternum, and meets point *C* over the ensiform cartilage.

(4) *Left Border*.—Connect points *B* and *D* by a line slightly convex to the left.

This outline represents the position of the heart in a well-formed chest. In an emphysematous thorax (permanent position of inspiration) it lies lower in relation to the ribs, its inferior border descending to the tip of or below the appendix. On the other hand, if the chest is of the alar or phthisical type (permanent position of expiration), its lower border may lie half an inch or more above the end of the sternum.

Mobility of the Heart.—In addition to the permanent variations in position just mentioned, the heart is movable by *posture* and *respiration.* If the individual lies in the left lateral position, the apex beat moves from 1 to 2 inches to the left; if flat on his back, the heart recedes from the chest wall, and the apex beat may become imperceptible; if on the right side, the impulse moves nearer to the median line; if he leans forward or lies face downward, the apex beat strengthens. In forced inspiration the diaphragm descends, drawing the heart downward and toward the median line by means of the attachment of the pericardial sac to its central tendon. The ribs move upward, thus increasing the displacement of the heart relatively to these bony landmarks. In forced expiration the diaphragm moves upward, elevating the heart and shifting its apex farther to the left. The excursion of the diaphragm may amount to 2 inches. An absence of the normal mobility of the heart is an important sign of adherent pericardium.

When the heart moves, it swings from its point of suspension by the great vessels at the base. Consequently, the excursion of its apical portion from side to side or from front to back is much greater than that of the base, except where there is a localized pull by retracting fibroid tissue or pressure by new growths.

Relation of Cavities and Great Vessels to Chest Wall.—The relations of the various cavities of the heart to the chest wall are shown sufficiently in Fig. 101.

The great-vessel area, so called for convenience, may be outlined by first drawing a horizontal line across the junction of the middle and upper 3ds of the manubrium. This line should lie about ¾ inch below the upper border of the manubrium (episternal notch). It should extend right and left sufficiently to permit a vertical line to be drawn downward from each end to points *A* and *B* (Fig. 102). The vessels contained in this space are shown in Fig. 101.

IV. PHYSICAL EXAMINATION OF THE HEART AND ITS NEIGHBOURHOOD

The *subjective symptoms* of cardio-vascular disease are summarized elsewhere (see Synopsis of Examinations), and referred to in detail in various places.

The *objective condition* of the heart is ascertained by inspection, palpation, percussion, and auscultation. The patient should be, preferably, semirecumbent, as there are fewer physical difficulties in the way than if sitting or standing.

A. Inspection and Palpation

By inspection and palpation, which are instinctively combined, the examiner determines the shape of the præcordium, certain facts regarding the apex beat and præcordial pulsation, the presence or absence of pulsating or distended veins, and other abnormal pulsations or thrills.

Elsewhere (see Index) have been considered the *shape of the præcordium*, *distended veins of chest*, and *pulsating jugulars*.

Apex Beat.—The apex beat lies normally in the 5th left intercostal space, ½ inch inside the mammillary line, and about 3 inches to the left of the midsternal line. In health the apex beat is limited to a space 1 inch in breadth, occupying one interspace only.

It is somewhat internal to the actual apex formed by the ventricles, the right ventricle, not the apex, striking against the chest wall. Thus when the apex is carefully outlined by percussion the lower left limit of cardiac dulness will usually be found, in health, about ¾ of an inch to the left of, and a little below, the maximum impulse. The actual position of the apex, therefore, is always outside of the palpable maximum impulse. This normal discrepancy is increased when the heart is hypertrophied, the enlarged and rounded organ holding the true apex still further away from the chest wall.

In the majority of cases the apex beat can be both seen and felt, in many felt but not seen, and in some it may be imperceptible. In palpating the apex beat, the whole hand should, first of all, be laid smoothly but firmly over the præcordial region, the fingers pointing downward and to the left. This is important, because the character of the heart action as well as the point of most marked impulse is at once appreciated. After this the finger tips may be employed to accurately locate the apex beat. As the impulse may be diffuse, the apex beat should be considered to lie at the point where the finger appreciates a distinct thrust from within. If a localized impulse can not be felt while the patient is recumbent, he should be required to sit upright, lean forward, or turn upon the face (if his condition permits), so as to bring the heart to the front of the thorax by gravity.

The apex beat is to be studied with reference to position, character, and extent (Fig. 103).

Position of the Apex Beat.—The position of the apex beat is of extreme importance, as it furnishes the most reliable information with reference to the situation of the heart. As it coincides with the ventricular systole, it also furnishes the standard for determining the time relation of other pulsations—i. e., whether they are systolic, presystolic, or diastolic. Aside from normal alterations in the position of the heart, the apex beat may be moved from its natural location, either by an actual dislocation of the entire heart caused by pressure or pull, or by changes in its shape due to hypertrophy or dilatation. It is to be borne in mind that deformities involving the chest, kyphosis, scoliosis, *et al.*, deprive the position of the apex beat of much of its diagnostic value, as it may be displaced in any direction and mistakenly considered a result of serious cardiac disease or as a marked congenital anomaly of position.

(1) Displacement to the *left and upward* is the result of varying lesions. It may be due to pericardial effusion, or to abdominal distention with upward pressure upon the diaphragm by gas, fluid, or tumour. Fibroid changes in the left lung or pleura, forming a part of chronic phthisis, interstitial pneumonia, and extensive pleural adhesions, by their contraction will pull the heart to the left and the diaphragm upward, carrying the apex in the same direction. It is also carried to the left and upward by the pressure of fluid or air in the right pleura or by mediastinal tumour. In severe cases of this displacement the apex may beat in the midaxillary line. It should be remembered that in infants, and in children up to the 10th and 12th year, the apex beat is normally in the 4th space, at or outside the mammillary line.

(2) Displacement to the *left* in a *horizontal line* may arise from any of the causes mentioned in (1), but is especially characteristic of hypertrophy and dilatation of the right ventricle. It rarely passes farther outward than the mammillary line.

(3) Displacement to the *left and downward* is characteristic of cardiac hypertrophy and dilatation, especially of the left ventricle, in which case it may be carried even to the 8th space and midaxillary line.

(4) *Downward displacement* of the beat may be induced by hypertrophic emphysema. In this disease the chest is in a permanent inspiratory position and the ribs move upward over the heart. The lungs become larger and press the heart downward, and the hypertrophied right ventricle contributes to its lowered position. Aneurism of the arch of the aorta and tumours in the upper portion of the mediastinum will, if of sufficient size, depress the heart and with it the apex beat. An enlarged liver, by dragging upon the central tendon of the diaphragm and the attached pericardial sac, has pulled the heart downward (PAUL). Bathycardia (low heart) may be due to the stretching of a sclerotic aorta. In the aged the apex beat may normally be found in the 6th interspace.

(5) *Displacement to the right*, very rarely further than the right mammary line, may be caused by the retracting power of chronic fibroid changes in the right lung, and adhesions in the right pleural cavity. A similar displacement may be caused by the pressure of left-side pleurisy with effusion, hydrothorax, pneumothorax, or tumour of the left lung.

Character and Extent of Apex Beat.—By constant practice the hand learns to appreciate very correctly any variations in the extent and power of the impulse of the heart against the thorax.

(1) A somewhat *forcible and extensive* apex beat of a knocking or slapping character may be due to mental excitement, nervousness, or physical exertion. It is important not to form a judgment of the cardiac status under these circumstances or, if unavoidable, to make proper allowance therefor. Certain drugs (tea, coffee, nicotine, alcohol, etc.) have a similar effect. In the early stage of acute fevers the same overaction is observed—indeed, it is present in almost all cases of palpitation (*q. v.*).

In persons with unusually thin chest walls the impulse of the heart is both visible and palpable over a much wider extent than in those whose ribs are well covered. Another cause of increase in extent, not necessarily in strength, of the impulse, is the uncovering of the heart to the left of the sternum by retraction of the corresponding lung. In a well-marked case the impulse is visible and palpable in the 3d, 4th, and 5th interspaces, sometimes slightly in the 2d, and the snap of the arterial valves may be very distinct. This is excellent evidence of the pulmonary condition, and should not be mistakenly referred to disease of the heart.

In all the foregoing cases the apex beat is not displaced, a prime fact in determining the absence of hypertrophy.

A strong lifting or heaving impulse, which can be felt over 2 or 3 interspaces, with displacement of the apex beat to the left and downward, is a most valuable sign of hypertrophy of the left ventricle.

(2) *Weakness or absence* of the apex beat may be caused by dilatation of the heart subsequent to hypertrophy, the lifting heave of the latter giving place to a diffused undulating impulse of little strength, which is quite distinctive. The disappearance of a defined and easily located apex beat is particularly marked in dilatation of the right ventricle, the distended roundness of the latter pushing the apex away from the chest wall. It disappears also with the onset of a considerable pericardial effusion which surrounds the apical portion of the heart and separates it from the thoracic wall.

Weakness or absence of the apex impulse is not incompatible with health. It may be due to a thick, fat chest wall, or to the fact that the apex strikes just behind a rib. In some otherwise normal cases there is no discoverable reason for its absence. If the heart is displaced to the right, the apex beat may be extinguished behind the lower sternum. If no apex impulse exists, the place where it should be found may be identified by auscultation—i. e., the point at which the 1st sound is heard with the greatest intensity. The excessive overlapping of the heart by the increased bulk of the lungs in emphysema may put a damper upon the impulse. Extensive or universal pericardial and mediastinal adhesions may so limit cardiac mobility that the apex does not strike with sufficient force to be felt. In this case a systolic drawing in or retraction, not alone of the apex space but of the lower sternum and several intercostal spaces and ribs, may replace the usual systolic protrusion, and possesses diagnostic value. In rare cases of extreme aortic stenosis the ventricle takes so long to propel the blood through the narrow orifice that the systole is deliberate and the celerity of movement necessary for the impulse lacking.

In general it may be said that, barring a thick chest wall or emphysema, a weak or absent apex beat indicates a weak heart from any cause—e. g., exhausting disease, shock.

Other Pulsations or Pulsating Swellings.—Inspection (especially by oblique illumination) and palpation may reveal centres of pulsation other than that of the apex beat as shown by the simultaneous presence of an apparent apex beat elsewhere.

A question frequently arises as to the exact rhythm of one pulsation as compared with another, comparison usually being made with the apex beat by placing a finger on each; or with the first sound, by auscultation while palpating. For greater accuracy indicators may be employed. The most convenient of these is a bit of absorbent cotton pulled out into a slender cone 2 or 3 inches long. Its base is applied to the pulsating spot, previously touched with mucilage or thick ointment. Having affixed one of these to each spot and placed the examining eye so that the cones are in a line, the movement of their extremities, by exaggerating the pulsations, enables a more ready determination of the rhythm of each pulsation. This method is also of service in determining the expansile character of a pulsating swelling, a cone being placed on each side of the tumour. If expansile pulsation is present the tips of the cones will approach and depart in an unmistakable fashion.

Some pulsations are visible, others not visible but palpable, and still others present a more or less marked prominence or bulging of the chest wall according to their nature.

These pulsations may originate from the great arteries (dynamic, aneurismal), the veins (pulsating liver), the heart (hypertrophy or dilatation), or may be communicated (pulsating empyema). Taking them, according to location, from above downward, and from right to left, they may be found as follows: As a general rule it may be said that pulsations above the level of the 3d rib belong to the great arteries, below that point to the heart.

(1) Pulsations in the *episternal notch* and at the root of the neck have been discussed elsewhere (page 284).

(2) Pulsation in the *right 1st or 2d interspace*, close to the right of the sternum (Fig. 103), systolic in time, expansile in character, may be due to aneurism of the ascending portion of the arch of the aorta or, without expansile character, to violent heart action. It is here that a diastolic shock accompanying the closure of the aortic valves is to be felt in aneurism of the arch.

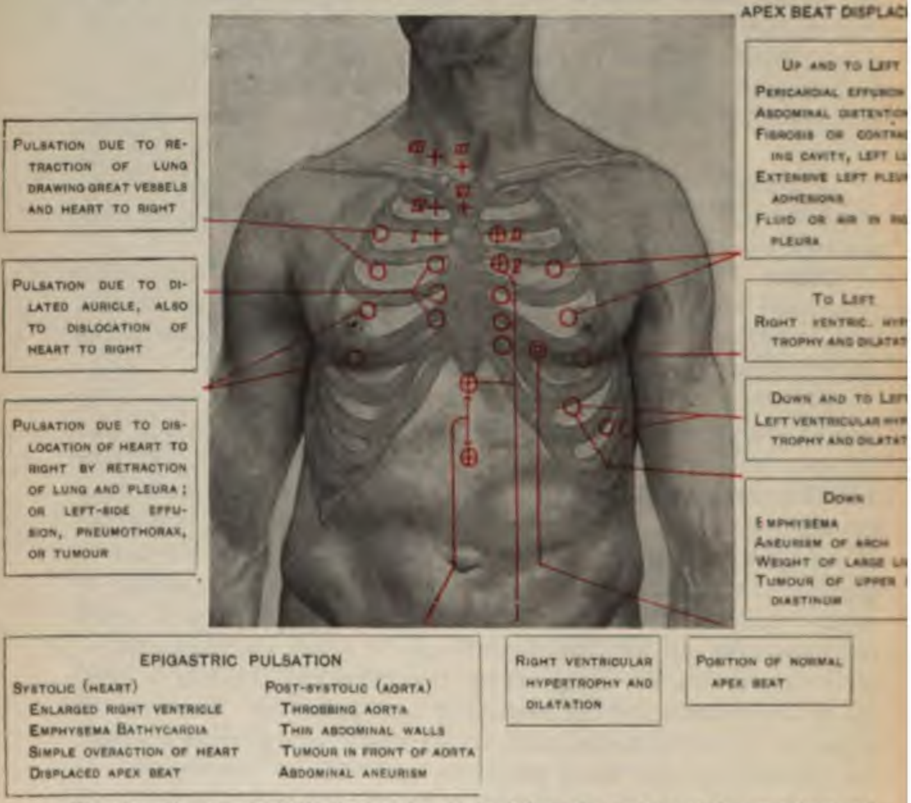
PULSATION DUE TO RETRACTION OF LUNG DRAWING GREAT VESSELS AND HEART TO RIGHT
PULSATION DUE TO DILATED AURICLE, ALSO TO DISLOCATION OF HEART TO RIGHT
PULSATION DUE TO DISLOCATION OF HEART TO RIGHT BY RETRACTION OF LUNG AND PLEURA; OR LEFT-SIDE EFFUSION, PNEUMOTHORAX, OR TUMOUR
APEX BEAT DISPLAC
UP AND TO LEFT
PERICARDIAL EFFUSIO
ABDOMINAL DISTENTIO
FIBROSIS OR CONTRA ING CAVITY, LEFT L
EXTENSIVE LEFT PLEU ADHESIONS
FLUID OR AIR IN RI PLEURA
TO LEFT
RIGHT VENTRIC. HY TROPHY AND DILATAT
DOWN AND TO LEF
LEFT VENTRICULAR HY TROPHY AND DILATAT
DOWN
EMPHYSEMA
ANEURISM OF ARCH
WEIGHT OF LARGE LI
TUMOUR OF UPPER DIASTINUM
EPIGASTRIC PULSATION
SYSTOLIC (HEART)
ENLARGED RIGHT VENTRICLE
EMPHYSEMA BATHYCARDIA
SIMPLE OVERACTION OF HEART
DISPLACED APEX BEAT
POST-SYSTOLIC (AORTA)
THROBBING AORTA
THIN ABDOMINAL WALLS
TUMOUR IN FRONT OF AORTA
ABDOMINAL ANEURISM
RIGHT VENTRICULAR HYPERTROPHY AND DILATATION
POSITION OF NORMAL APEX BEAT

FIG. 103.—Showing the indications to be obtained from the position of the apex beat and other thoracic and epigastric pulsations. For pulsation in the episternal notch, see page 284. *I, II, III, IV, V, VI* = pulsations of aortic aneurism (arch), numbered in order of frequency of occurrence. *VII* = innominate aneurism. *II* = also pulsation of pulmonary artery (and left auricular appendix?). *V* = also pulsation of right ventricle (and left auricular appendix?). ○ = cardiac impulses. + = aneurismal pulsations. ⊕ = both.

(3) Pulsation in the *right 2d and 3d interspaces*, ranging from the sternal edge to the mammillary line, may be caused by a dragging over of the heart and great vessels due to the retraction of a fibroid

lung. In one extreme case of this kind under observation the impulse extended upward into the first interspace as well.

(4) Pulsation in the *right 3d, 4th, and 5th interspaces* close to the edge of the sternum results from a dilated right auricle or a dislocation (displacement) of the heart.

(5) Pulsation in the *right 4th and 5th spaces*, at any point between the sternal edge and the mammillary line, is produced by displacement of the heart. This is the usual location of a heart dislocated by great fluid or gaseous pressure from the left pleural cavity, but retraction of the lung may pull the heart over so that it pulsates in these spaces as well as in the 2d and 3d (see (3) preceding).

(6) Pulsation over the *manubrium* is significant of aneurism of the aorta, especially of the transverse portion of the arch, which has eroded the bone.

(7) Pulsation in the *2d* and sometimes the *3d left interspace* close to the sternal edge may be indicative of aneurism of the aorta, particularly the descending portion of the arch, in which case the impulse is systolic; or of pulsation of the pulmonary artery, systolic if due to the filling of the artery by ventricular systole; and immediately following the 2d sound if it is the rebound of the artery following the closure of the pulmonary valve. It may possibly, for there is a difference of opinion on this point, be due to the impulse of an hypertrophied left auricular appendix, in which case it precedes the apex beat and 1st sound.

(8) Pulsation in the *3d, 4th, 5th, and 6th interspaces* just to the *left* of the sternum is usually caused by hypertrophy and dilatation of the right ventricle.

(9) Pulsation in the *3d and 4th spaces* in the *left mammillary line* is usually the apex beat of the heart displaced up and to the left by pressure from the right side, or fibroid contraction of the left lung.

(10) Pulsation in the *left 5th space* outside the mammillary line is usually the apex beat of a right ventricular hypertrophy.

(11) Pulsation in the *6th, 7th, or 8th left interspaces* anywhere between the mammillary and midaxillary lines is usually the apex beat of left ventricular hypertrophy and dilatation. As a rule, the farther the apex from the median line, the lower the interspace it occupies.

(12) Pulsation over the *left half of the præcordial area*, and in the left axillary region may in rather rare cases be caused by "pulsating empyema" or, still more rarely, by a very vascular malignant tumour.

Aside from pulsations strictly confined to the thorax, it is necessary to ascertain whether or not there is epigastric pulsation, or throbbing of the liver.

(13) *Epigastric pulsation*, if found, should be tested as to rhythm by placing one hand upon the apex beat, the other over the epigastrium. The epigastric impulse will be found either to coincide with the apex beat (systolic), or to occur immediately but distinctly after the heart beat (post-systolic).

If *systolic*, the impulse comes directly from the heart, which may indicate enlargement or dilatation of the right ventricle, from various causes, but is much more commonly due to an apex beat displaced to the right behind the sternum, a transmitted pulsation through the left lobe of the liver, bathycardia (low heart, cardioptosis), emphysema, or simple overaction of the heart from any cause. In the latter case the pulsation is hardly more than a systolic quiver or trembling. An abnormally short sternum may also be a responsible cause.

If *post-systolic*, the impulse is from the abdominal aorta, and its most common cause is the functional (dynamic) throbbing of the vessel which is seen in many neurotic and neurasthenic individuals, or in those suffering with disorders of gastric digestion. It may be present as a result of anæmia and hemorrhage, and may be perceived with a little care in any person with thin abdominal walls. A very marked pulsation may arise from the presence of a tumour—e. g., enlarged lymph glands, pyloric cancer, tumour of pancreas, hypertrophied or sclerosed left lobe of the liver, and hepatic abscess. Finally, it may be an aneurism, but this diagnosis is to be made with much hesitation, and only when the pulsation is distinctly expansile. The pulsation due to a tumour overlying the aorta will disappear in the knee-chest position, the mass falling forward and away from its contact with the artery.

Systolic depression of the epigastrium and lower sternum is of value in making a diagnosis of extensive pericardial and mediastinal adhesions (indurative mediastino-pericarditis). One may mention here a systolic depression of the left back in the region of the 11th and 12th ribs as a sign of extensive pericardial adhesions, the pull of an hypertrophied heart adherent to the diaphragm making traction through the latter upon the 2 movable lower ribs (BROADBENT).

(14) *Pulsation of the liver* is to be noted as present only when the entire organ is enlarged and pulsates, not when the pulsation is limited to the epigastrium. General hepatic pulsation is best determined by placing one hand with firm pressure over the right hypochondrium, the other posteriorly so that the liver lies between the two. The whole organ is then felt to expand and, if timed, the pulsation is found to immediately follow the apex beat. This pulsation

is due to and pathognomonic of tricuspid insufficiency, the regurgitant wave being transmitted backward along the inferior cava and hepatic veins (Fig. 100).

Thrills and Friction Fremitus.—Pulsation may be attended by thrill. Palpable vibrations produced by the passage of blood over a roughened surface, through a narrowed orifice or a leaky valve, are called thrills. A thrill is discernible as a fine whizzing or purring sensation (*frémissement cataire*), but the vibrations may be distinctly coarse. A palpable vibration, due to the sliding of two roughened pleural or pericardial surfaces over each other, is termed friction fremitus.

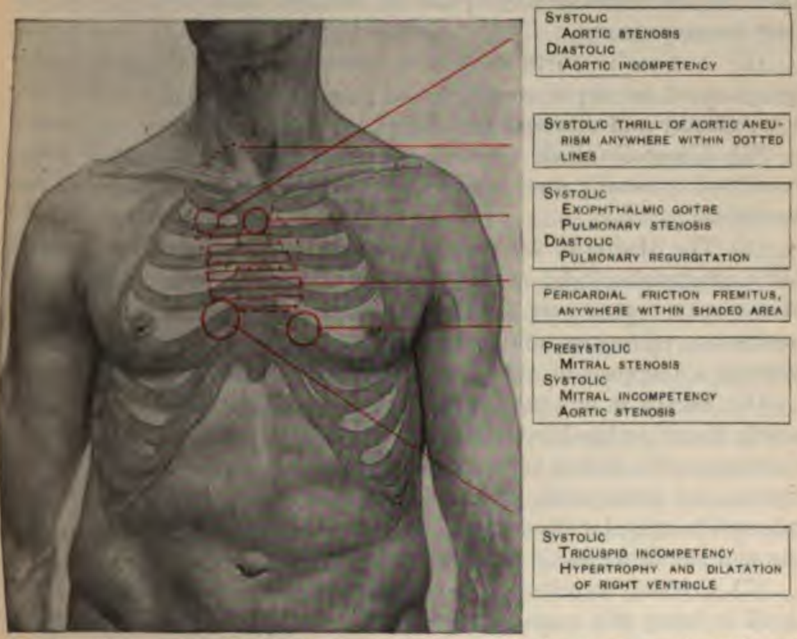

FIG. 104.—Diagram showing the diagnostic significance of the location and rhythm of thrills and friction fremitus.

When a thrill or friction fremitus is perceived, it is necessary to determine its position of maximum intensity and its time relation to the apex beat—i. e., is it systolic, diastolic, or presystolic?

Friction fremitus is caused by inflammation of the pericardium, or of those portions of the pleural surfaces which overlie or are in close contact with the heart. In both cases the inflamed or roughened surfaces are rubbed together by the movements of the heart, and therefore the vibrations exhibit a rhythmic cardiac sequence. It is usually easy by auscultation to distinguish between friction fremitus and a true thrill. The former is to and fro in character, and does not bear a constant and definite relation to the events of the cardiac cycle. The thrills of most common occurrence are those mentioned in (1) and (4) following.

(1) At and above the right 2d cartilage and interspace a systolic thrill, due to aortic stenosis (see Fig. 104). At the same point, and perhaps felt for some distance down the sternum, a diastolic thrill, due to aortic incompetency.

(2) At and above the left 2d cartilage and interspace, a systolic thrill, which is not infrequent in exophthalmic goitre. Very rarely it may indicate pulmonary stenosis. A diastolic thrill in the same spot may be found as a symptom of another rare condition, pulmonary incompetency.

(3) A systolic thrill over the lower part of the sternum as a symptom of tricuspid regurgitation; or, without valvular disease, in hypertrophy and dilatation of the right ventricle.

(4) A systolic thrill over the apex beat and its immediate neighbourhood is usually due to mitral incompetency, sometimes to aortic stenosis.

(5) The thrills so far referred to are fine and soft in character, but a presystolic thrill at and somewhat above and to the inner side of the apex beat is very distinctive. It is composed of rather rough, hesitating vibrations, and even in the absence of other symptoms permits a diagnosis of mitral stenosis.

(6) Thrills in any part of an area extending from the episternal notch above to the level of the 4th rib below, and on either side, for a distance of 2 inches beyond the sternal edge, may be due to aneurism of the aortic arch. A thrill discerned in this area should not be referred to valvular disease unless in the absence of a pulsating swelling and the other signs of aneurism.

(7) Friction fremitus, of pericardial or pleural origin, differs from thrill in being of a to-and-fro character, and having no very definite relation to the heart sounds. It is ordinarily found in that part of the præcordium lying between and including the 2d and 4th interspaces.

B. Percussion of the Heart

The object of percussing the heart is mainly to determine its shape, size, and location. Incidentally, in doing this we may discover dulness due to pericardial effusion, and also, from the position of the left anterior border of the left lung, whether the latter is emphysematous, retracted, or normal.

Areas of Cardiac Dulness and their Relative Value.—Percussion of the heart under normal conditions affords two areas of dulness: *first*, the absolute or superficial dulness developed by that portion of the heart which is not covered by lung (Plate I) and, therefore, lies in close contact with the chest wall; and *second*, the deep or relative dulness of that portion of the heart overlapped by the lungs. As in practice these terms are apt to be confused, the first will be called, from personal preference and to avoid uncertainty, the *exposed dulness*; the second, the *covered dulness*; both together, the *entire dulness* of the heart.

The area of exposed cardiac dulness in the normal heart is bounded by a line drawn from the level of the 4th cartilage downward along the left edge of the sternum, and a second line starting from the upper end of the first and running downward to a point on the left 6th rib in the parasternal line. This gives an area of triangular shape.

The size and shape of the covered dulness depend upon the method of examination used. The more accurate the method the more nearly will the outline of the covered dulness correspond to the anatomical or projection outline of the heart.

The clinical importance of the exposed cardiac dulness in determining the size of the heart has been much overrated, and many clinicians of to-day pay no attention to it in estimating cardiac conditions. The size of the exposed dulness does change to some extent with the size of the heart, but to a much greater degree it varies with the position of the overlapping lung borders. Practically, the only utility of outlining the exposed dulness is to demonstrate the condition of the lung—viz., emphysema *versus* shrinking—and to account for an unusual corresponding absence or increase of præcordial pulsation. As a matter of fact, what the clinician desires in percussing the heart is to delimit upon the chest wall the entire (exposed *plus* covered) dulness of the heart (Fig. 105), which should correspond, as accurately as the limitations of the method of examination will permit, to the projection of the heart upon the anterior wall of the thorax (Fig. 101). Inspection of the outline obtained enables one to determine the shape, size, and situation of the heart, provided pericardial effusion is absent. If effusion is present, one simply outlines a bag of fluid, as in the abdomen an ovarian cystoma or a distended bladder is outlined by dulness on percussion.

Choice of Percussion Methods.—There are three methods of percussion which may be employed in ascertaining the areas of cardiac dulness: *first*, ordinary percussion; *second*, auscultatory percussion; *third*, threshold percussion (GOLDSCHEIDER, EWART).

These methods differ in accuracy, none being absolutely reliable in enabling the observer to obtain the true projection outline. The first method is of little or no use; the second and third are clinically trustworthy. It is proper to state that in percussing the heart the personal skill of the examiner has much to do with the result, as correctness depends largely upon the detection of very slight differences of sound and feeling.

There is another method of obtaining the projection outline of the heart which is superior to percussion, and is as nearly accurate as can be conceived, but which at present is in limited use, largely on account of the expensive apparatus and special training required in its employment—viz., the Röntgen ray and the fluoroscope. Orthodiagrams, obtained by devices which employ parallel rays only, afford a knowledge of the size and shape of the heart not otherwise to be secured. But such work is possible only for the expert X-ray specialist.

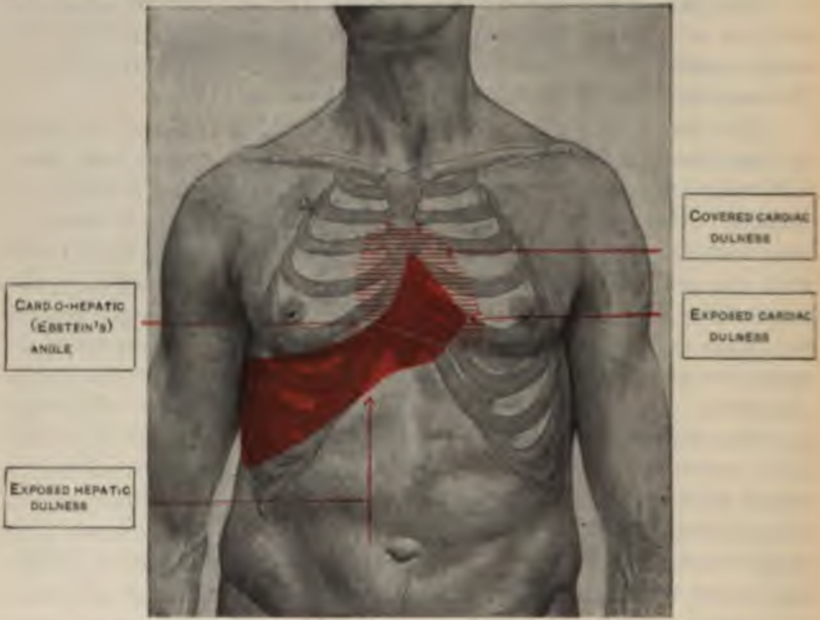

Fig. 105.—Showing the exposed and covered dulness of the normal heart.

Technic of Cardiac Percussion.—In percussing the heart by either of these methods the general direction of the lines along which the percussion strokes are conducted is the same.

(1) In using *ordinary percussion* to determine as fully as possible the entire cardiac dulness, first find the upper limit of the covered hepatic dulness by percussing from the 2d right interspace, at or somewhat outside of the parasternal line, vertically downward

(1, in Fig. 106 to which subsequent numbers refer) with heavy strokes, until the dulling effect of the upper border of the liver is noted, sometimes at the 4th space, usually at the 5th rib or interspace. Determine also the line of exposed (absolute) liver dulness, generally at the 6th rib. Then percuss successively from right to left, beginning sufficiently far out to get unmixed pulmonary resonance, along lines 2 and 3 until the first trace of impaired or modified resonance is noted. It can not be too strongly emphasized that one does not search for flatness or even dulness, but for the first trace of impairment or variation in the pure pulmonary sound. Then on the left side percuss from above downward in the parasternal line, 5, and from left to right along lines 6, 7, and 8, in the 3d, 4th, and 5th interspaces respectively, noting in each case the points of beginning impaired resonance, and exercising care not to mistake the muffling of the pulmonary resonance caused by the thick pectoral muscle for the dulling effect of the heart. The mammary gland must be held aside for a similar reason. Percussion is to be made along as many other additional lines as the examiner may think best.

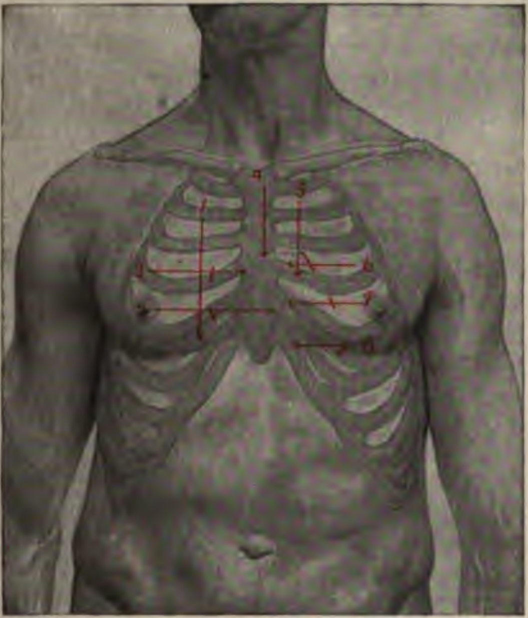

Fig. 106.—Showing the principal lines (numbered in order of performance) along which percussion should be conducted to ascertain the area of cardiac dulness, both covered and exposed. Compare with Fig. 105.

Percussion along line 4, from above downward, along the left half of the sternum, is intended to discover the line of demarcation between the base of the heart and the origin of the great vessels. Always compare rib with rib and interspace with interspace, in order to equalize the comparison.

To find the exposed dulness, percussion with light strokes is carried still farther to the left on line 2, farther to the right on lines

7 and 8, and from above downward about an inch to the left of the left sternal edge on line 9 until, on each line, the modified pulmonary resonance or covered dulness is replaced by the absolutely dull sound of the exposed heart.

(2) *Auscultatory percussion* and its technic (*q. v.*) has been described elsewhere. For the heart the tip of the stethoscope should be placed above and to the inner side of the apex beat, so that it surely rests over the heart. Percuss from all sides toward the stethoscope, making one outline with heavy strokes, and then repeating with light strokes, but never intermingling light with heavy.

It is claimed by the advocates of this method that the lower border of the heart can usually be determined without much difficulty, except where the costal cartilages and the ensiform appendix are close together and projecting. The upper limit under the sternum is harder to obtain on account of the osseous resonance, particularly when the patient is thin and the sternum prominent, but with repeated trials it is said that a sufficient change of sound can be observed to mark the line of separation between the cardiac and great-vessel areas.

Of late years, judging mainly from personal observation and experience, auscultatory percussion in general, and that of the heart in particular, is little used. I believe Goldscheider's method, "threshold percussion," to be the best for determining the cardiac dulness.

"*Threshold*" *percussion* has, with me, supplanted the Sansom pleximeter described in former editions. The accuracy of this method (GOLDSCHEIDER'S orthopercussion) of outlining the heart has been repeatedly confirmed by X-ray examination.

The two important items of this mode of percussion are the very light stroke employed and the direction of the stroke. The middle finger is used as a pleximeter. It is flexed to a right angle, at the junction of the proximal and middle phalanges, keeping the middle and terminal phalanges in a straight line. Those whose joints are sufficiently free of motion may, preferably, flex the finger at the junction of the middle and terminal phalanges (Fig. 107). The rigid, flexed portion of the finger is placed upon the chest-wall, not at right angles to the surface of the latter, but parallel to the anteroposterior (sagittal) diameter of the thorax (orthopercussion). The stroke is delivered upon the proximal end of the middle phalanx. It must be so light that, with the ear close to the percussing fingers, it can just be heard—*i. e.*, the sound produced is on the "threshold of

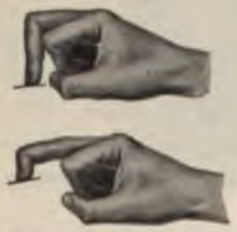

FIG. 107.—Two modes of using the finger as a pleximeter.

audibility." Percussion is to be conducted along the usual lines from lung to heart (Fig. 106, page 353).

In percussing from lung to heart by this method it is most interesting to note the sudden change from the faintly heard but unmistakable pulmonary resonance to the toneless, dull, and still fainter sound which announces the situation of the edge of the airless heart. At each of these points along the lines of percussion a mark is made upon the skin. Finally the marks thus obtained are united as shown in Fig. 86, page 310. The use of this method has been repeatedly demonstrated to students and house physicians. It has been found that there is scarcely any one whose hearing is not sufficiently acute to perceive the change of sound. This method of percussion gives an outline which includes not only the right border, left border, and apex of the heart, but also the right and left borders of the great-vessel area above.

Limitations of Cardiac Percussion.—It is rarely possible by ordinary percussion, and always difficult by any method, to find the entire lower border of the heart, owing to the fact that its exposed dulness merges with the dulness of the left lobe of the liver, but in some cases a slight difference in the sound can be detected. A similar difficulty exists with reference to the upper border. Fortunately, the outlines which can be obtained—viz., the right border, the left border and, usually, the outline of the apex—are sufficient for most clinical purposes (Fig. 108). As a rule, the dulness covers a larger area of the chest wall than would seem, at first, to be correct. The accuracy of the special technic employed in Goldscheider's "threshold," or orthopercussion has been laboriously tested by post-mortem verification (SIMONS) and orthodiagraphy (DIEBLEN). Much depends on adequate practice and the requisite manual and aural delicacy. It may, therefore, be affirmed that for ordinary clinical purposes cardiac percussion, if carefully performed, is sufficiently accurate in determining abnormal increase in the size of the heart, especially if conjoined with equally careful palpation.

Indications from Abnormal Areas of Dulness.—Having determined cardiac and great-vessel dulness, it remains to discuss the meaning of observed variation from the normal in shape, size, and position.

(1) **Enlargement of the Normal Outline.**—An increase, mainly to the *left and downward*, with a heaving apex beat displaced in the same direction, is characteristic of hypertrophy and dilatation of the left ventricle. The apex outline is unusually pointed (Fig. 109). It is quite certain that if the entire cardiac dulness is increased, dilatation as well as hypertrophy is present. It is difficult to recognise enlargement unless the heart weighs at least 12½ oz. (400 grms.).

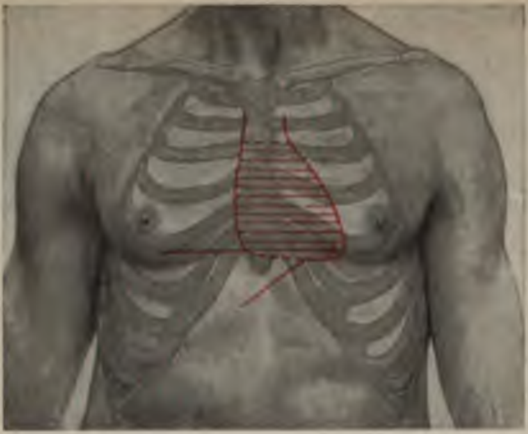

Fig. 108.—Normal
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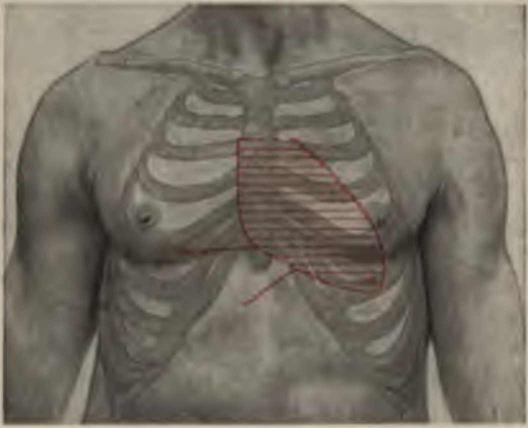

Fig. 109.—Dulness in
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Apex pointed.

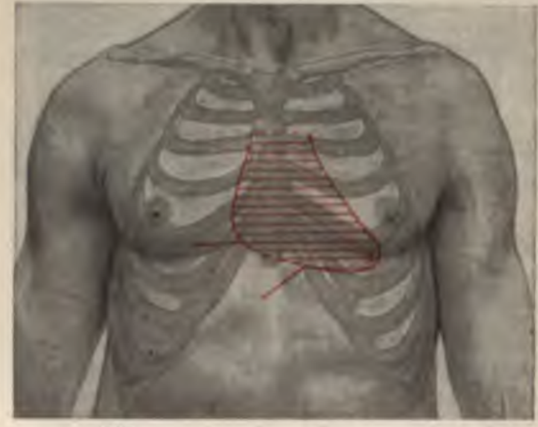


Fig. 110.—Dulness in
trophy and dilata
the right heart.
apex beat moved
left, and dulnes
creased to the ri
the sternum.

FIG. 111.—Showing the dulness due to dilatation and hypertrophy of *both ventricles*. Apex rounded and apex beat diffused. Compare with Figs. 109 and 110. Shows also (first and second right interspaces) the dulness of aortic aneurism.

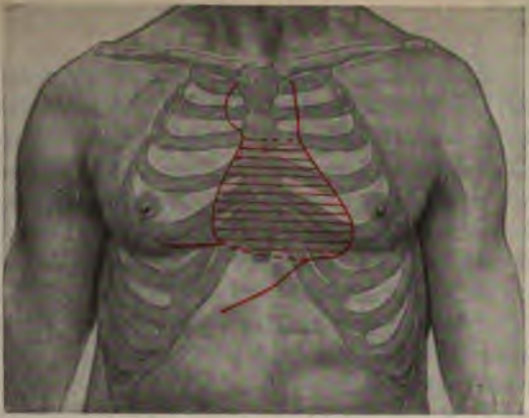


FIG. 112.—The triangular area of dulness due to a *large* pericardial effusion is shown by the outer solid line. For comparison the normal cardiac dulness is shown by the inner shaded area. Note position of apex beat with reference to the pericardial dulness.

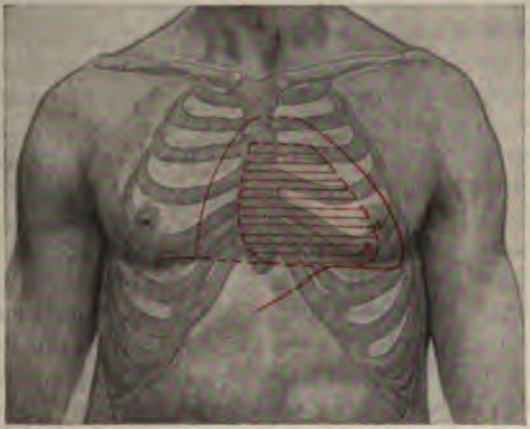
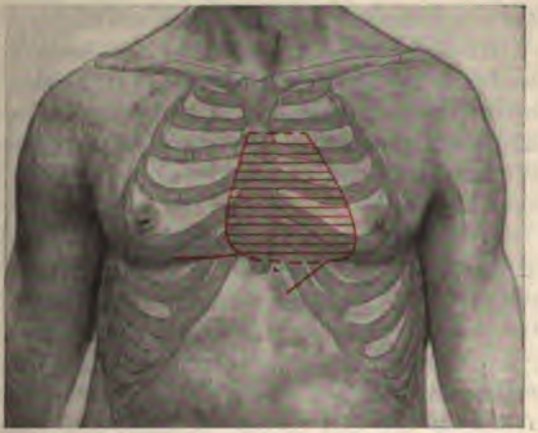


FIG. 113.—Showing the dulness of a *moderate* pericardial effusion.



Enlargement, mainly to the *right* (Fig. 110), with a rounded apex outline, is significant of hypertrophy and dilatation of the right ventricle and auricle, especially if there is dulness in the 3d and 4th spaces to the right of the sternum. If the usually resonant (Ebstein's) angle in the right 5th space, between the lower portion of the right border and the upper limit of hepatic dulness (Fig. 105), is obliterated, it may be due to a dilated right auricle, dilated inferior vena cava, or pericardial effusion.

Enlargement, both to *right and left*, with a weak and diffused apex beat, indicates much dilatation as well as hypertrophy of both ventricles (Fig. 111).

Enlargement to the *right*, *left*, *and upward* is found as a result of pericardial effusion. It is somewhat triangular or pear shaped, with the small end upward, often entending into the 2d interspace. It is a diagnostic sign that the edges of the dull area are sharply marked, the transition from pulmonary resonance to fluid flatness being noticeably abrupt. The area of dulness is somewhat larger when the patient is sitting than when lying down. As the overlapping lungs have been pushed away, there is uniform dulness over the entire area, and the distinction between the covered and exposed dulness disappears. In very large effusions there may be an area of dull tympanitic resonance in the left axillary region, below the level of the nipples and over the left base, due to compression of the left lung. The apex beat, or, if this can not be felt, the point of greatest intensity of the first sound, is well inside of the dull area, rarely outside of the mammillary line (Fig. 112). It is at times difficult and even impossible to distinguish between a moderate pericardial effusion and extreme dilatation of both ventricles (compare Figs. 113 and 111). In addition to the points above given, the differential diagnosis is discussed elsewhere (Index—Heart, dilatation of).

An apparent enlargement of the heart by inspection alone may be due to retraction of the lung, or to a pushing forward of the heart by tumour or aneurism in the posterior mediastinum; but, as the enlargement affects only the exposed cardiac dulness, a determination of the entire cardiac outline will reveal it to be apparent and not actual.

(2) **Diminution of the Normal Area.**—This may be due to atrophy of the heart or absorption of fat in wasting diseases, notably consumption and long-continued typhoid fever. Left pneumothorax, the air-containing pleural sac overlying the heart, may substitute a tympanitic sound for a portion of the normal modified pulmonary resonance; marked emphysema greatly increases the difficulty in obtaining the full cardiac outline; and gaseous distention of the

stomach may prove deceptive for similar reasons. Two rare conditions, pneumo-pericardium and emphysema of the mediastinum, may be responsible for great diminution in the size of the heart dulness.

(3) **Displacement of the Area.**—Dislocation of the heart does not usually alter the size of its outlines, but the causes of such displacements (already discussed, page 343) often render it difficult to properly percuss the heart—e. g., large pleural effusion. In such a case one must estimate the place of the heart by the position of the apex beat. In the infrequent examples of transposition of the heart (dextrocardia) the heart dulness gives a "mirror image" of the usual outlines.

(4) **Great-vessel Dulness.**—If the aorta and pulmonary artery are of normal size, the dulness (or modified vibrations) to which they give rise in plessimetric percussion does not extend beyond the edge of the sternum to either side. If the dulness extends to the right of the sternum into the 2d, or 1st and 2d interspaces, with a rounded projecting outline (Fig. 111), it is good evidence of aneurism of the aorta. Lesser degrees of dilatation may be shown in a similar manner by a correspondingly smaller increase in the area and alteration in the shape.

C. AUSCULTATION OF THE HEART

The object of auscultation is to determine the character and rhythm of the heart sounds, and the presence or absence of adventitious sounds.

Position of Valves and their Areas of Audibility.—In addition to the facts already stated with reference to the valvular element of the heart sounds (*q. v.*) a further analysis will show that each sound may be separated into 2 components. Thus the valvular portion of the 1st sound results from the closure of 2 valves, the mitral and tricuspid; the 2d sound also from the closure of 2 valves, the aortic and the pulmonary. The 1st, therefore, consists of 1 muscular and 2 valvular components; the 2d of 2 valvular components alone. Depending upon the location of the valves and the greater degree of audibility of their respective closures in certain directions, it is practicable to auscultate each one separately. Of the 4 valves, the pulmonary and tricuspid, belonging to the right ventricle, lie nearer the surface than the aortic and mitral, which are more deeply seated. Taking them in order from above downward, their positions are as follows (Fig. 114):

(1) *Pulmonary Valve.*—Mainly behind 3d left costal cartilage. Of the 3 segments comprising this valve, 2 are anterior and 1 posterior.

(2) *Aortic Valve.*—Behind the left half of the sternum on a le with the 3d space. Of the 3 segments 1 is anterior and 2 poster

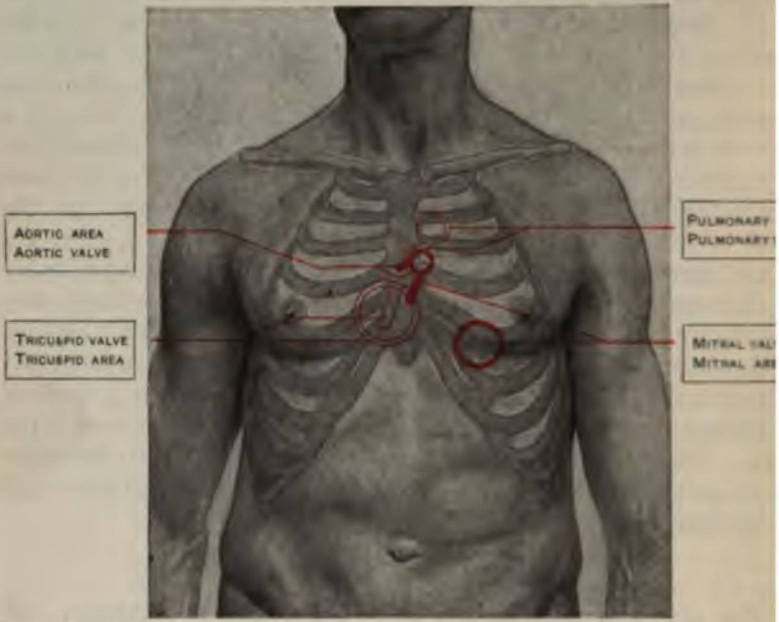


FIG. 114.—Showing the positions of the valves of the heart and the areas of their gre audibility. Solid circles and blocks = deep valves (aortic and mitral). Light ci and blocks = superficial valves (pulmonary and tricuspid).

(3) *Mitral Valve.*—Behind the left half of the sternum on a le with the 4th cartilage, 4th space, and upper border of the 5th ca lage. Of the 2 segments of the valve 1 is anterior, the other poster

(4) *Tricuspid Valve.*—Behind the lower 4th of the sternum the right of the middle line, from the 4th right cartilage to a po behind the junction of the 6th right cartilage to the sternum.

It will be noted that a stethoscope placed directly over the g eral valve area will receive auditory impressions from them all, the sounds of the 2 deep valves must pass through the cavities of heart which overlie them. Consequently the separate sound of e valve is sought for at a point where the chamber or vessel to wh it belongs approaches most closely to the chest wall and at the sa time is most distant from the other chambers or vessels. Th points or areas of auscultation at which the sound of the correspo ing valve is loudest and most distinct are as follows:

(1) *Pulmonary Area.*—At the 2d interspace to the left of sternum, upon the pulmonary artery and above the pulmonary val

(2) *Aortic Area.*—At the 2d interspace to the right of the sternum, upon the aorta and above the aortic valve. The 2d right cartilage is often called the aortic cartilage.

(3) *Mitral Area.*—At the apex of the heart where the tip of the left ventricle approaches the chest wall.

(4) *Tricuspid Area.*—Over the lower end of the sternum, a point at which the right ventricle lies close to the chest wall and at the same time is most distant from the left ventricle.

In auscultating the heart a convenient order is, first, the mitral area; then the aortic, giving the character of the left ventricular valve sounds; then the pulmonary and tricuspid areas, belonging to the valves of the right ventricle.

It is essential during auscultation to place a finger upon the apex beat or upon the carotid pulse in order to place the sounds with reference to the events of the cardiac cycle. The radial pulse follows the apex beat at a slight interval, but long enough to make it untrustworthy for careful timing. As the respiratory sounds may obscure or complicate faint cardiac sounds or murmurs, it should be made a routine practice to direct the patient to suspend breathing, one or more times, during the examination.

Variations in the Intensity and Character of the Heart Sounds.—The heart sounds are to be studied at each of these points with reference to their intensity, quality, and rhythm; also the presence of reduplication.

It is possible with care to separate the muscular component of the first sound into 2 portions, 1 furnished by the left ventricle, heard a little to the left of the apex; and that coming from the right, heard over the middle of the sternum at the level of the 3d interspace. While of equal intensity, the muscular sound of the left ventricle is appreciably longer than that of the right. The following modifications of the heart sounds are of clinical value, but require for their recognition a thorough familiarity with the normal sounds.

(1) **Accentuation of Both Sounds.**—Increased loudness or accentuation of both sounds of the heart is very common. In well-marked cases the sounds may be heard over the entire chest in front, and sometimes over the back of the thorax. It is frequently due to mental excitement. An apparent increase in the strength of the sounds may be present in persons with thin chest walls, or in whom the heart is uncovered by phthisical shrinkage of the lung; both of these conditions permitting a more ready transmission of the sounds. Overactivity of the heart, as in cardiac neuroses (e. g., palpitation), exophthalmic goitre and the early stage of fevers, causes an unnatural loudness of both sounds. Certain conditions of debility, notably

anæmia and chlorosis, may be attended by loud heart sounds, which, despite their intensity, possess a short and flapping quality, indicative of weakness rather than strength. On the other hand, the sounds may be loud because of cardiac hypertrophy, the abnormally strong muscular walls imparting unusual vigour to the closure of the valves, and adding strength to the muscular element of the first sound. Consolidated lung may, by acting as a better conductor of sound than healthy lung, increase the loudness of the sounds. Excessive distention of the intestines and stomach, the latter especially, and, less frequently, pneumothorax or a large, smooth-walled pulmonary cavity, or the rare condition of pneumopericardium may, by their resonance, impart a loud, ringing, metallic quality to the heart sounds.

Comparison of the sounds may show that one sound, or one of its components, may predominate; consequently, the observer may find—

(2) **Accentuation of the First Sound.**—Normally the first sound, when auscultated at the apex, is louder than the second sound. If it is heard more distinctly at the base than is the second sound, it is either accented, or the second sound is abnormally weak. An unusually loud, prolonged, thumping first sound at the apex is heard in hypertrophy, especially of the left ventricle. The peculiar quality of this sound is due to its large muscular component. On the other hand, a loud but short, flapping first sound having a marked resemblance to the valvular quality of the second sound and lacking the prolonged dull muscular component, is associated with moderate dilatation and weakness of the heart chambers. If the dilatation or weakness is extreme, the accentuation disappears. An uncommonly sharp and clear first sound is heard in cases of mitral stenosis.

(3) **Accentuation of the Second Sound.**—Normally the 2d sound is louder at the base than at the apex. If this relation is reversed it means either an accented 2d sound or a weakened 1st sound. In judging the 2d sound its 2 components, aortic and pulmonary, should be carefully compared by repeated auscultation of the aortic and pulmonary areas. Under thirty years of age the pulmonary closure is louder than the aortic; during middle age they are equal; at sixty years of age, because of degenerative changes, the aortic closure sound predominates. Allowing for age, it may be found that—

Aortic Closure is Accentuated.—One of the principal causes of a loud and sudden aortic 2d sound is the presence of high arterial pressure, such as may exist in nephritis and arteriosclerosis. It has been compared to the popping of a cork when pulled from a bottle.

A loud, distinct, but somewhat harsh aortic sound, perhaps of a ringing metallic or clicking quality, is indicative of sclerosis or atheroma of the aortic valves and the adjacent portion of the aorta, without narrowing or incompetence. The significance of this sound is often overlooked, and yet, as I have repeatedly demonstrated by autopsy, it is a most reliable sign. It is most frequently encountered in old labouring men. Aneurism of the aorta causes a loud aortic closure, accompanied by a palpable "diastolic shock."

Accentuation of the aortic closure is also evidence of left side hypertrophy. The strong contraction of the left ventricle distends the aorta so forcibly that the rebound closes the aortic valves with unusual force. Indeed, the majority of cases with an accentuated aortic sound, except when due to a temporary heightening of the arterial pressure, present also evidences of left hypertrophy. On the other hand, the absence of accentuation does not negative the existence of hypertrophy, for, if the latter results from aortic incompetency, the valve may be so deficient that the aortic sound may be almost nil. If marked dilatation and muscular weakness succeed to hypertrophy, the intensity of the aortic sound will lessen *pari passu*.

Pulmonary Closure is Accentuated.—The pulmonary component of the 2d sound is an invaluable index of the strength of the right ventricle and the competence of the tricuspid valve. If it is well marked it shows that the tricuspid valve is competent, and the ventricle is sufficiently strong to sustain a good blood pressure in the pulmonary artery. The causes of an unduly accentuated pulmonary 2d sound are those conditions which hinder the flow of blood through the pulmonary circuit. Thus pneumonia, emphysema, and mitral regurgitation or stenosis may be responsible for the accented sound. In certain varieties of pneumonia a well-marked pulmonic 2d sound is a favourable prognostic, and its disappearance the reverse. It may be readily seen that the presence of pulmonic accentuation is an important corroborative sign of mitral lesions, of right ventricular hypertrophy with good muscle, and of the absence of tricuspid regurgitation.

(4) **Weakening of Both Sounds.**—The thick chest wall of the obese, and in particular the large mammary gland of stout women, may cause a great diminution in the normal intensity of the heart sounds as a whole. For somewhat similar reasons, viz., the interposition of a muffling body between the heart and the stethoscope, pulmonary emphysema, pericardial effusion, or a large pleural effusion may be responsible for a very material decrease in the audibility of the sounds.

Aside from these, the main significance of weakened heart sounds is a weak heart muscle from various causes, such as the broken compensation of valvular lesions, degenerative disease of the heart muscle, dilatation of the heart, exhausting disease (especially long-continued fevers and septic affections), hemorrhage, shock, and overexertion; or pneumogastric paralysis, nuclear or peripheral.

(5) **Weakening of the First Sound.**—The causes of a weakened first sound are for the most part those which have just been enumerated as producing weakness of both sounds. But, as any weakness of the cardiac muscles is apt to manifest itself primarily by weakening of the muscular component of the 1st sound, weakness of the 2d sound appearing subsequently, the character of the 1st sound is of the greatest importance in estimating the strength of the heart.

In exhausting fevers one may observe day by day a progressive decline in the strength of the 1st sound, its muscular component gradually lessening until the 1st sound bears a close resemblance to the 2d, or in bad cases becomes almost inaudible. In rare instances nothing but the short 2d sound may be heard at the apex, the 1st sound practically disappearing. The left ventricle is usually the first to yield. A less marked weakness of the 1st sound is noticeable in a fatty or degenerated heart, and in anæmia and other conditions of debility.

(6) **Weakening of the Second Sound.**—In addition to the weakening of both components (aortic and pulmonary) of the second sound, by conditions already mentioned (4), weakening of the individual valve sounds under special circumstances needs a reference.

Aortic Closure Weakened.—This is frequently found in marked cases of mitral stenosis, the left ventricle receiving through the narrowed orifice a charge of blood which is inadequate to fully distend the aorta, and in consequence the blood pressure in the vessel is not raised to the point required to close the valve with normal sharpness. In aortic stenosis the stiffened segments, lacking flexibility, may not vibrate sufficiently to produce a closure sound of proper intensity. Finally, in aortic insufficiency the defects in the valve may be so great that the sound of valve closure disappears and nothing but the murmur is heard over the aortic area.

Pulmonary Closure Weakened.—This is a significant, valuable, and almost the only physical sign of the giving way of the right ventricle. The disappearance of a good or accentuated pulmonic second sound in pneumonia or any condition attended by increased resistance in the pulmonary circuit indicates one or both of two things—failure of the right ventricle or tricuspid insufficiency.

(7) **Reduplication of the Heart Sounds.**—In certain cases auscultation reveals a triple instead of the normal double sound of the heart, due to a doubling or reduplication of either the first or the second

sound. In rare cases both sounds may be doubled, causing four sounds. If the first sound is doubled, the normal *lubb-dupp* is replaced by *lublubb* —— *dupp* (pronouncing the duplicated syllables quickly together); or by lubb —— dupdupp if the reduplication, as most commonly found, affects the second sound. If these sounds are so accented as to resemble the cadenced canter of a horse, it is called the "*bruit de galop*," or galloping rhythm. In the form of reduplication which goes under this name the doubling usually affects the second sound while the accent is upon the first sound, lubb —— dupdupp, one long and two short (–́ ⏑ ⏑), or upon the third (⏑ ⏑ –́).

There are many variations in time and accent, and quite as many largely theoretical explanations of their exact causation. In the presence of contradictory opinions one may be permitted to select the simplest explanation extant, which is that the reduplicated sounds are due to non-simultaneous closure of the valves, the segments of one coming together a short but appreciable time before those of the other. In the case of the mitral and tricuspid valves the closure is successive and not simultaneous, because the systoles of the ventricles are asynchronous; while the closure of the aortic and pulmonary valves is not synchronous because of heightened pressure either in the aorta or the pulmonary artery, the valve exposed to the higher back pressure closing first; but this theory does not fit all cases.

Clinically, reduplication with galloping rhythm is found not infrequently in arteriosclerosis and chronic interstitial nephritis, conditions in which there is heightened arterial pressure. It is very characteristic of mitral stenosis and diseases of the lungs which cause an increase of blood pressure in the pulmonary circuit. It is found especially in cardiac dilatation and broken compensation; and in diphtheritic paralysis of the heart, pericardial effusion, and the rapid heart action of exophthalmic goitre.

(8) **Alterations in the Relative Length of the Silences.**—In a normally acting heart, the two sounds and the long silence follow one another in triple time—i. e., 1st sound (one), 2d sound (two), long silence (three), 1st sound (one), etc. Even if the heart is acting rapidly this rhythm is preserved.

In disease there are two variations (Fig. 115) which are of importance as indicating serious weakness of the cardiac musculature. The first is embryocardia, so called because it approximates the character of the fœtal heart sounds. The 1st sound resembles the 2d sound very closely, and the short and long silences become of equal length, so that the rhythm is in double time—1st sound (one), 2d sound (two), 1st sound (one), etc. It is very like the tick-tack of a watch or the regular rapid click of a short pendulum. It is a grave prognostic omen, as it indicates a profound enfeeblement of the cardiac muscle, such as occurs in the specific infectious fevers or in chronic degenerative disease of the myocardium.

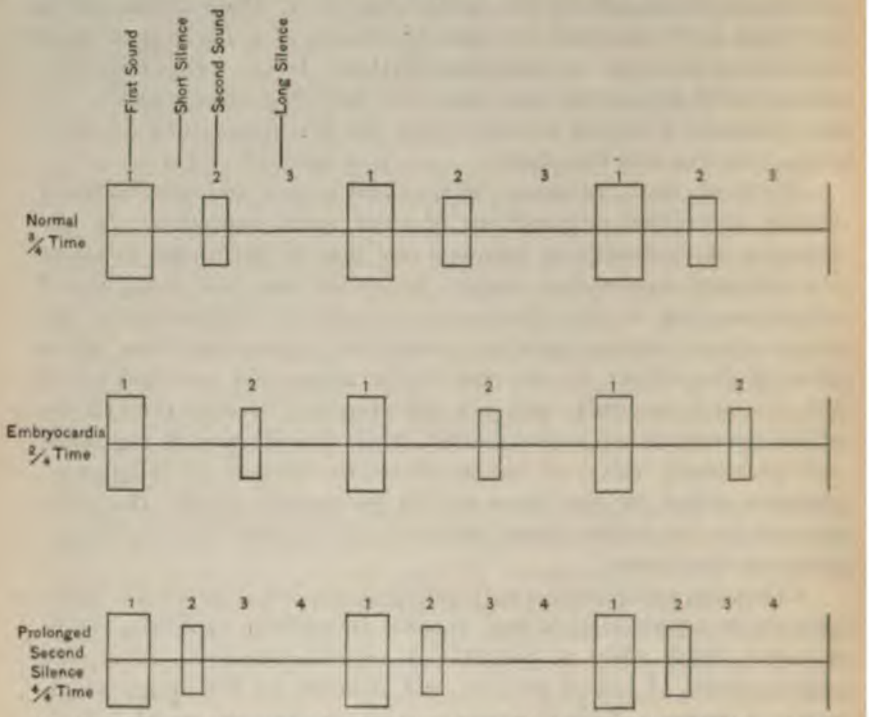


FIG. 115.—Diagram representing two variations from the normal cardiac rhythm. To be read from left to right.

The other variation is an undue prolongation of the second or diastolic silence. The 1st and 2d sounds are short and irritably sharp, and the interval between the 2d and 1st sounds is almost alarming in its length. One may say that it is in common (4–4) time, 3 and 4 representing the pause. The unusual prolongation of the diastolic silence indicates one of two things: either an overdose of digitalis, or the spasmodic effort of an overworked, weak, or degenerated heart to continue its labours. In the latter case it is not of necessity a fatal prognostic. It has been met with more particularly in pneumonia.

Adventitious Sounds.—In addition to or in place of the variations in the ordinary heart sounds which have been considered, auscultation may reveal the presence of certain incidental or adventitious sounds. The sounds may originate within the heart—*endocardial;* or arise from outside the heart—*exocardial.* An endocardial sound is called a murmur or bruit, and is produced at one of the orifices of the heart as a result of certain conditions of the valve, the orifice, the vessel, the blood, or the force and velocity of the blood stream. Murmurs caused by permanent anatomical changes in the valves or orifices are called *organic;* if due to blood changes or modifications of cardiac muscular action, *functional, accidental,* or *hæmic* murmurs. Exocardial sounds for the most part originate in the pericardium, pleura, or lung.

ENDOCARDIAL SOUNDS (MURMURS)

The Physical Explanation of Murmurs.—The physical conditions are somewhat complex. The most important factors are:

(1) When a fluid under a certain degree of pressure is forced from a narrow into a wide channel, or through a narrowed opening into a relatively large cavity, a *fluid vein* is formed, the particles of which are in a state of rapid vibration. If the necessary conditions are produced in the heart, either by the narrowing of an orifice leading into a larger but normal cavity or by a dilatation of the cavity, the orifice remaining normal but relatively narrowed, the vibrations of the resulting fluid vein will throw the walls of the containing cavity or tube into lateral vibrations which are conducted to the surface of the body. In rare cases the vibrations may be so strong that sound waves are produced at the surface of such intensity as to be heard at a distance of several inches from the surface. Ordinarily the ear must be placed in contact with the surface, or some conducting medium (stethoscope) used which will carry them to the ear, in which they are translated and perceived as sounds. If the edges of the opening, as in the orifices of the heart, are furnished with projecting membranous flaps (valve segments), these, by their capacity for being thrown into periodic oscillation, add to the number and intensity of the vibrations, and consequently modify very considerably the character of the sounds perceived.

As a rule, vibrations originating in this manner travel most rapidly in the direction of the fluid vein. Murmurs due to a narrowed orifice move *with* the blood stream (the fluid vein running in the same direction) and are heard further along in its course—e. g., aortic stenosis, in which the murmur is heard also in the carotids; while in regurgitation the murmur travels *backward* in the blood stream (the fluid vein opposing the main current)—e. g., aortic regurgitation, in which the murmur is heard also over the lower sternum.

(2) The *density* or relative fluidity of the blood is a second factor. The thinner the blood (the less its specific gravity), the more readily is it thrown into vibration. Hence, partly at least, the great frequency of murmurs in anæmic states.

(3) A certain *force of current* is required. Thus a murmur may be loudly audible so long as the heart is beating strongly, but will diminish or totally disappear if great cardiac weakness supervenes.

The General Characteristics of Murmurs.—It is necessary to study a murmur with reference to its time relations, tone and quality, point of maximum intensity and lines of propagation; and also endeavour to decide whether it is organic or functional.

(*a*) **The Time of Murmurs.**—Murmurs are timed with reference to the ventricular systole (first sound, apex beat, or carotid pulse) and the ventricular diastole (beginning with the second sound). Endocardial murmurs always bear a definite relation to the heart sounds. Three types are distinguished: *Systolic*, beginning with the first sound; *diastolic*, beginning with the second sound; and *presystolic*, immediately preceding the first sound.

The presystolic murmur is in reality diastolic, occurring as it does during the auricular systole, which takes place late in the ventricular diastole, but it is very desirable to restrict the term diastolic to murmurs *beginning* with the second sound. These chronological types are represented by the graphic method in Fig. 116.

(*b*) **The Quality of Murmurs.**—The character or quality of a murmur, while its diagnostic value is not great, is often of service, and should always be noted.

In general, a rough, harsh, rasping, or sawing murmur (in mitral stenosis, blubbering or churning) attends obstruction or stenosis of an opening or roughening of the valve surfaces; while if the murmur results from insufficiency or regurgitation it has a soft and blowing quality. The murmur of aortic regurgitation may be so soft as to escape detection except by a careful examiner. A musical murmur, one having an element which resembles a high-pitched musical note, has no special significance with reference to any particular heart lesion. It may be due to a perforated valve or shreds or threads of fibrin. In many cases nothing exists to account for its peculiar *timbre*. Murmurs having a peculiar echoing quality are due to the same causes as metallic heart sounds (*q. v.*), the near presence of a resonating air cavity.

(*c*) **Intensity of Murmurs versus Replacement of Sounds.**—The loudness or intensity of a murmur is by no means an index of the gravity of a valvular lesion. A murmur which is well-nigh inaudible is oftentimes indicative of vastly greater damage than one of striking intensity. A judgment with reference to the seriousness of a valvular lesion must be founded on other evidence—e. g., the presence or absence of hypertrophy and dilatation, indirect symptoms (page 336), etc.

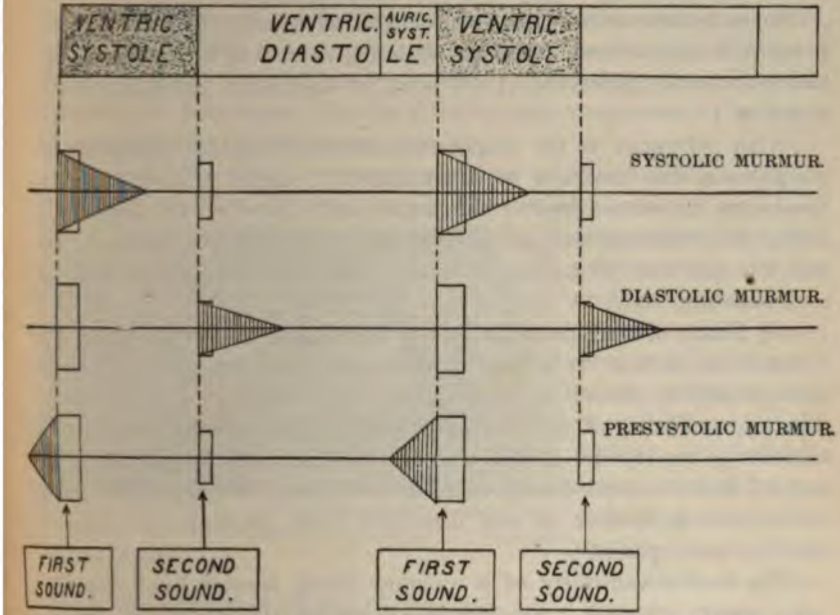


FIG. 116.—Diagram showing the three chronological types of murmurs and their relation to the sounds of the heart. The events of the cardiac cycle are given above for comparison. Murmurs shaded with vertical lines. To be read from left to right.

One of the principal points to be determined, especially with reference to regurgitant murmurs, is whether the murmur *accompanies* or *replaces* the valvular sound or component. Replacement indicates material and important anatomical changes in the valve. In such a case the sound of valve tension is absent. Thus, if the aortic valve is seriously incompetent, the snap of its valve segments will largely disappear and be replaced by the murmur. On the other hand, if the valvular sound is present and the murmur simply trails off from it, we may infer that a goodly portion of the valve action is preserved and that the actual physical alterations in its shape and structure are not extreme.

The loudness of a murmur is dependent partly upon the nature of the lesion, partly upon the strength of the heart. As a rule, systolic murmurs are louder than diastolic, particularly those originating at the aortic orifice. An extremely weak heart is incapable of maintaining the degree of force of the blood stream which is requisite to produce strong vibrations and a resulting loud murmur. Hence, as a rule, a loud murmur implies fair compensation, and a very loud murmur which has become weak may be restored to its former intensity by rest and treatment of the heart muscle.

Some murmurs may be felt as well as heard (thrill, *q. v.*). In general it is the loud murmurs which give rise to a thrill, and the latter is usually perceived at the point of maximum intensity of the murmur.

With reference to the respective effects of *posture* (standing or lying) upon the loudness of murmurs, it is stated (GORDON) that recumbency increases hæmic murmurs, and those of tricuspid and mitral insufficiency and aortic stenosis; decreases the venous hum and the murmur of mitral stenosis; and does not affect that of aortic insufficiency.

(*d*) **Point of Maximum Intensity and Lines of Propagation.**—A murmur is heard at its loudest at some one point, the point of maximum intensity, which is usually, but not always, in the area of greatest audibility of the normal sound of the valve at which the murmur is produced. It will also be found that it can not be heard equally well at equal distances from this point, but that it is transmitted much farther in one direction than another—the line of selective propagation.

The better audibility of a murmur along special lines depends upon several factors. As previously stated, the sound is apt to travel in the direction of the vibrating fluid vein, either with or against the main current. Another factor is the presence of structures differing in their power of conductivity—e. g., the sternum, ribs, walls of the heart, and the presence or absence of consolidated lung or lung cavities. Moreover, the relation of the cavity in which the murmur originates to the chest wall (in contact or at a distance) will modify the readiness with which the sound reaches the ear. Special lines of transmission will be discussed in connection with murmurs arising at particular orifices.

(*e*) **Discrimination between Organic and Functional Murmurs.**—It is of prime importance to distinguish between temporary, recoverable, functional murmurs, not due to valvular disease, and organic murmurs, caused by permanent and incurable stenosis or incompetency. The exact genesis of functional (hæmic, anæmic, accidental) murmurs is not settled. An abnormal fluidity of the blood is an important agency in their production. According to their location they are probably due to moderate dilatation of the pulmonary artery giving rise to a fluid vein; or to dilatation of one or the other ventricle in consequence of a poorly nourished heart muscle with resulting relative incompetency at the mitral or tricuspid opening. It is probable that in some instances the edges of the valve cusps turn over because of weakness and consequent stretching of the papillary muscles, thus permitting a slight regurgitation. Functional murmurs are most frequent by far in the pulmonary area, next in the mitral, then in the tricuspid, and occur more rarely in the aortic area. It is at times extremely difficult to determine that a murmur is functional, but the discrimination may usually be made by remembering the following points:

(1) The *functional* or *hæmic* murmur is almost always, but not invariably, systolic, soft, and blowing (sometimes harsh), found most frequently over the pulmonic area where an organic murmur is extremely rare, and a venous hum is heard in the veins of the neck. Above all, the patient is distinctly anæmic or chlorotic, or has some febrile or wasting disease, and the murmurs disappear as the condition of the blood improves. Moreover, ventricular dilatation, if ascertained to be present, is slight and unattended by hypertrophy or secondary symptoms.

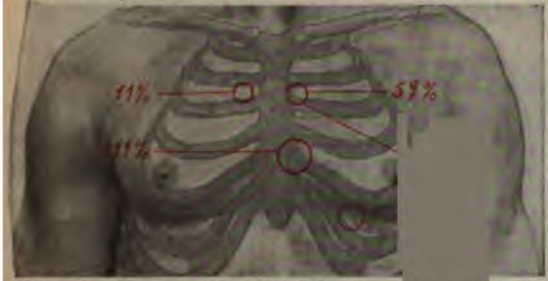


Fig. 117.—Showing the relative frequency [redacted] murmurs at the various orifices of the heart. Percentages from Sansom.

(2) The *organic* murmur, on the other hand, is systolic or diastolic, there may be marked hypertrophy or dilatation, there are symptoms referable to stasis in the blood current, and there is a history of rheumatism or other disease capable of causing endocarditis. The somewhat sweeping assertion may be made, without fear of serious contradiction, that if the apex beat is not displaced or altered in such a manner as to indicate hypertrophy or dilatation, the presence of a murmur does not signify organic valvular disease; although it is possible that pathological changes may have been initiated which will ultimately lead to anatomical valvular lesions and consequent alterations in the size of the heart.

There are certain sources of error. The history of rheumatism may be indefinite, and anæmia alone may cause dyspnœa and œdema of the feet. Finally, anæmia may occur in certain cases of valvular disease, notably mitral stenosis, as an association or a consequence of the valvular affection.

The Diagnostic Value of Individual Murmurs.—Taking the orifices of the heart singly, one proceeds to study the character and meaning of the different murmurs arising at each opening.

Mitral Murmurs.—These may be either *presystolic* or *systolic*.

(*a*) *Mitral Presystolic Murmurs.*—A presystolic murmur at this orifice usually, but not invariably, indicates:

(1) Mitral Stenosis or Obstruction.—The murmur in this case is caused by the systole of the left auricle driving the blood through the narrowed opening. The murmur begins during the latter part of the ventricular diastole with the systole of the auricle and is often accompanied by a thrill. It is harsh, rough, and vibratory, with its area of maximum intensity a little inside and above the apex beat (Fig. 118). It is usually strictly localized, but it may be heard over an area beginning at the lower end of the sternum, passing to the left, and widening until it reaches the midaxillary line, perhaps extending vertically in this line from the 3d to the 8th rib (GRIFFITH). The murmur runs up to and terminates in an abrupt 1st sound or "snap" (phonetically "rrup"), which is very characteristic. The abrupt 1st sound has been variously explained; but the most plausible theory is that which attributes it to a sudden tension of the tricuspid valve caused by the increased pressure in the right heart consequent upon the damming back of the blood by the mitral obstruction, the sound of valve tension thus predominating over the muscular component of the 1st sound. The 2d sound is frequently reduplicated and its pulmonary component accentuated. The murmur of mitral stenosis is variable, appearing and disappearing according to the strength of the auricular systole. A few moments of exercise may elicit it when previously absent. It is necessary to be aware of the fact that in rare instances the murmur

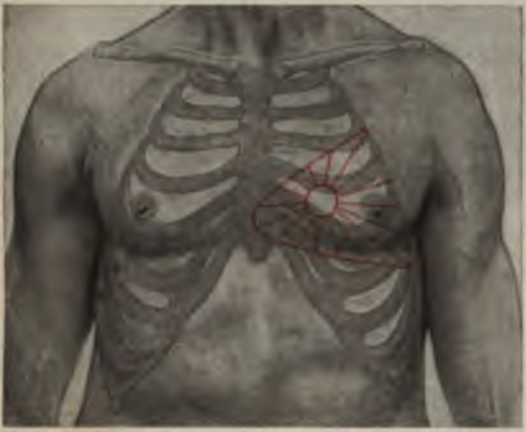


FIG. 118.—MITRAL PRESYSTOLIC MURMUR. This may be caused by mitral obstruction, aortic incompetence ("Flint" murmur), and slight aortic stenosis and adherent pericardium. The circle shows the point of maximum intensity and the usual strict localization of this murmur. The radiating lines represent its *possible* extent of audibility. Apex indicated by the cross.

of mitral stenosis may be diastolic, mid-diastolic, or occupy the entire diastole (Fig. 119), the vibrations being excited at any time during the diastole by the blood which begins to flow into the ventricle from the auricle at the close of the ventricular systole.

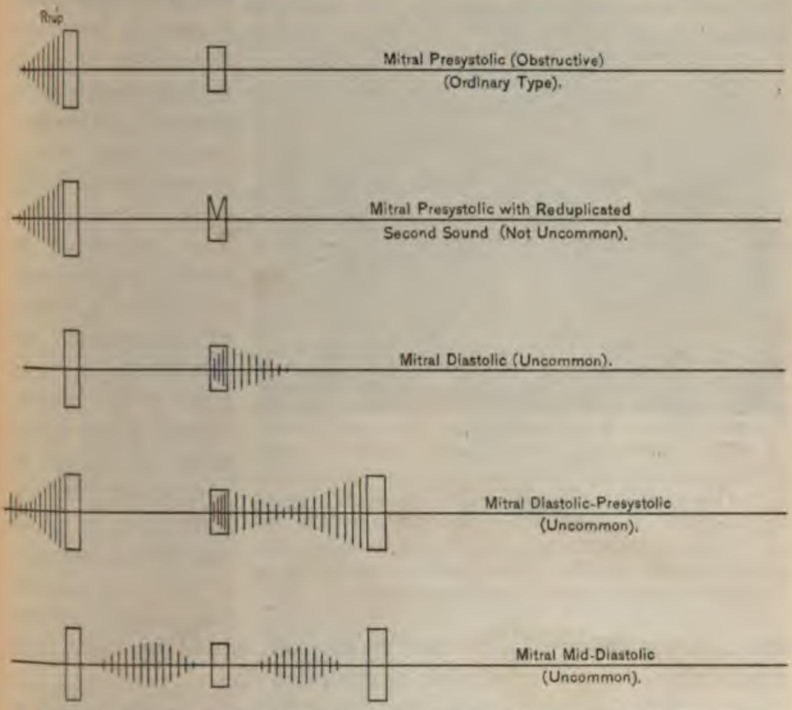

Snap
Mitral Presystolic (Obstructive) (Ordinary Type).
Mitral Presystolic with Reduplicated Second Sound (Not Uncommon).
Mitral Diastolic (Uncommon).
Mitral Diastolic-Presystolic (Uncommon).
Mitral Mid-Diastolic (Uncommon).

FIG. 119.—Diagram showing the chronological varieties of the murmur of mitral stenosis. To be read from left to right.

(2) A presystolic mitral murmur is also caused by some forms of aortic disease, particularly aortic insufficiency. This, the "Flint" murmur, is attributed to an extreme dilatation of the left ventricle preventing the cusps of the mitral valve from folding back to the ventricular walls during diastole. By remaining in the blood current a species of relative narrowing is produced and a vibrating presystolic murmur arises. The snap of the first sound is, however, absent, and if the dilatation grows less, the murmur disappears.

(3) This murmur has been noted very infrequently in connection with slight aortic stenosis and adherent pericardium.

(*b*) ***Mitral Systolic Murmurs.***—A murmur heard in the mitral area, beginning with the first sound of the heart (Fig. 120), may indicate:

(1) Mitral Insufficiency.—In this case the murmur is soft and blowing, either accompanying the first sound or replacing its valvular component more or less completely. It is transmitted through the left axillary region and posteriorly to the angle of the left scapula. In some cases it may be heard over the entire chest. Its maximum intensity is generally at the apex, but it may be most intense along the left edge of the sternum. The pulmonary 2d sound is accentuated, and there is evidence of hypertrophy and dilatation. In the absence of the last-mentioned signs a diagnosis of organic or permanent relative insufficiency of the mitral valve is untenable, as there are many systolic mitral murmurs due to a temporary relative insufficiency and caused by slight dilatation, either of the left ventricle or the valve ring.

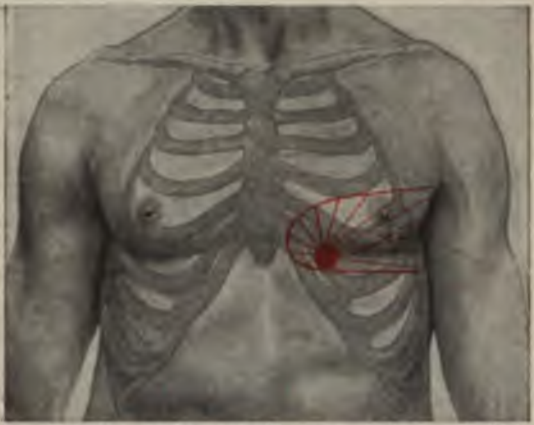


Fig. 120.—Mitral Systolic Murmur. This may indicate mitral insufficiency, anæmia, acute infectious disease (myocarditis), left ventricular dilatation, malformation of the heart, or acute endocarditis. The circle indicates the point of maximum intensity, the arrow the line of selective transmission. The radiating lines represent the area of audibility. If of sufficient intensity, the murmur may be heard over the entire chest.

It is to be borne in mind that a loud systolic murmur, heard at the back as well as in front, is sometimes present in the dilatation of the left ventricle following hypertrophy (especially that due to arteriosclerosis), and due to relative insufficiency. This murmur may totally disappear if the heart regains its strength and the dilatation is overcome.

(2) The murmur due to anæmia.

(3) The murmur occurring in the course of acute infectious or other febrile diseases, especially in children. In these cases an acute myocarditis is often responsible for temporary dilatation and consequent murmur.

(4) The murmur, varying from day to day, of acute simple or ulcerative endocarditis.

(5) The intermittent murmur, very rare (cause discovered only at autopsy) and due to a ruptured tendinous cord, or a pedunculated vegetation floating from time to time between the valve cusps.

(6) A loud and widely diffused murmur in the mitral area of a more or less permanently cyanotic infant may indicate complicated malformations of the heart, especially a defective auricular septum, patent ductus arteriosus, or transposition of the great vessels.

Any one of these murmurs may disappear if the heart becomes sufficiently weak, excepting the murmur mentioned in the second paragraph under (1) preceding.

Aortic Murmurs.—These are either *systolic* or *diastolic.*

(*a*) *Aortic Systolic Murmurs.*—If due to (1) aortic stenosis, the murmur coincides with the 1st sound (Fig. 121), is harsh, accompanied by a thrill, heard with maximum intensity at the 2d right interspace or cartilage, transmitted into the carotids, possibly into the axillary artery, and the aortic closure sound is frequently abolished as the valve cusps are too stiff to vibrate and the aortic pressure is lowered. Finally, there is left hypertrophy. If these signs occur in an elderly person, a reasonably certain diagnosis of a narrowed aortic orifice may be made. With broken compensation the murmur may become soft and distant.

(2) If the presence of a systolic aortic murmur is taken as presumptive evidence of stenosis, mistakes will be extremely common. In a majority of cases narrowing does not exist, and one of the following conditions is present: dilatation of the aorta; roughening or calcification of the aortic segments or of the aorta just beyond the valve ring; and anæmia (not uncommon). The diastolic murmur of aortic insufficiency is often accompanied by a systolic murmur due to the inequalities of the valve or opening.

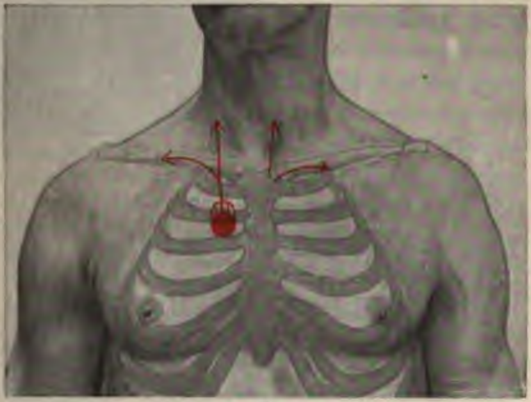


FIG. 121.—AORTIC SYSTOLIC MURMUR. This may be due to aortic stenosis, anæmia, dilatation of the aorta, roughening of the aortic segments of the aorta, inequalities of the valve in aortic incompetence, or aneurism of the arch of the aorta. Circles show the points of maximum intensity and the arrows the lines of propagation (into carotids and subclavians) of the murmur.

(3) A systolic murmur heard in or above the aortic area, or to the right of the area, may be due to aneurism of the arch, especially the ascending and transverse portions. Careful inspection and palpation of the great-vessel area should be made to discover a dull area, a thrill,

or a pulsating swelling. The maximum intensity of the murmur will in this case be over the dulness or the swelling.

(*b*) *Aortic Diastolic Murmur.*—This is the most trustworthy of all murmurs and almost invariably signifies:

(1) Aortic insufficiency, due to deformation of the valve cusps. It is a soft, long drawn out, sometimes almost inaudible murmur, beginning with the 2d sound and replacing the aortic closure sound more or less completely. Its point of maximum intensity is usually over the left half of the sternum on a level with the 4th costal cartilage (Fig. 122). It is transmitted to the lower end of the sternum, and in some cases is very distinctly audible at the apex (mitral area). Dilatation and hypertrophy always exist. Like other murmurs, it may disappear, and rarely autopsy shows that the murmur has ceased because the valve defect has been closed by a vegetation or plug of fibrin.

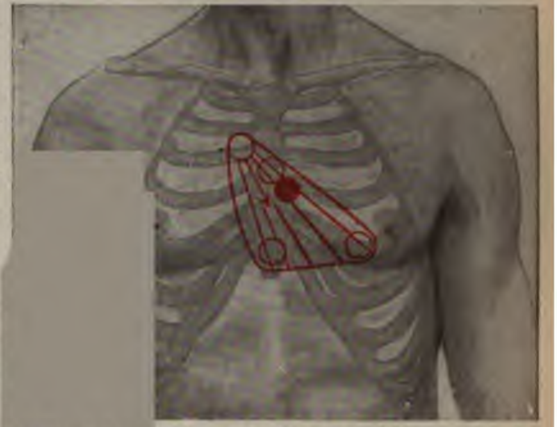


FIG. 122.—AORTIC DIASTOLIC MURMUR. This may be due to aortic incompetency, relative aortic incompetency, or anæmia (rare). The solid circle shows the usual point of maximum intensity, the white circles show the occasional points of maximum intensity, and the arrow shows the direction of selective transmission.

(2) It occurs occasionally in connection with adherent pleura and pericardium, in which case it is probably to be classed as a cardiorespiratory murmur, caused by pressure or suction of the heart upon adherent portions of the lung.

(3) It is unquestionably heard at times in extreme anæmia (1,000,000 or less red cells). In such instances the mode of its production is as yet unexplained.

(4) A diastolic venous hum originating in the internal jugular, or in the superior or inferior cava, may simulate a diastolic aortic murmur, but in such a case there will be little if any evidence of left ventricular hypertrophy or dilatation, such as would be found in aortic incompetence.

(5) Relative aortic incompetency, the aorta dilating or the sinuses of Valsalva yielding because of atheroma, inflammation or aneurism; the ventricle dilating as the result of fibrous myocarditis, general

arteriosclerosis, etc., without anatomical changes in the aortic valve, is perhaps not as uncommon as is generally supposed (EDWARDS). The simultaneous dilatation of the aorta on one side and the ventricle on the other side of the aortic orifice renders the aortic valve relatively incompetent. A musical diastolic murmur, with increase of dulness over the great-vessel area (to the right), may lead to a diagnosis. A cylindrical, greatly dilated atheromatous aorta may cause a diastolic murmur, the valve being perfectly competent. It is presumably due to the reflux of blood from the large arteries into the sac.

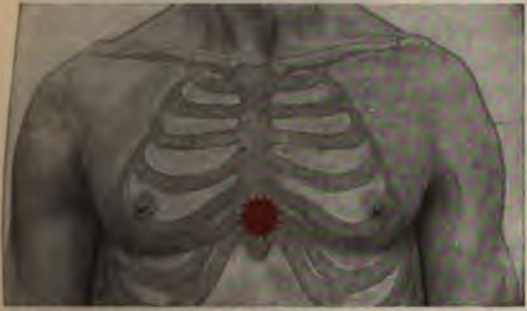


FIG. 123.—TRICUSPID PRESYSTOLIC MURMUR (rare). This is due to tricuspid stenosis, congenital or acquired.

Tricuspid Murmurs.—These are either *presystolic* or *systolic*.

(*a*) *Tricuspid Presystolic Murmur.*—This is a rare murmur and indicates tricuspid stenosis, either congenital or acquired. The acquired form is almost invariably a sequence of left heart lesions, especially mitral stenosis. The murmur (Fig. 123) resembles that of mitral narrowing, and is sometimes accompanied by a thrill. Its maximum intensity is over the lower end of the sternum at the base of the ensiform cartilage, or a little to its right. It is usually not transmitted.

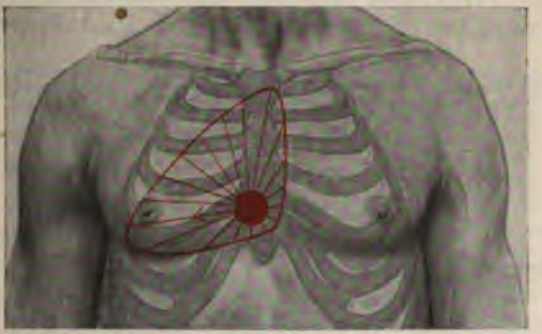


FIG. 124.—TRICUSPID SYSTOLIC MURMUR. This is significant of relative tricuspid incompetency due to dilatation of the right ventricle arising from left-side disease or anæmia. The circle shows the point of maximum intensity. It is sometimes heard over the area indicated by the radiating lines.

(*b*) *Tricuspid Systolic Murmur.*—This murmur is not uncommon and indicates tricuspid regurgitation, sometimes from puckering of the valves, but usually on account of relative insufficiency from dilatation of the right ventricle, consequent upon left-side (particularly mitral) lesions. The murmur resembles that of mitral regurgitation, but with its area of maximum intensity over the lower end of the sternum. It is usually localized, but may be propagated to the right as far as the anterior axillary line, and if the patient is recumbent it may be perceived over the manubrium (Fig. 124). Engorged and pulsating jugulars, and pulsation of the liver, are concomitant signs.

This murmur may also be significant of the slight right ventricular dilatation resulting from anæmia, but when due to blood conditions the venous phenomena are not marked and the bruit disappears as the anæmia lessens.

Pulmonary Murmurs.—These may be *systolic* or *diastolic.*

(*a*) *Pulmonary Systolic Murmurs.*—In rare cases this murmur is due to—

(1) Congenital malformation of the heart. In this case there may be a thrill, and the pulmonary 2d sound is weak or obliterated. The murmur is loud and widely diffused. If occurring in an infant with cyanosis, the abnormality is usually a pulmonary stenosis; without cyanosis, a defective ventricular septum or a patent ductus arteriosus. In a certain proportion of cases these defects coexist.

(2) In some instances a systolic pulmonary murmur is caused by traction upon or narrowing of the pulmonary artery by shrinking and contraction of the upper portion of the left lung.

(3) Almost invariably this murmur is not due to disease of the valve, but is of anæmic origin. It is a soft, sometimes slightly rough, murmur, heard best in the 2d left interspace, 1 inch or more from the sternal edge, and perhaps as high as the 1st rib (Fig. 125). It may be audible only in the recumbent position, and may disappear during deep inspiration. The pulmonary 2d sound is accentuated.

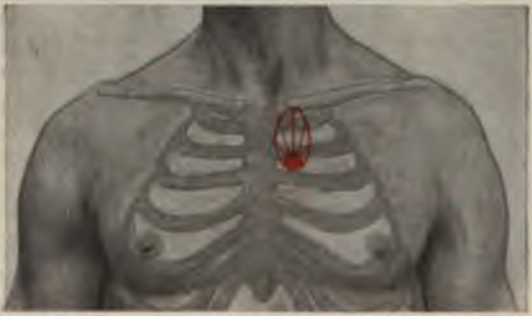


FIG. 125.—PULMONARY SYSTOLIC MURMUR. This may be due to congenital malformation, traction on the pulmonary artery, anæmia (very common), or debility. The circle shows the point of maximum intensity, the arrow the line of transmission, and the outline the usual area of audibility. This murmur, if due to malformation, may be audible over the entire chest.

It occurs in thin, nervous children, in anæmia, and in conditions of debility without notable impoverishment of the blood, but which involve slight dilatation of the pulmonary artery beyond the valve ring (relative stenosis). It is of frequent occurrence in exophthalmic goitre, or in rapid heart action from fever or muscular exertion. A cardio-respiratory murmur is often heard in the pulmonary area; so also is the transmitted murmur of mitral incompetency.

(*b*) *Pulmonary Diastolic Murmur.*—This is very rare, and, although its maximum intensity is in the pulmonary area, it is extremely difficult to distinguish it from the corresponding aortic diastolic murmur. It may be due to (1) congenital malformation of the valve, or a lesion of ulcerative endocarditis; (2) to an extreme increase of tension in the pulmonary artery, the murmur of high pressure (GIBSON, STEELL).

Combined Murmurs.—When two or more murmurs coexist it is easy to identify them if they occur at different periods in the cardiac cycle. If they are synchronous, an endeavour must be made to ascertain the point of maximum intensity and the line of propagation of each. Moreover, each murmur may have a distinctive quality of its own, which may generally be ascertained by listening along lines connecting the various orifices, thus finding intermediate areas where one quality ceases and another begins. The use of the differential stethoscope is helpful. The presence of definite circulatory disturbances and the particular forms of hypertrophy and dilatation known to be due usually to some special lesion, will aid in the discrimination. The combinations most commonly met with, and in order of frequency, are as follows (Fig. 126):

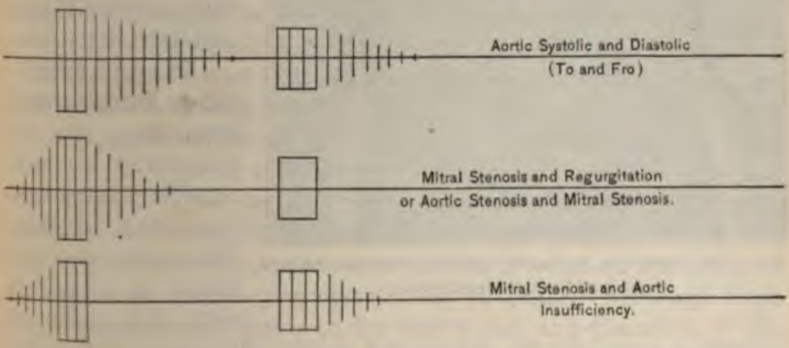

Aortic Systolic and Diastolic
(To and Fro)
Mitral Stenosis and Regurgitation
or Aortic Stenosis and Mitral Stenosis.
Mitral Stenosis and Aortic
Insufficiency.

FIG. 126.—Combined murmurs.

(1) Aortic Regurgitation and Stenosis, and Mitral Regurgitation.—Systolic murmur at apex and a to-and-fro murmur at the aortic area (Fig. 127), the "to" murmur (systolic) running up to the 2d

sound, almost immediately followed by the "fro" murmur (diastolic).

(2) Mitral Stenosis and Regurgitation.—Presystolic murmur at the apex terminating in an abrupt 1st sound, and followed immediately by a systolic murmur.

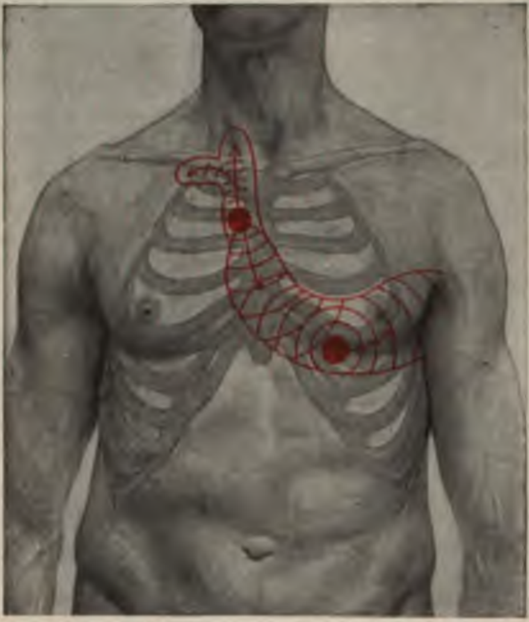


FIG. 127.—Combined murmurs of aortic incompetence and stenosis and mitral incompetency.

(3) Aortic Stenosis and Mitral Stenosis.—Presystolic murmur at apex, systolic at the base.

(4) Mitral Stenosis with Aortic Insufficiency. — Presystolic murmur at apex and diastolic at base.

Other Adventitious Endocardial Sounds. — Not infrequently certain sounds, in some cases "qualities" might be the better term, are found in connection with the closure of the valves, especially the mitral and aortic. They may be variously described as harsh, grating, rasping, clicking, or murmurish. The clicking sound may be mistaken for reduplication of the 1st or 2d sounds; another may, as implied by the last word of the preceding sentence, suggest a murmur without possessing sufficient duration to deserve the name. It is probable that in all cases the valve cusps are thickened and rough, and, when possessing a murmurish quality, slightly deformed as well, so that their apposition is, for a brief instant, hesitating, imperfect, and with some friction. A loud aortic sound or quality of the clicking or harsh variety is excellent evidence of atheromatous changes.

EXOCARDIAL SOUNDS

In addition to the adventitious sounds which originate in the interior of the heart, others which arise from conditions external to the heart may be heard over the pericardium. Exocardial sounds vary considerably in character, from the scratching or whiffing pericardial friction to those which are decidedly "murmurish" in quality, and may give rise to much perplexity as to their diagnostic significance. Although in not an inconsiderable proportion of individual cases the exact mechanism by which some of these exocardial sounds are produced can not be positively ascertained, yet it is almost always practicable to determine the fact of their exocardial origin. Contrasting them with intracardial murmurs, it will be found that they do not, in general, bear the same definite relation to the phases of the cardiac cycle; they are notably inconstant and variable, changing in intensity with the phases of respiration, the position of the body, or pressure upon the pericardium; and are for the most part superficial—i. e., convey an impression of nearness to the examining ear. Most important of all, the evidences of organic valvular disease, hypertrophy, dilatation, alterations in the pulse, and circulatory disturbances, are conspicuously absent. The varieties of extracardial sounds and their characteristics, arranged as nearly as possible in the order of frequency, are as follows:

Pericardial Friction.—Pericardial friction sounds are variously described as scratching, rasping, shuffling, rubbing, scraping, or puffing, more rarely as grating or creaking. They are due to the mutual friction of the apposed and inflamed surfaces of the pericardium and will disappear if these surfaces are separated by effusion. They are usually double (to and fro), corresponding to the systole and diastole of the heart, but if the surface of an auricle is inflamed, a tripling of the sound, due to auricular systole, may be heard. Although their rhythm corresponds roughly to the contraction and dilatation of the heart, yet it may be perceived that they do not bear any definite and clear relation to the heart sounds, and may occur during any portion of the cardiac cycle.

These sounds are heard, as a rule, earliest and with greatest intensity at the base, in the 3d and 4th, not infrequently in the 2d, left interspaces and corresponding half of the sternum. In an extensive pericarditis the point of greatest audibility is just internal to the left nipple. The location of a pericardial friction sound may change from day to day, but in all cases it is strictly localized, not transmitted. Such sounds are always superficial, may be intensified by the pressure of the stethoscope or finger, are intermittent, and, if of sufficient intensity and roughness, may be perceived as a thrill by the palpating hand. In a patient with thin chest walls and a much-dilated heart pulsating in the interspaces to the left of the sternum, firm pressure with the stethoscope may develop a rubbing sound somewhat resembling pericarditic friction.

Pleuro-pericardial Friction.—If there is inflammation of the pleural surface of the pericardium and the adjoining visceral pleura, friction sounds synchronous with the heart's action may be heard. They result from the rubbing together of the inflamed surfaces by the excursions of the heart. Such sounds are superficial and are most intense during full inspiration, as the lung becomes distended, overlapping the heart and apposing a larger area of roughened surfaces; conversely, they frequently cease during expiration. These friction sounds are most commonly heard over the 4th, 5th, and 6th interspaces where the lingula, or lappet of lung, overlies the apical portion of the heart.

Pleural Friction.—Ordinarily pleural friction is synchronous with the respiration, and ceases if the breath is held; but in some cases of dry pleurisy, affecting the interlobar sulci or that portion of the complementary pleura overlying the heart, crackling friction sounds synchronous with the action of the latter are heard, which may persist with much lessened intensity even during the cessation of breathing. Such friction sounds are not uncommon in phthisis pulmonalis.

Cardio-pulmonary Murmurs.—Occasionally a short, whiffing murmur or murmurish sound may be heard over and around the præcordial space, which, in the absence of other cardiac signs or symptoms, may be considered as due to the action of the moving heart upon the surrounding lung. While these sounds may be present with apparently normal lungs, it is probable that a certain amount of emphysema, usually localized or compensatory, is requisite for their production. This murmur may be produced when the heart in its excursion compresses a certain portion of the lung, thereby driving the air suddenly from the air cells into the bronchioles; or *per contra*, when the heart abruptly recedes from a portion of lung thus aspirating air into the alveoli. The cardio-pulmonary murmu is extremely variable, appearing or disappearing during inspiration or expiration, so also in the erect or recumbent posture, depending upon the exact relation of the portion of lung in which the murmu originates to the moving heart, the active agent in its production. I have more than once heard a whiff of this kind very distinctly by auscultating the open mouth of the patient.

Subphrenic Friction.—A friction sound heard over the lower end of the sternum and the adjoining costal cartilages, synchronous with the action of the heart, may be due to subphrenic peritonitis o abscess, or perhaps to a diaphragmatic pleuritis.

Crepitation.—Fine crepitating râles over the præcordium, accompanying the movements of the heart, may signify the rather uncom mon condition of mediastinal emphysema resulting from traumatism (tracheotomy in particular), pertussis, and diphtheria. Pneumothorax often coexists.

Splashing Sounds.—Churning, splashing, or "water-wheel" sounds are heard in the rare traumatic hydro-pneumopericardium. Similar sounds have been heard arising from a large, partly filled lung cavity close to the heart, and from a dilated stomach containing air and fluid. In all such cases the movements of the heart cause succussion and consequent splashing of the cavity contents.

V. PHYSICAL EXAMINATION OF THE BLOOD VESSELS (INCLUDING THE PULSE)

A. Examination of the Arteries

Because of its especial importance, the examination of the radial artery—i. e., the pulse—will be separately considered.

Inspection and Palpation of Arteries.—Certain points in the examination of the larger arteries have been already considered, mainly with reference to the aorta—viz., pulsation in episternal notch (page 284), epigastric pulsation and pulsating liver (page 348), and pulsations in the neighbourhood of the heart (page 345).

(1) Excessive pulsation of the medium-sized and smaller arteries is due to the same causes as an abnormally strong carotid pulsation (page 284).

Normally one may feel pulsation in the larger accessible arteries —viz., the carotid, subclavian, brachial, and radial—very frequently also in the temporal, femoral, popliteal, and posterior tibial. If the pulsation is abnormally strong and extensive it may be felt in the small arteries of the lips, fingers, and toes, and in the dorsalis pedis artery. In old people the pulsations of the medium-sized arteries are visible because of the thickness of the vessel walls and the consequent prominence of the vessels.

(2) Palpation of the accessible arteries furnishes valuable information with reference to the existence of general arteriosclerosis. If this condition is present these arteries are found to be hard, cordlike, and tortuous; and if calcareous degeneration has taken place, the finger tip passed along the vessel will perceive a more or less distinct sensation of irregular "beading," due to the presence of the calcareous plates.

Auscultation of Arteries.—The arteries which may be auscultated with profit are the aorta, pulmonary, carotid, subclavian, brachial, and femoral arteries, rarely the radial and posterial tibial. For sounds and murmurs in the thoracic aorta and pulmonary artery see (Index) the examination of the heart and its neighbourhood.

In auscultation of the accessible arteries one should listen first with the lightest pressure of the stethoscope compatible with excluding outside noises, then, with sufficient force to partly, but not entirely, occlude the vessel. In the first case sounds may or may not be heard; in the second case a systolic murmur is developed in any artery accessible to pressure. This murmur arises from the vibrations which are caused by the rhythmic passage of the blood stream through the narrowed portion of the vessel and varies in intensity with the degree of pressure.

(1) *Carotid and Subclavian Arteries.*—Normally, with light pressure one can hear over these vessels 2 sounds (not murmurs), systolic and diastolic in time, which are the transmitted 1st sound and aortic 2d sound of the heart. The 1st sound is sometimes absent.

A harsh systolic murmur heard over these vessels is a transmitted murmur from the aortic orifice, a roughened aorta, or an aortic aneurism. If softer and more blowing in character, especially if on the right side, it is the hæmic murmur of anæmia. Inability to hear the normal diastolic (aortic 2d) sound of the heart over these vessels may be significant of aortic regurgitation, as the conditions necessary for its production—viz., a competent aortic valve—are lacking. A diastolic murmur in the carotid and subclavian is due either to the conduction against the regurgitant blood stream of the bruit of aortic incompetency, or a murmur arising from a reflex current in the vessels created by the defective valve.

A short, whiffing systolic murmur in one or both subclavian arteries is heard in some healthy individuals when the chest is fully expanded and the breath held, or the arms extended vertically above the head. It is more commonly associated with apical pulmonary phthisis and is presumably due to bending of the vessel under the traction of adhesions or shrinking lung with consequent narrowing of its calibre—a condition equivalent to pressure upon the vessel by a stethoscope.

(2) *Sounds in Other Arteries.*—Under normal conditions in some persons, with light pressure, a *single* systolic sound (not murmur) may be heard in the femoral artery and the abdominal aorta, the sound of systolic tension of the vessel. In health this sound is entirely lacking in the smaller arteries. If *single sounds* are heard with light pressure, not only in the largest but also in the smaller arteries like the brachial, radial, ulnar, posterior tibial, and others even less in size, it is indicative of aortic incompetency. The abrupt filling of the vessels gives rise to a sound of tension. Such sounds

may also be caused by the bounding pulse waves of anæmia and acute fevers.

A *double sound*, systolic and diastolic, heard with light pressure in the femoral artery, is a rare sign in aortic incompetency, lead poisoning, pregnancy, and mitral stenosis, the second sound arising from the sudden collapse of the vessel. A *double murmur heard with pressure* over the femoral is found only in aortic incompetence, the first (systolic) murmur resulting from the onward rush of blood through the narrowed vessel; the second (diastolic) portion arising from a backward flow of the blood stream, the incompetent aortic valve failing to sustain the peripheral direction of the current.

Murmurs may also be heard in arteries compressed by enlarged lymph nodes or other tumours, and over a goitrous thyroid gland.

B. CAPILLARIES

Aside from the degree of fulness of the capillaries as evidenced by redness or pallor of the skin, the only sign of importance which can be found by examination of these minute vessels is the "capillary pulse" previously considered (Subungual Pulse, page 288).

C. VEINS

Inspection and Palpation of Veins.—General and local venous distention (page 99) has been considered; so also has jugular pulsation (page 286). In addition the following signs may be observed:

(1) A *systolic* (true) venous pulse like that seen in the jugular, and occurring under the same conditions, has been noted in the veins of the face, the superficial veins of the arm, and the branches of the internal mammary veins.

(2) A *centripetal* (progressive) venous pulse, the wave passing from instead of toward the periphery, may exist under certain conditions in the dorsal veins of the hand and foot. It is always associated with a capillary pulse and, like the latter, is due to aortic insufficiency or great relaxation of the arterioles (as in anæmia, phthisis, neurasthenia, etc.), so that the arterial pulse is transmitted through the capillaries into the veins. In rare instances a direct communication between the artery and vein (aneurismal varix) is responsible for this phenomenon.

(3) If a vein is perceived by palpation to be *firm and cordlike* and œdema of the extremity coexists, thrombosis of the vein is present. It occurs most frequently in one or both femoral veins, extending perhaps into the iliac trunks, as the result of infectious diseases and septic processes, more rarely in the weakness of age.

Auscultation of Veins.—(1) *Jugular and Innominate Veins.*—The only auscultatory evidence of any importance to be derived from the veins is the so-called *bruit de diable*, or venous hum. It is a continuous humming and sometimes musical murmur, heard over the jugular veins of both sides, but with greater loudness over the right jugular.

It may be caused by the pressure of the stethoscope or the constriction of the vein which occurs when the head is turned far to one side. Consequently, to determine its presence as a real and not an artificial sign the stethoscope must be applied evenly and lightly, with a degree of pressure just sufficient to maintain the apposition of the mouthpiece to the skin, and the head kept in a symmetrical and unconstrained position. It occasionally happens that the tracheal inspiratory sound resembles the venous hum so closely that an error may arise, but the question is readily solved by having the patient hold his breath. The maximum intensity of the bruit is usually in a somewhat triangular space having the inner third of the clavicle as a base, especially over the interval between the clavicular and sternal attachments of the sterno-cleido-mastoid muscle.

The mechanism and meaning of the murmur are still in dispute. It arises either from the formation of a fluid vein at the point where the narrow jugular opens into a wider cavity—the jugular bulb—or from lateral vibrations of the vessel walls. In both cases an abnormal fluidity of the blood is usually but not always a prerequisite. The intensity of the murmur is greatest in the upright position, also during inspiration and the diastole of the heart, all of these factors increasing the rapidity with which the blood flows from the jugulars through the innominate veins into the superior cava. Thus the murmur, while continuous, rises and falls with rhythmic intensity. If the murmur is unusually loud, it may be heard not only above the clavicle, but may be followed as far down as the 2d right interspace and rib.

While the venous hum occurs in some apparently healthy individuals, its association with anæmia, particularly the chlorotic and pernicious varieties, is so frequent that its presence must be held as a suggestive, but by no means pathognomonic, sign of this disease.

(2) *Sounds in Other Veins.*—In extreme grades of anæmia similar humming murmurs may be heard in the subclavian veins and the axillary and other large veins of the extremities. In advanced cirrhosis of the liver a venous hum may be heard over the lower costal margin in the right hypochondrium. In the jugular veins exhibiting the systolic pulse of tricuspid incompetency, a sound (not a murmur) may usually be heard, due to the sudden tension of the vessel.

A similar sound of the same origin may sometimes be heard in the femoral vein.

D. THE PULSE

The value of the pulse examination depends primarily upon a knowledge of the physiology of the heart and blood vessels, but also very largely upon the personal experience of the examiner.

Elements of the Pulse.—The elements of the pulse which are of clinical value, and therefore require investigation, are:

(1) The pulse rate (frequency), (2) the rhythm, (3) the condition of the vessel walls and incidentally the size of the vessel itself, (4) the tension (blood pressure), (5) the character of the pulse wave with reference to amplitude, duration, and celerity. Finally, (6) the pulse in other arteries of the same individual.

Technic of Examination.—Because of its accessibility, the radial pulse is chosen for examination. The patient should be either lying or sitting in an easy position and, unless for a particular object, should not have made any physical exertion just previous to the examination. The mere act of taking the pulse will greatly accelerate its frequency in many persons, even in those who are outwardly calm and composed.

The patient's forearm should be semi-pronated, as the artery is thus more readily palpated, and the arm supported. It is always desirable to examine both radial arteries in every patient who is seen for the first time, in order to detect a not uncommon anomaly of distribution in which the radial winds around the styloid process of the radius to the dorsum of the bone, while the superficialis volæ pursues the usual course of the radial. Three fingers—first, middle, and ring—should be laid upon the artery. For certain purposes the simultaneous employment of the fingers of each hand is desirable.

(1) *To Determine the Pulse Rate.*—Count the pulse for 15 seconds and multiply by 4. If it is irregular, count for a full minute. An extraordinarily frequent pulse, 200 or over, if regular, may be determined by counting every 2d or every 3d beat and multiplying by 2 or 3 as required; or by making a line of dots with pencil on a sheet of paper, each dot corresponding to a pulse beat, and afterward enumerating the marks.

(2) *Note the Rhythm.*—Are the successive beats equidistant in point of time—i. e., are they regular?

(3) *Note the Condition of the Vessel Walls and the Size of the Vessel.*—Empty the vessel by pressure and roll it under the fingers, slipping the skin over the vessel. A normal artery is scarcely to be felt except in a very thin wrist, but one that is sclerosed is firm, cordlike,

and tortuous. While empty, endeavour to estimate its size. Run the finger along the vessel in order to detect calcareous "beading"—plates of lime salts. As a high-tension artery feels very much like one which is sclerosed, cut off the direct blood flow by firm pressure with the finger nearest the heart, and the recurrent flow (from palmar arch) by firm pressure with the finger nearest the hand. The middle finger can then determine the condition of the arterial wall, the element of blood pressure having been eliminated.

(4) *Estimate the Tension.*—To estimate the arterial tension (page 330), first ascertain the condition of the arterial walls, as in (3), to eliminate a deceptive arteriosclerosis; then cut off the recurrent wave from the palmar arch with the finger nearest the hand, and make increasing pressure with the finger nearest the heart until the direct wave is no longer perceived by the middle of the 3 fingers employed. The degree of pressure required to extinguish the direct wave is the measure of the tension, and the ability to gauge it correctly requires considerable practice.

It is to be borne in mind that while the pulse wave itself may have a high tension, as in aortic incompetency, the blood pressure in the intervals between the beats may be low, as it is in the same lesion. It is only a continuous or prolonged high-tension pulse, the blood pressure remaining high between beats, that is significant. Its presence may be assured by endeavouring to palpate the artery between beats, rolling it from side to side, and finding it firm and not easily compressible.

Further assistance may be derived from the fact that a low-tension pulse is most distinctly felt with light pressure; a pulse of moderate tension with moderate pressure, while a high-tension pulse develops its greatest force with firm pressure. Moreover, if variations in the size of the vessel be excepted, a high-tension pulse is usually, but not always, small, and conversely.

If with light pressure upon a soft, easily compressible artery a 2d weak rebound is felt, immediately sequent to the first expansion, it is the dicrotic wave or pulse, a sign of low tension.

THE SPHYGMOMANOMETER

The Sphygmomanometer.—The sphygmomanometer is an instrument of great value which has lately been added to the armamentarium of the internist (FINCKE). An exact determination of the blood pressure, which has always been an affair of much practical difficulty, is, by means of this apparatus, reduced to a matter of relative accuracy.

Thus it is now possible not only to make a correct measurement of the actual pressure, but also, from time to time, to note changes so slight as to be entirely inappreciable by ordinary palpation.

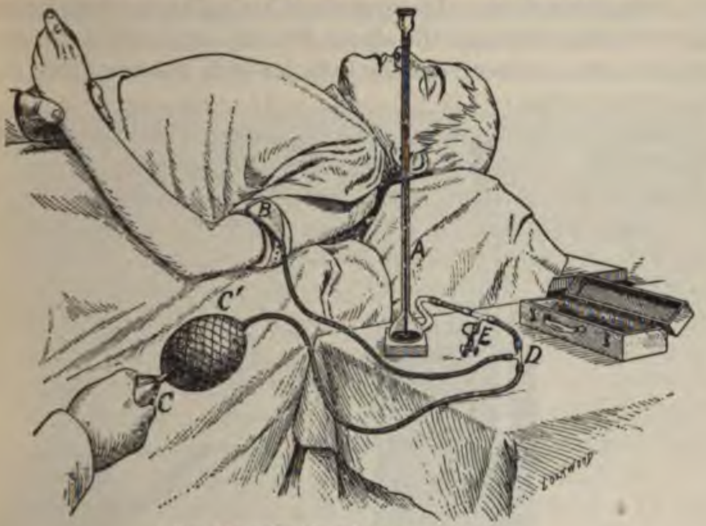


Fig. 128.—Cook's sphygmomanometer.

Technic of the Sphygmomanometer.—There are three forms of this apparatus which are practicable for clinical use: the Riva-Rocci (Cook's modification), Stanton's, and Janeway's. All these instruments employ the same principle, the circular compression of the arm by a closed system of air contained within the manometer A (Figs. 128,

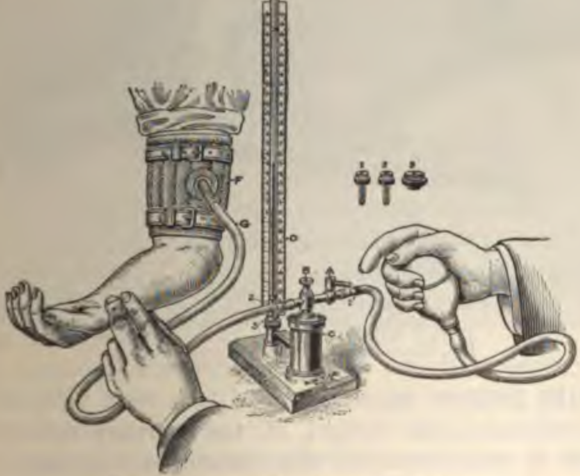


Fig. 129.—Stanton's sphygmomanometer.

129 and 130), the arm-piece B, and the necessary connecting tubing, which, with the inflating apparatus C, constitute the essential features of the instrument. The arm-piece having been applied midway between the shoulder and the elbow, the pressure within the system is raised to the required point by inflation with the hand-bulb.

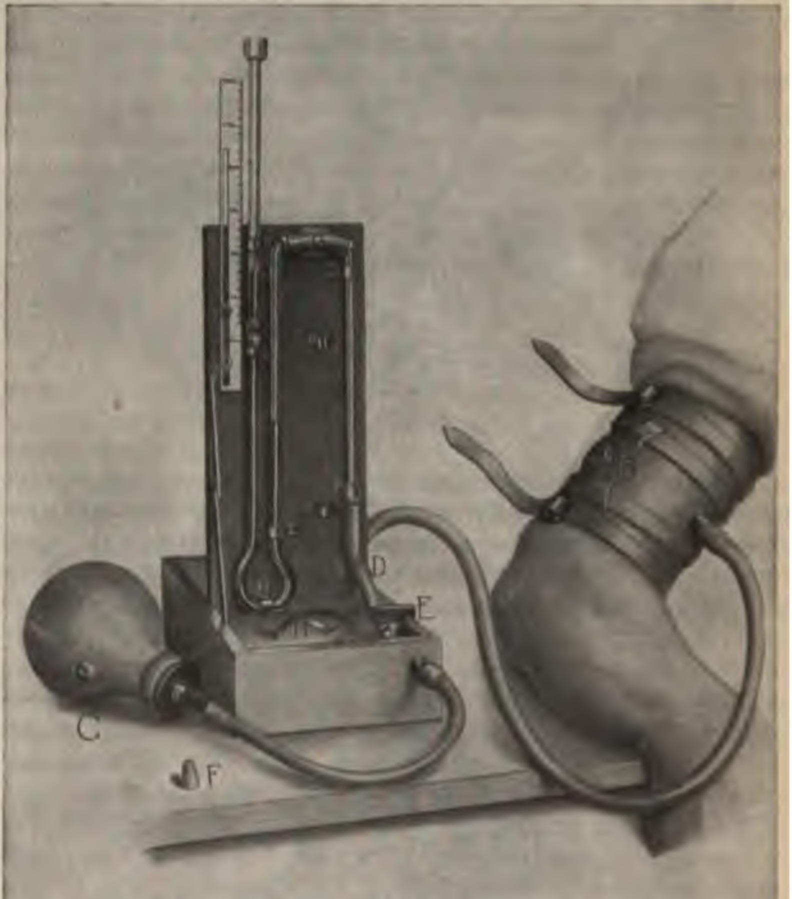


Fig. 130.—Janeway's sphygmomanometer.

When the pressure reaches such a point that the pulse at the wrist is obliterated, the height of the mercury column in the manometer is equivalent to the maximum (*systolic*) pressure, depending upon the principle that equal pressure is transmitted to every point throughout the system and exerts an equal external force. It has been found experimentally that the return of the pulse after obliteration is a more exact and constant point than its disappearance, so that this is generally taken as the criterion.

The diastolic (*minimum*) pressure is obtained, with the instruments which permit such a determination (Stanton's, Janeway's), in the following manner: After obtaining the systolic reading, allow the pressure to fall 5 mm. at a time, watching the pulsation of the mercury column produced by the pulse wave. The amplitude of this pulsation will increase for a time, then continue the same for a few millimetres. The last point at which the *amplitude is greatest* marks the diastolic pressure. Unless a pulsation of 5 mm. is obtained, no accurate measurement is possible. Deficient pulsation happens in a rapid or a very small pulse, and with too loose adjustment of the cuff. *Mean pressure* is obtained with a sufficient degree of accuracy, by taking the average of systolic and diastolic pressures.

Certain precautions are to be taken with all these instruments. Successive observations in the same case should be taken in the same position, either sitting or lying. The arm-piece should always be at the heart level. The adjustment of the cuff should be snug, without exerting undue pressure.

In taking the diastolic reading, time must be allowed after each fall in pressure for the mercury to adjust itself, and from ten to twenty oscillations should be observed at each level. For children and for thin adults, the readings of these three instruments will be the same.

In the average adult the Riva-Rocci, because of its narrow arm-piece (5 cm.), and to a much less extent Stanton's machine (arm-piece 8 cm.), give pressures too high, while in very stout and very muscular arms the indicated reading is far above the actual intravascular pressure. Janeway's instrument, with its 12 cm. arm-piece, is free from this error, and its readings may be accepted as accurate. The presence of sclerotic or calcareous changes in the artery under examination produces an increase of only 5 to 10 mm. and may be neglected. In children the cuff may be applied to the thigh.

(5) *Determine the Character of the Pulse Wave with Reference to Amplitude, Strength, Duration, and Celerity.*—The *amplitude* or volume of the individual pulse wave is measured by the amount of expansion of the artery—i. e., its increase in diameter—the arterial diastole, coincident with the systole of the heart.

The volume of the pulse wave will vary from large, to medium, to small. The pulse may be found to be irregular in volume and strength as well as rhythm.

Here may be mentioned the volume or fulness of the artery *be-*

tween beats. Palpation between the successive pulse waves will show that the vessel remains full, or that it empties out more or less completely. If full, the tension may be high, as in chronic renal disease, or low, as in the febrile pulse with relaxed arterioles. A full artery does not necessarily imply a large pulse wave. In aortic incompetence the pulse wave is extraordinarily large, while the artery is quite empty in the intervals.

It is hardly possible to separate the *strength* of the pulse wave from its tension, and the two are practically synonymous.

The *duration* and *celerity* of the pulse wave refer to the manner of its ascent, summit, and descent—i. e., of the rise of blood pressure, of its maintenance, and finally its fall. It is to be observed, first, whether the ascent or increase of pressure is sudden, moderately rapid, or slow; second, whether the pressure is well sustained, or whether, having reached its height, it falls off abruptly; third, whether the descent or fall of pressure is rapid, gradual, or slow. As a rule, in a high-tension pulse the rise is gradual, the height well sustained, and the descent gradual, while in a low-tension pulse it is sudden, brief, and quick.

During the descent there may be perceived a second slight impulse or secondary wave. If this is felt only—or best—with considerable pressure in a high-tension pulse, it is the tidal or predicrotic wave. On the other hand, if the pulse is one of low tension and the secondary impulse is felt only or best with very light pressure, it is the dicrotic wave (dicrotic pulse), and may be intensified by cutting off the recurrent wave from the palmar arch. The dicrotic wave is much more frequently observed than the predicrotic.

(6) *Compare the Pulse in Other Arteries in the Same Individual.*—Compare both radials, and, if practicable, both femorals, and both posterior tibial arteries as well. It may be found that the pulse wave, which should reach symmetrical arteries at the same instant, is delayed in one, or obliterated, or will present differences in amplitude, strength, and character on the two sides.

The Normal Pulse.—In an adult at rest the *frequency of the pulse* varies from 70 to 75; it is *regular;* the *artery* can be felt scarcely or not at all; the *tension* is *moderate;* the pulse wave is of *medium amplitude*, its rise and fall are neither extremely abrupt nor abnormally slow, and the pressure is sustained without undue brevity or prolongation.

The Significance of Variations in the Pulse.—The following variations are recognised:

Increased Frequency (*Tachycardia, Pulsus frequens*).—Bearing in mind the nervous mechanism of the heart (page 330), it is evident that increased frequency of the pulse may be due either to paralysis of the pneumogastric or to irritation of the sympathetic nerves or the intracardiac ganglia. Conversely, a decreased pulse rate may be due to pneumogastric irritation or paralysis of the cardiac sympathetic nerves and ganglia.

The source and causes of irritation or depression of the cardiac mechanism are diverse. It may be an organic lesion affecting the centres in the medulla or the nerves connecting it with the heart—e. g., tumour, hemorrhage, meningitis, gumma, exostosis, or neuritis—or inflammation of the endocardium or pericardium, or degeneration of the cardiac muscle containing the ganglia. It may be functional—e. g. reflex—from the cerebrum (emotion), or from connected, somewhat distant organs (stomach, intestines), or from poisonous substances circulating in the blood (alcohol, bile), or from general fatigue of the nervous system.

(1) The pulse rate is normally more frequent in infants (140 at birth) and children (90–100 at 3 years of age) than in the adult, slightly more frequent in women (75) than in men (70), is more rapid in the evening than the morning, in the erect than the recumbent position, after than before meals and the taking of hot beverages, in hot than in cold weather, during exercise than at rest, and may be extremely rapid in consequence of mental excitement. Rapid heart action is usually associated with a low-tension pulse. Indeed, low tension is the most important factor in almost all cases of frequent pulse. The lessening of the peripheral resistance removes a check upon the heart's action, and therefore permits it to contract with more ease and consequent increased rapidity.

(2) In all febrile conditions the pulse rate is usually increased, 8 to 10 beats for each degree above the normal. This ratio does not always hold, as in typhoid fever, where the pulse is less frequent, and in scarlet fever, in which it is more frequent, than would be expected, constituting in each case a diagnostic finding of some value. If the fever is of septic or suppurative origin, the frequency is excessive as compared with the temperature.

(3) A frequent pulse attends all well-marked valvular defects of the heart (except aortic stenosis, in which the pulse may be slow), and is found in nearly all cases when compensation fails.

(4) When a persistently frequent pulse is encountered in persons who are not at all or but slightly febrile, and who present no physical signs of gross cardiac disease, it should create suspicion of the existence of certain diseases which are characterized more or less constantly by this symptom, and other corroborative evidence should be sought. These ailments are early phthisis, exophthalmic goitre,

Addison's disease, chlorosis and pernicious anæmia, arthritis deformans, and locomotor ataxia.

(5) It is also to be remembered that neurasthenic conditions may exhibit a rapid pulse rate. The abuse of alcohol, tobacco, coffee, and tea may account for otherwise inexplicable cases. Sexual excesses, lack of sleep, and disorders of digestion are not infrequent causes, and there is frequently a tachycardia during convalescence from acute diseases, and in hemorrhage and conditions of general debility.

(6) There is a peculiar form of accelerated action of the heart which, in the absence of demonstrable organic alterations, must be considered a neurosis. It consists in recurring attacks of rapid beating of the heart (paroxysmal tachycardia), lasting for an hour or more and recurring at varying intervals, in some cases for years. The paroxysm may or may not be attended by nausea, anxiety, and substernal oppression. The pulse rate may rise to 240 or over.

Decreased Frequency (*Brachycardia or Bradycardia, Pulsus rarus*).—The normally slow pulse rate runs from 60 down to 40, while in disease the heart may beat in very rare instances but 4 times per minute. An infrequent pulse is usually of high tension, an increase of the peripheral resistance forcing the heart to contract more slowly.

(1) The pulse is slow in some healthy, usually strong and large-bodied individuals. It is often slow in old age, and is normally infrequent in the puerperium.

(2) Bradycardia may be indicative of some cardiac lesion, most frequently fatty degeneration, chronic myocarditis, or sclerosis of the coronary arteries. The pulse of aortic stenosis is usually slow, while mitral lesions may also cause an infrequent pulse, probably because of associated changes in the heart muscles. If bradycardia continues, notwithstanding the use of large doses of atropine, it is presumably due to an affection of the heart (DEHIO). Because of the impediment in the pulmonary circuit, the pulse is slow during an attack of spasmodic asthma and in emphysema.

(3) Certain diseases of the nervous system may be responsible for a reduction in the pulse rate, notably cerebral hemorrhage, tumour, meningitis, or other lesions which give rise to an increased intracranial pressure. It is also seen in epilepsy, diseases or injuries of the cervical cord, mania, melancholia, and general paralysis of the insane. Myxœdema with its general apathy exhibits a lessened pulse rate.

(4) Poisonous substances circulating in the blood may, by acting upon the cardiac centres or ganglia, retard the pulse beat to a marked degree. Among these substances the most important are bile (in jaundice), urea and other retained excrementitious materials (in

uremia), glucose, lead, opium, carbon dioxide, alcohol, and occasionally tea, coffee, and tobacco.

(5) A slow pulse is frequently present in cases of inanition—e.g., gastric ulcer and cancer; still more frequently in convalescence from certain acute, usually specific infectious, diseases, as diphtheria, erysipelas, pneumonia, typhoid fever, malaria, and acute articular rheumatism—due probably (HEUBNER) to a temporary toxic depression of the conductivity of the heart-muscle.

(6) Chronic digestive disorders often retard the pulse because of mental depression, poisonous substances in the blood, or reflexes from the digestive organs, one or all.

(7) *Heart-block.*—A disturbance due to depressed conductivity in the muscle-bundle of His. It is by way of this band, and not through any nervous connections, that the wave of contraction, beginning in the right auricle, passes to the ventricles. Interference with the function of this band will, therefore, according to its degree, partly or completely "block" the passage of the auricular stimulus to the ventricles. While the auricles, as shown by the jugular pulsation, will continue to contract at the usual rate, the ventricles respond only to such stimuli as are transmitted. If no stimuli pass, the ventricle contracts at a rate which bears no relation to that of the auricles—complete heart-block. If every 2d, 3d, or 4th contraction of the auricle is transmitted, and causes a ventricular contraction, it is an incomplete heart-block.

Depression of conductivity, usually partial and temporary, may be of toxic origin, occurring during convalescence from diphtheria, typhoid, and rheumatic fevers, and from the use of digitalis. Degenerations (fibroid or fatty) or syphilitic lesions of His's bundle have been demonstrated as organic causes of a condition which presents a typical, and usually fatal, heart-block, the *Stokes-Adams syndrome.* This is characterized by a slow arterial pulse (30 or less), syncopal, apoplectiform, or epileptiform convulsions, and pulsation of the neck-veins (auricular) occurring at more frequent intervals than the radial pulse-beat (2 : 1 to 4 : 1).

Irregular Pulse (Arrhythmia).—Much has been accomplished (MACKENZIE, WENKEBACH, HIRSCHFELDER, HEWLETT) in the classification of irregularities of the pulse-rate. This knowledge has been derived from the study of graphic records of the venous pulse (usually the right internal jugular) in comparison with similar records made from an artery (radial, brachial, carotid), or the apex-beat.

It is inexpedient here to attempt a minute study of the complicated elements involved in the interpretation of such tracings, particularly of pathological pulses. It must suffice to present a normal tracing.

For clinical purposes one may recognise three types of arrhythmia, which together comprise 95 per cent of all the irregularities of the heart-action (Hewlett), as follows:

(1) *Respiratory Arrhythmia.*—The pulse-rate is more rapid during inspiration than during expiration. The degree of this irregularity varies from barely perceptible to very marked alterations in rate during the two phases of respiration. Such alterations are pathological when recognised during quiet, ordinary breathing. In well-defined cases the rate increases rapidly during inspiration, and slows abruptly at the beginning of expiration.

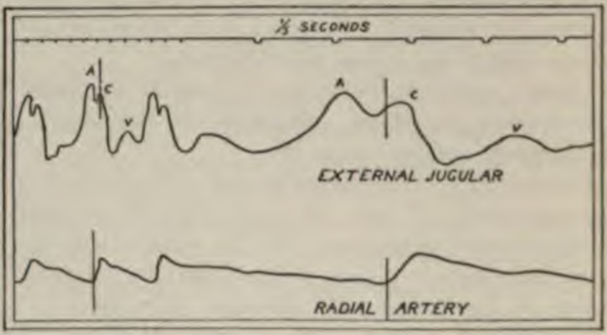


FIG. 130*a*.—Normal venous pulse tracing. (Hewlett.) A, Contraction of right auricle; C, either transmitted carotid pulsation, or systolic pressure against tricuspid; V, filling of auricle while tricuspid is closed.

To determine clinically the presence of this deviation, the movements of the chest must be watched and compared with the pulse-rate. To confirm the finding, request the patient to take slow, deep respirations, which will exaggerate the arrhythmia; and to hold the breath, after which the rhythm will become regular.

This form of arrhythmia is of nervous origin, due, apparently, to an unusual activity or irritability of the pneumogastric (vagus) centre. When present it is usually associated with a slow pulse (bradycardia).

Respiratory arrhythmia is normal in young persons up to the age of puberty, and, when due to disease, is rarely seen after twenty-two years of age. The vagus centre is distinctly more excitable earlier than later in life. It occurs in the slow pulse which may follow unusual exertion. It is noted frequently during convalescence in the febrile infections, especially diphtheria, scarlet fever, and typhoid fever, or during the course of diseases in which bradycardia is a symptom—e. g., typhoid fever and meningitis. It may be found in hysterical and neurasthenic persons, the vagus centre participating in the general state of nervous irritability and instability, or in cerebral lesions which irritate the vagus.

This form of arrhythmia rarely possesses any serious significance. When marked, and occurring after infectious fevers, alarm is apt to be felt, but usually, after continuing for a few days or weeks, it disappears without unpleasant consequences.

The *pulsus paradoxus,* a pulse which is weak or imperceptible during inspiration, is not to be confused with respiratory arrhythmia. It is of mechanical, not of neurogenic, origin. If marked it may indicate large pericardial effusions, indurative mediastino-pericarditis, mediastinal tumours, or some obstruction to the entrance of air into the lungs.

(2) *Extrasystoles.*—An "intermittent" pulse is usually due to extrasystoles. To each stimulus of strength sufficient to cause a contraction the heart responds to the extent of its maximum contractility at the time. A brief refractory stage follows, during which the heart-muscle is not responsive to an ordinary stimulus. But if the stimulus is extra and abnormal, or if the heart-muscle is unnaturally excitable, an extra or premature systole will occur. Thus the heart is in a (second) refractory phase at the instant of arrival of the normal stimulus and does not contract. A long or compensatory pause follows, succeeded by a regular contraction, often felt as a "thump." An extrasystole is usually weak, and, if occurring early in diastole, may lack sufficient strength to open the aortic valve (dropped pulse-beat), or sends but a feeble wave into the arteries. Extrasystoles occurring consecutively give rise to the *pulsus bigeminus* (*D,* Fig. 131), *trigeminus,* or other numerical varieties.

Clinically, during auscultation, an extrasystole may be recognised as a contraction, occurring prematurely, immediately after a normal systole. The effect is that of two beats close together followed by an intermission. The first sound (ventricular contraction) can almost always be heard; the second sound (aortic closure) may or may not be audible, according to the strength of the extrasystole. Extrasystoles appear especially when the pulse is slow, and vary in number from hour to hour or days to months. When found, especially if persistent, the following conditions should be considered as possible causes:

In some persons this form of arrhythmia persists for years without ill consequences (congenital, or physiological, cardiac hyperirritability). It occurs most frequently in people at or past middle age, with arteriosclerosis and myocarditis. It is probable that the heart-muscle becomes more irritable with advancing age. High arterial tension is a rather frequent cause, and both in young and old abnormally low tension as well. Extrasystoles are not common during the acute myocarditis of specific infections, but if present are of a more serious import than usual. They often occur in organic cardiac disease, but a severe lesion may run its course without them. The heart of extreme obesity may exhibit extrasystoles.

Extracardial causes are not lacking. Extrasystoles occur rather frequently during convalescence from infectious diseases, especially typhoid fever, pneumonia, and diphtheria; also as a result of intoxications due to uræmia, hyperthyroidism, intestinal disorders, digitalis, tea, coffee, and tobacco. Nervous influences (hysteria, neurasthenia), either directly or indirectly, may produce them, perhaps by raising the blood-pressure. The *significance* of the extrasystole is indefinite, as it is not diagnostic of a particular disease. It indicates in general an excessive cardiac irritability. Its most important association is with myocardial disease and arteriosclerosis, and its frequent occurrence in those past middle age is suspicious.

(3) *Absolute Arrhythmia.*—This is recognised clinically by the fact that no two pulse-beats are of the same length—the heart action is totally and absolutely irregular. The irregularity may come on suddenly or slowly, and pass similarly after lasting days, weeks, or months, but it tends to return, and oftentimes becomes a permanent condition. The pulse-rate may be slow or very rapid; if the latter, this form of irregularity is termed *delirium cordis.* Some of the ventricular systoles do not produce a pulse-wave, and in many systoles there is no sound of aortic closure. The right auricle is usually paralyzed (HEWLETT), and tricuspid insufficiency often coexists.

The *significance* of the absolutely irregular pulse is always grave. It is most commonly found in the late stages of mitral lesions; next most frequently in arteriosclerosis and myocardial disease. It is practically never seen except when compensation is broken.

(4) *Pulsus Alternans.*—This (*C*, Fig. 131) is an extremely rare finding. It is recognised clinically by the alternate occurrence of a small and a large pulse-wave at *regular* intervals. There is no compensatory pause. It is due to depressed contractility, and is seen in severe myocardial and valvular diseases.

(5) *Heart-block.*—See (7), page 395.

Thickened Arteries.—If the radial and other accessible arteries are found to be thickened, rigid, tortuous, or calcified, it indicates a general arteriosclerosis (*q. v.*), which may account for an observed cardiac hypertrophy, and renders possible the occurrence of intracranial hemorrhage or aneurism. This form of vascular disease has widespread relations to disease of other organs.

Variations in Tension.—The following table gives the normal limits of systolic pressure. These and subsequent values are for the Janeway apparatus (p. 390).

Infants	75– 90 mm.
Children over 2 years	90–110 mm.
Young adults	100–130 mm.
Older adults	110–145 mm.

Over 200 mm. is very high tension.

Under 70 mm. is very low tension.

Diastolic pressure in normal pulses is 25–40 mm. below systolic. In high tension it may be 50–80 mm. below, and in aortic insufficiency it may be as much as 100 mm. below.

Normal Blood Pressure.—The pressure maintained within the blood vessels is dependent upon four factors: The energy of the heart, the peripheral resistance, the elasticity of the vessel walls, and the volume of the circulating blood. Under normal circumstances each factor is more or less variable and such variations result in normal fluctuations in the intravascular pressure. These alterations, in a state of health, remain within certain limits, due largely to the power possessed by the various factors, of compensating for a change in one or more of the others. The greatest single element in the control of general blood pressure is found in the abdominal vessels of the splanchnic area, depending on the facts, first, that this area is large enough to contain almost the whole blood volume of the body; and second, that the splanchnics are very easily affected by reflexes from any sensory nerve.

As will be seen from the table given above, normal blood pressure varies considerably at different ages. The cause of the higher readings with increasing age may be assumed to be increased peripheral resistance, due to reasons not, as yet, entirely determined. Women, as a rule, give an average reading a little lower (5 to 10 mm.) than men. In those accustomed to continuous hard manual labor the pressure is often a little higher than the average figures given. Temperament, and perhaps heredity, have considerable influence. In excitable and neurotic individuals, because of their greater susceptibility to psychical influences, a rise is produced by slight emotional causes, so that a series of observations in these cases would probably afford a result somewhat above the general average.

In the individual there are certain variations under changing physiological conditions. Position has some influence: pressures taken lying down being from 5 to 10 mm. less than in the sitting or standing positions. The influence of meals is inconstant and somewhat in dispute. Alcohol causes no increase; tobacco, a brief rise. Excitement of any kind produces a marked rise in pressure which may amount to as much as 40 mm. During physical exertion the increase may be considerable, depending partly upon the work done, but perhaps to a greater extent upon the amount of voluntary effort required for its accomplishment. For example, it has been found that the attempt to walk in a straight line has more effect than traversing the same distance without special attention.

Blood Pressure in Pathological Conditions.—In pathological conditions a change in blood pressure in either direction may be caused by an alteration of one or more of the four factors controlling it, if not compensated by the others.

A hemorrhage, therefore, produces a rapid fall unless its influence is minimized or entirely compensated for by increased peripheral resistance from contraction of the arterioles, and to a less extent by increased rapidity of the heart beat. Such a result may occur in hæmoptysis or other visible hemorrhage, when the attending excitement may even cause a rise. On the other hand, when the psychical element is absent, as in intestinal or other concealed hemorrhage, or in the anæsthetized patient, the fall is marked, although unless collapse or shock supervenes, there is an early return to the normal.

Shock and collapse give with the sphygmomanometer excessively low readings. In these conditions there is a general vaso-motor paralysis; the splanchnic vessels especially are greatly dilated and engorged with blood, with resultant fall in general pressure. The heart for a time is able to counteract this condition, but unless the vaso-motor tone is restored, death eventually ensues.

In chronic diffuse arterial disease (sclerosis and calcification), blood pressure is generally markedly high. The increased pressure depends upon the loss of elasticity of the vessel walls; upon the increased tension of the arterioles, which is usually an accompaniment of this condition; and, finally, upon the secondary hypertrophy of the heart which follows. Local peripheral sclerosis is practically without influence unless the splanchnic area is involved. Without such involvement the pressure is not raised. High pressure without apparent sclerosis is not infrequently met with, due to an early sclerosis of the splanchnic veins (phlebosclerosis, angiosclerosis), or possibly, in some cases, to an hypertrophy of the muscular coats of the arteries with consequent increased tension (hypermyotrophy).

Diseases of the Heart.—In primary myocarditis the sphygmomanometer shows a low pressure: in cases secondary to or associated with arteriosclerosis or kidney disease, the readings are high, the instrument thus easily differentiating between these conditions. Compensation and non-compensation can not be distinguished by the sphygmomanometer, high pressure often existing and even increasing when compensation fails. Acute endocarditis has no definite relation to blood pressure. Chronic valvular disease, except aortic regurgitation, fails to show any constant pressure changes. In aortic insufficiency, and in insufficiency combined with stenosis or with certain mitral lesions, the sphygmomanometer affords efficient aid. It is necessary, however, to employ an instrument measuring both systolic and diastolic pressure. In compensated cases the systolic readings are high, the diastolic low, this wide difference constituting the instrumental interpretation of the water-hammer pulse. In double aortic lesions, the sphygmomanometer determines with accuracy the important problem as to which predominates. Stenosis of a high degree prevents the influx of the full contents of the hypertrophied ventricle into the aorta and thus diminishes the size of the systolic impulse, while a less marked lesion permits the full height of the systolic wave to show itself. In aortic regurgitation with mitral leakage, the extent of the latter may be judged in the same way by the size of the systolic wave in its relation to the diastolic pressure.

Angina Pectoris.—This condition is frequently associated with high blood pressure. If high pressure readings are found together with a history of substernal and pectoral pain, the presence of a true angina may be assumed. The absence of high pressure does not negative the disease, which may exist with a comparatively low pressure.

Nephritis.—Acute nephritis is generally considered to produce increased blood pressure. Especially is this true of the cases following scarlet fever. Nevertheless many cases fail to show this rise, so that it can not be considered of much diagnostic value. Acute exacerbations of chronic kidney disease cause an increase in the previous pressure. In the interstitial forms of chronic nephritis the highest readings (160–200) are found, especially when, as so often is the case, arteriosclerosis coexists. But high readings are not invariably present, as in the terminal stages heart weakness may bring the pressure to normal or below. Chronic parenchymatous nephritis may or may not cause high pressure. If the reading is high it is an additional symptom; its absence does not negative the disease. In uræmia, associated with any form of lesion, the pressure is invariably high (180–300), with the exception of a terminal fall in the fatal cases. The height of the mercurial column varies quite accurately with the uræmic manifestations, and serves as an index of the severity and the progress of the condition.

Acute Diseases.—In most febrile conditions, associated as they are with infectious toxæmias, the blood pressure shows a tendency to fall. Especially is this the case in typhoid fever where a very low level is a constant feature. The three most important and serious phenomena of this disease manifest marked changes in pressure which are of great importance in their diagnosis. Collapse produces a progressive fall; a marked and rapid fall is seen in hemorrhage. Perforation with beginning peritonitis causes a sharp rise, one of the earliest, if not the very first, symptoms of this complication.

Pneumonia is somewhat irregular in its relation to blood pressure. As a rule, for the first day or two, there is a normal or slightly increased pressure, gradually declining to a comparatively low level as the crisis approaches, and falling still lower with its occurrence. A rapid fall is an ominous sign, and points to impending collapse.

Diseases of the Nervous System.—Disease of the cerebral vessels may or may not be associated with general arteriosclerosis, so that the determination of general blood pressure can not decide the arterial condition in the brain. With a general high tension, however, the liability to cerebral hemorrhage is greater, therefore the determination of the tension may be of value as a forewarning of the likelihood of such an event. Hemorrhage having taken place, the sphygmomanometer now shows the highest readings obtained, and so may be of the greatest help in diagnosis. No other cause of coma, except uræmia, gives rise to such a high pressure, and even uræmia rarely produces figures so enormous as are common in cerebral hemorrhage. By continued observation the course of the hemorrhage may be watched; increasing symptoms, with a further rise in pressure, signify continued bleeding; a stationary or falling pressure, without increase in symptoms, denotes a cessation of the hemorrhage.

Melancholia generally shows a rise in pressure proportional to the intensity of the mental suffering. Facial neuralgia, in common with the majority of severe pains, will cause a rise. Hysteria frequently presents a high pressure, and its finding may in some cases be of diagnostic value. Certain forms of insomnia are apparently due to increased intravascular tension.

Miscellaneous.—Pulmonary tuberculosis, in its later stages at least, is associated with a low pressure pulse. Cachexias and most simple anæmias give a low pressure, especially the former, which show some of the lowest readings observed. Chronic bronchitis and emphysema generally produce some increase; lead poisoning likewise. Gout is generally considered to be a hypertensive disease. Diabetes in itself probably has no influence on blood pressure.

Summary.—The sphygmomanometer may be expected to show *low pressures* in all cachexias and wasting diseases; in most simple anæmias; in the majority of acute infectious diseases, especially typhoid fever; in shock and collapse, and in the terminal stages of all fatal illness; and in hemorrhages when profuse or occurring in persons whose vaso-motor centres have, from any cause, lost their normal function.

High pressure is found in general arteriosclerosis; in early sclerosis involving only the splanchnic vessels; in myocarditis secondary to or associated with arteriosclerosis or chronic kidney disease; in aortic regurgitation (high systolic wave only); in chronic interstitial, and often in chronic parenchymatous, nephritis; in uræmia; in cerebral hemorrhage; in neuralgia and other conditions associated with mental or physical suffering; and in lead poisoning, and perhaps other chronic toxæmias, as gout.

Full Artery.—The term "full" (*pulsus plenus*) refers to the condition of the artery *between beats.* If the total amount of circulating blood is large, as in conditions of plethora, the vessel will be full and the pulse hard. In all high-tension pulses the artery is full, but not necessarily large. A full but soft and compressible vessel occurs in fevers and other conditions of vascular relaxation, constituting one feature of what the older writers called a "gaseous pulse."

Empty Artery.—The vessel is empty or collapsed (*pulsus vacuus*) *between beats* in aortic incompetence, that portion of the *vis a tergo* derived from the elasticity of the aorta being nullified by the lack of support from the aortic valve. The pulse is comparatively empty in malnutrition and wasting diseases, and to a noticeable extent in mitral stenosis, in consequence of the small quantity of blood discharged from the left ventricle. An extreme grade of emptiness as well as a great diminution in the amplitude of the pulse wave is seen in the "thready" or "running" pulse, indicative of great cardiac weakness and impending death.

Large Pulse.—This refers to a pulse wave of much amplitude or volume (*pulsus magnus*), an unusually great expansion of the artery *during the beat*, usually with low tension. It is found in fevers (bounding pulse), relaxation of the arterioles, aortic incompetency, and occasionally in left ventricular hypertrophy.

Small Pulse.—A small excursion of the artery *during the beat* (*pulsus parvus*) is found in aortic stenosis and mitral lesions, especially mitral narrowing, the quantity of blood delivered into the aorta

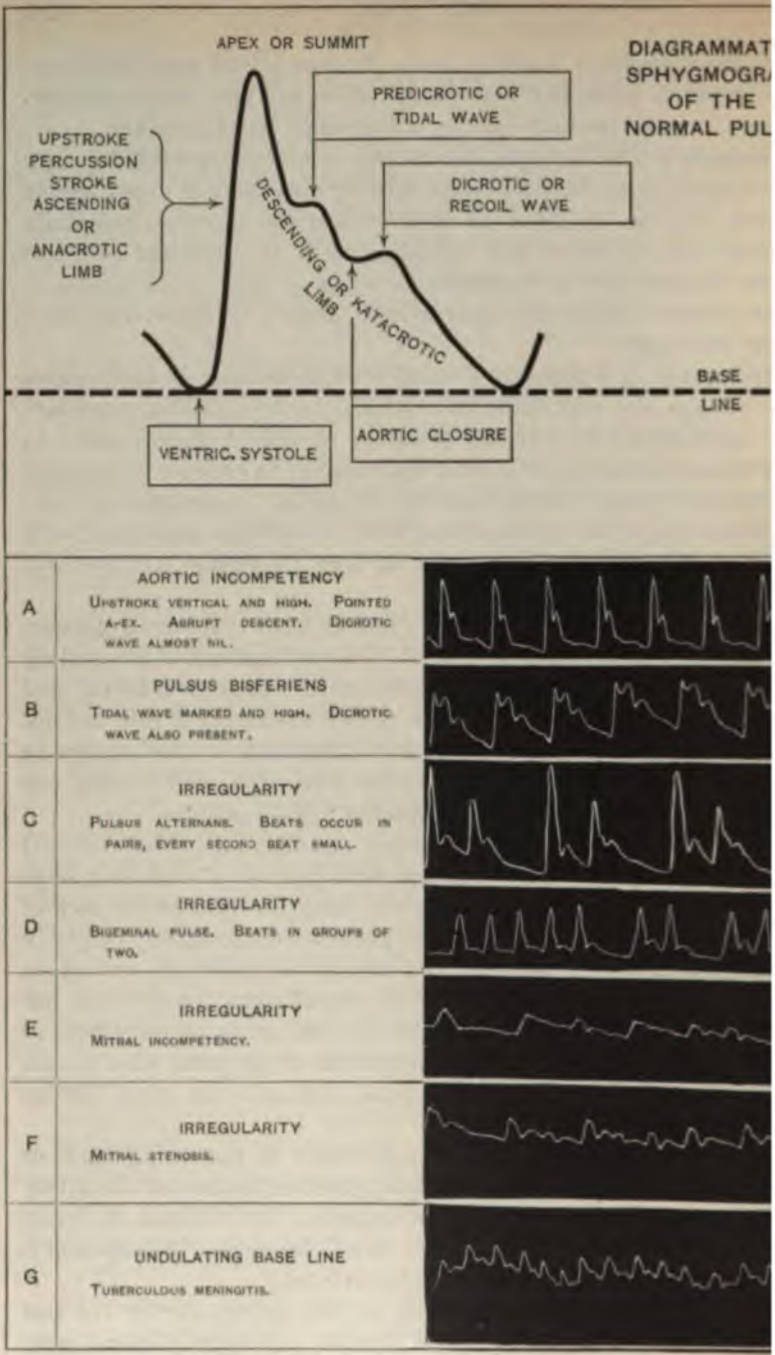


Fig. 131.—Sphygmograms, diagrammatic and actual.

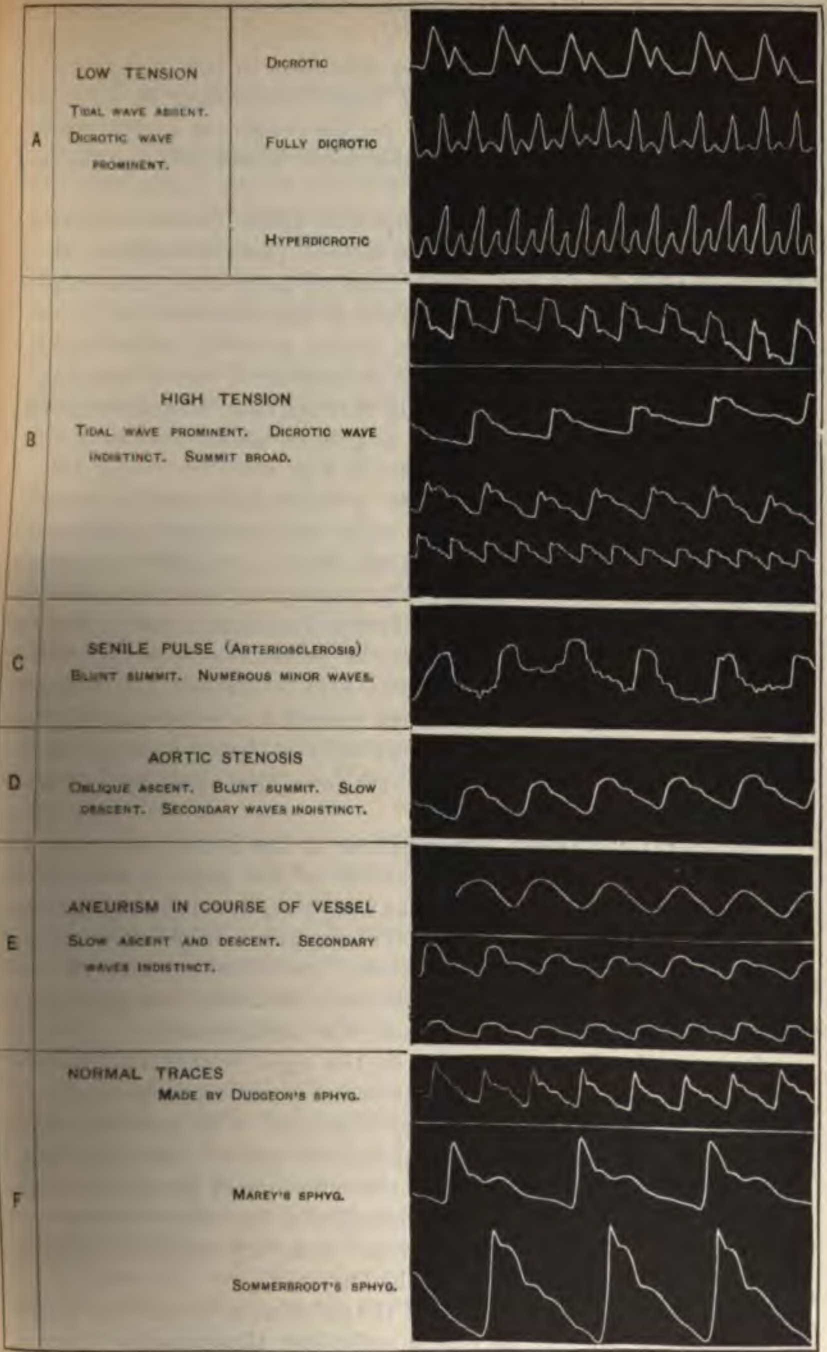

A
LOW TENSION
Tidal wave absent.
Dicrotic wave prominent.
Dicrotic
Fully dicrotic
Hyperdicrotic
B
HIGH TENSION
Tidal wave prominent. Dicrotic wave indistinct. Summit broad.
C
SENILE PULSE (Arteriosclerosis)
Blunt summit. Numerous minor waves.
D
AORTIC STENOSIS
Oblique ascent. Blunt summit. Slow descent. Secondary waves indistinct.
E
ANEURISM IN COURSE OF VESSEL
Slow ascent and descent. Secondary waves indistinct.
F
NORMAL TRACES
Made by Dudgeon's sphyg.
Marey's sphyg.
Sommerbrodt's sphyg.

Fig. 132.—Sphygmograms, actual.

being limited; in aneurism, if the dilatation affects the artery between the heart and the palpating finger, thus acting as a reservoir to extinguish the pulse wave; in cardiac weakness and in wasting diseases. A moderately small pulse wave is not infrequently conjoined with high tension.

Slow or Tardy Pulse.—The adjective refers to the character of the pulse wave—slow rise and slow descent (*pulsus tardus*)—not to the frequency of the beats. This form of pulse wave, also called a long pulse, belongs as a rule to pulses of high tension (*B*, Fig. 132), and is seen in contracted kidney, angina pectoris, arteriosclerosis, and old age. A slow pulse may be indicative of aortic stenosis (*D*, Fig. 132), the blood passing tardily through the contracted orifice. If the ascent of the pulse wave is as gradual as the descent it is significant of an intervening aneurism (*E*, Fig. 132).

Quick Pulse.—A pulse wave with a rapid ascent and an immediate quick falling off of pressure (*pulsus celer*) belongs in almost all cases to a pulse of low tension (*A*, Fig. 132). A "quick" pulse must not be confused with a rapid or frequent pulse. It is found when the arterioles are relaxed as in fevers and anæmia. Aortic regurgitation exhibits this form of pulse in perfection (Corrigan's, shot pulse or water-hammer pulse, *A*, Fig. 131). One can not from the presence of this pulse infer without reserve the existence of aortic incompetence, as it occurs very typically at times in anæmia and other conditions, such as those which cause the subungual or capillary pulse (page 288).

Unilateral and Other Abnormalities of the Pulse.—Delay, weakness, abnormal character or obliteration of the pulse in corresponding arteries of the two sides of the body; or, in rare cases, unusual weakness, extinction or obliteration of the pulse on both sides in particular vessels may be caused in general (excluding anatomical variations) by aneurism, embolism, thrombosis, pressure from tumours or wounds involving the vessels. Particular instances are:

(1) A weak or extinct pulse in the *right radial*, indicative of an aneurism of the ascending aorta or innominate artery; in the *left radial*, of the descending aorta, and if delayed, of an aneurism of the arch between the origins of the innominate and left carotid arteries; in *either radial*, of the presence on the same side of pneumothorax or large pleural effusion, embolism, thrombosis, tumours of the neck or axilla creating pressure upon the vessel and, very rarely, of aneurism of the subclavian, axillary, or brachial arteries.

(2) Weakening or extinction of the pulse in *one femoral* or *one posterior tibial* artery may be due to embolism, thrombosis or tumour; in the same vessels on *both sides* to similar causes, as well as to an

abdominal aneurism, or the excessively rare congenital obliteration of the aorta.

Pulses Possessing Special Diagnostic Value.—(1) *Mitral Stenosis.*—Pulse wave small, its rise and descent rather slow, the artery not well filled, the successive beats irregular in time and strength (*F*, Fig. 131).

(2) *Aortic Stenosis.*—The pulse is small, its rise and fall gradual, the tidal wave marked and not infrequently higher than the initial wave. The successive beats are equal, regular, and not unduly frequent (*D*, Fig. 132).

(3) *Aortic Incompetency.*—The pulse wave is ample, quick (sudden rise, immediate sudden fall), successive beats generally regular and equal, frequency variable (*A*, Fig. 131).

(4) *Arteriosclerosis and Atheroma of Aorta.*—A pulse wave of good amplitude, of somewhat slow ascent, of considerable duration (the artery remaining dilated for a noticeably long period), of slow descent—resembling, indeed, the pulse of aortic stenosis, except that the latter is smaller (compare *C* and *D*, Fig. 132)—suggests very strongly the existence of atheromatous changes, especially in the aorta. The characters of this pulse are due to the loss of aortic and general arterial elasticity which results from the degenerative process. It is often called the "senile pulse."

(5) *Aneurism.*—A discrepancy between the pulse waves in symmetrical arteries, either in time or character, is more significant of aneurism than the character of the wave in a single artery. Yet if an aneurism lies in the course of an artery the pulse wave exhibits an ascent and descent which depart from the normal in being of nearly equal duration (*E*, Fig. 132).

(6) *Myocarditis.*—A pulse wave which is unusually soft and small, conjoined with intermittency or irregularity in force and strength, and without other symptoms of cardiac disease, should lead one very strongly to suspect myocarditis if the causative conditions of the latter are present.

The Sphygmograph.—The sphygmograph is useful in making a permanent record of the pulse for future comparison; in showing graphically some, but not all, of the elements of the pulse; in teaching precision in the pulse examination; in analyzing some of the finer details which may have been too delicate or insignificant for palpation (small waves or oscillations); and in corroborating a diagnosis previously made by palpation. Although there are some points which are not shown at all, or not as well, by the sphygmograph as by palpation (fulness and size of artery, thickness of walls), it is esteemed by those who understand how to use and interpret it as a valuable supplement to the educated finger.

Technic of the Sphygmograph.—The instruments of Marey, Mahomed, and Sommerbrodt are in use, but the most convenient as well as reliable form for clinical use is that of Dudgeon (Fig. 133).

There is no clinical instrument the value of which depends so largely upon the person who is using it. Two tracings, taken one after the other from the same artery by different observers, may differ considerably in form; but each examiner, if well practised, will reach substantially similar conclusions. The technic is as follows:

The slips of paper upon which the tracing is to be made should be procured from the instrument maker, as they require to be of special texture and accurate cut. Otherwise the needle does not play freely over the surface or the slip binds on the rollers. To smoke the slip, place it in a holder, a strip of tin with the ends turned over half an inch, by which the extremities of the slip are held and covered. In a room free from draughts, place a piece of gum camphor on some suitable metal or porcelain surface. Ignite the camphor, using, if required, a drop or two of alcohol to facilitate the lighting. Move the strip in the holder to and fro over the ascending column of smoke, taking care not to scorch the paper, and endeavouring to get an even deposit of carbon over its surface. The makers furnish a box in which half a dozen smoked slips can be carried for outside use.

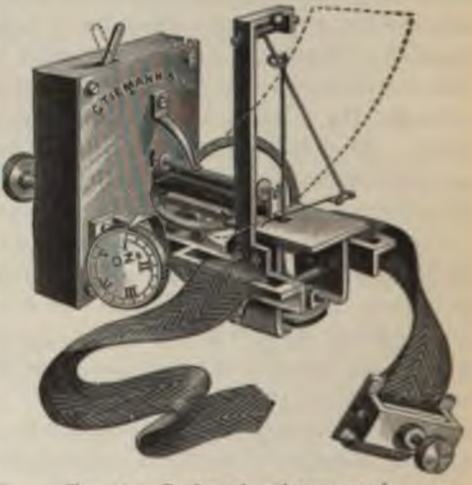


Fig. 133.—Dudgeon's sphygmograph.

After the tracing is made, write upon the smoked surface with a pin point or a pen with one half of the nib broken off, or with ink upon the unsmoked ends, name, date, vessel from which made, pulse, respiration, temperature, and disease. Pour into a footed cylindrical jar, 6 or 7 inches long and 1¼ inch in diameter, or into a saucer, a quick-drying varnish—viz., photographer's negative varnish, *or* gum benzoin 1 ounce, alcohol 6 ounces, *or* gum damar 1 ounce, rectified benzoline 6 ounces. The tracing may be dipped slowly into the jar, or slidden, smoked side uppermost, through the solution in the saucer, and allowed to dry. A second coat may be applied if the tracing is to be much handled.

To Use the Instrument.—(1) Find the exact spot where the radial beat is felt most distinctly, and mark it *accurately* (ink or aniline pencil). (2) Wind the clockwork and insert the smoked paper. (3) Let the patient take an easy position and hold out the hand toward you, palm upward and slightly dorsiflexed, fingers quiet. *If the band is to be used:* (4) Having passed the free end of the band through the clamp, slip it over the patient's hand and tighten it moderately. (5) Place the metal pad over the artery, clock case nearest elbow, and steady it with one hand while the other still further tightens the band until the needle plays freely at or about the central line of the paper strip, not running off either edge. *If the band is not employed*, support the patient's hand and wrist on the arm of a chair, edge of bed or observer's knee, and hold the instrument with one hand. This can be readily done after a little practice, or the band may be passed around the wrist and held from underneath without clamping. (6) Turn the milled head which regulates the pressure of the spring until the needle attains its greatest amplitude of movement. The pressure is graduated in ounces, but the nominal reading is never reliable. (7) Push over the projecting lever which starts the clockwork, and either stop it just before the slip runs out or catch the latter in the free hand.

It is often desirable to take two tracings at varying pressures, or, better yet, to stop the clockwork after half the slip has been written upon and increase the pressure to the maximum, thus exhibiting the effect of medium and heavy pressures upon the same slip. A useful wrinkle (DANA) is to stick 2 or 3 thicknesses of adhesive plaster upon the metal pad which is applied to the artery. The prolongation of this artificial finger makes it mechanically easier to set the needle in motion.

Interpretation of the Tracing.—If it is clearly understood that an *upstroke* in the tracing corresponds to a *rise of pressure* in the vessel, and a *downstroke* to a *fall of pressure*, the interpretation of the tracing is relatively easy. After what has been said concerning the palpation of the pulse, a study of the sphygmograms (Figs. 131, 132) in connection with the descriptions about to be given will be sufficient without detailed reference.

(1) *The Normal Tracing and its Elements.*—A tracing from a normal pulse (diagram, Fig. 131) shows an almost vertical upstroke (percussion stroke, ascending or anacrotic limb) and a sloping downstroke (descending or katacrotic limb), the latter interrupted by two principal secondary elevations, the first of which is the tidal or predicrotic wave, the second the recoil or dicrotic wave. Subsequent to the dicrotic wave there may be some minor elevations.

The percussion stroke or ascending limb is due to the sudden increase of pressure caused by the transmission of the force expended by the left ventricle in driving its contents into the aorta, along the practically incompressible blood columns in the arteries. The walls of the suddenly distended artery then reactively contract and again expand by virtue of their elasticity, thus giving rise to the pretidal notch and tidal wave, after which the pressure again falls until the moment of closure of the aortic valve. The blood column, which has fallen back against the aortic valve, is suddenly checked, and the resultant recoil produces an increase of pressure which forms the dicrotic wave in the tracing. Subsequent small elevations are, like the tidal wave, due to elastic oscillations of the arterial walls. It is proper to state here that according to some authorities the tidal wave is caused by the blood stream coursing through the artery, but in view of the fact that the tidal wave is large if the tension of the arterial wall is great—i. e., the more immediate its power of elastic recovery—the foregoing explanation is probably correct.

The characters of a normal pulse tracing may be summarized as follows: (1) *Upstroke*, straight, nearly vertical, of moderate amplitude; (2) *apex*, moderately acute; (3) *descent*, gradual; (4) *tidal wave*, small; (5) *dicrotic wave*, well marked.

(2) *Diagnostic Indications from the Sphygmograph.*—Inspect the tracing systematically with reference to the following points:

Is the upstroke long or short, vertical or sloping?

Is the apex pointed or broad and blunt?

Is the tidal wave marked, faint, or absent?

Is the dicrotic wave marked, faint, or absent?

Are the successive beats regular, irregular, or intermittent?

Is the line of descent regular or irregular?

Is the base line (the line connecting the bases of the successive beats) straight or irregular?

As a whole, of what is the trace characteristic or indicative?

The significance of the variations in the individual elements of the tracing are:

1. Long Upstroke.—Corresponds to large volume, and indicates a quick systole or the relaxed arterioles, and free capillary circulation of low tension or aortic regurgitation.

2. Short Upstroke.—Corresponds to small volume and indicates mitral regurgitation, aortic stenosis, aneurism, or obstructed peripheral circulation (high tension).

3. Vertical Upstroke.—Corresponds to a quick systole of either a weak or strong heart, or a large amount of blood discharged at each systole, and is often associated with a long upstroke in low-tension pulse and aortic incompetency.

4. Oblique or Sloping Upstroke.—May be due to a considerable layer of fat over the artery or slow filling, as in aortic stenosis or mitral incompetency, or aneurism in the course of the vessel, or arteriosclerosis or high tension; or, if these causes are absent, a weak left ventricle.

5. Pointed Apex.—Indicates an unobstructed peripheral circulation (low tension) or aortic incompetency.

6. Blunt or Broad Apex.—Indicates strong heart and obstructed peripheral circulation (high tension), or aortic stenosis, or arterio-

sclerosis, or aneurism in the course of the vessel, or, last but not least, too great pressure by the spring of the sphygmograph.

7. Marked Tidal Wave.—Indicates strong and obstructed peripheral circulation (high tension), or aortic stenosis, or arteriosclerosis.

8. Small or Absent Tidal Wave.—Indicates a weak heart, or a strong heart with free peripheral circulation (moderate or low tension), or mitral or aortic incompetency.

9. Marked Dicrotic Wave.—Indicates a weak or moderately strong heart with free peripheral circulation (low tension), in some cases high tension with a failing heart.

10. Small or Absent Dicrotic Wave.—Indicates obstructed peripheral circulation with a strong heart (high tension), arteriosclerosis, aortic stenosis, aneurism, or (because of the failure of the recoil) aortic incompetency.

11. Irregularity of the Line of Descent.—Is seen in mitral stenosis and regurgitation.

12. Irregularity of the Base Line.—Undulations of the base line corresponding to the respiratory acts occur in conditions attended with dyspnœa or irregular breathing from involvement of the nerve centres (*G*, Fig. 131).

13. Irregular or intermittent beats are graphically illustrated in the sphygmographic tracing, but require no further notice than has been given.

SECTION XXXII

EXAMINATION OF THE LUNGS AND PLEURÆ

The lungs are to be examined by inspection, palpation (including mensuration and sometimes spirometry), percussion, and auscultation.

I. TOPOGRAPHICAL ANATOMY

Certain facts regarding the relation of the boundaries and lobes of the lungs and the pleural sacs to the external surface of the thorax must be clearly in mind preceding an examination of these organs.

Right Lung.—The *apex* (see Plates I, II) rises from 1 to 1¾ inch above the level of the clavicle. From here the *anterior border* runs downward, forward, and inward, passing nearly behind the right costo-sternal articulation to the midsternal line at the level of the 2d rib. From this point it runs vertically downward to the level of the

6th chondro-sternal articulation, where it turns sharply to the right and becomes the lower border. The *lower border* follows the 6th rib to the right mammillary line, cuts the 8th rib in the midaxillary line, the 10th rib at the scapular line, and the upper border of the 11th rib close to the spinal column. For brevity, remember, front, 6th; side, 8th; back, 10th rib. In old people the lower borders of the lungs extend 1 rib farther down, in children they lie 1 rib higher than those just given.

The right lung has 3 lobes (Fig. 134). Posteriorly the upper and lower lobes are separated by a fissure which starts at the spinal column, on a level with the spine of the scapula, and runs outward, downward, and forward to the 4th rib in the axillary line, where it divides into 2 secondary fissures. The uppermost of these runs almost horizontally forward, and reaches the anterior border of the lung at about the level of the 4th cartilage. The lower fissure passes downward and somewhat forward to the lower border of the lung in the mammillary line. These two fissures bound the middle lobe.

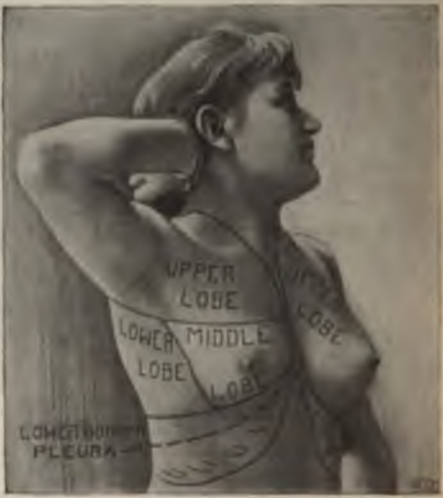


Fig. 134.—Showing the lobes of the lung (and the lower limit of the pleura) on the right side of the chest.

Left Lung.—The *apex* of the left lung and its *anterior border*, as far down as the 4th rib, correspond to those of the right lung, except that this border lies farther from the midsternal line. At the level of the 4th rib the anterior border curves outward, downward, and then moderately inward to the 6th rib, exposing a somewhat semicircular area of the pericardium (superficial or exposed cardiac dulness). From this point on the 6th rib the lower border runs outward and around to the spinal column, its course corresponding in all respects to that of the lower border of the right lung, save that it lies a trifle lower.

The left lung has two lobes (Fig. 146), upper and lower, separated by a fissure, which begins and runs as in the right lung, but, instead of bifurcating, passes downward and forward to end at the 6th rib in the left mammillary line.

It will be seen from this description that we have

Posteriorly, on *both sides* (Fig. 135), from above downward, upper lobe as far as the spine of the scapula; below this, lower lobe.

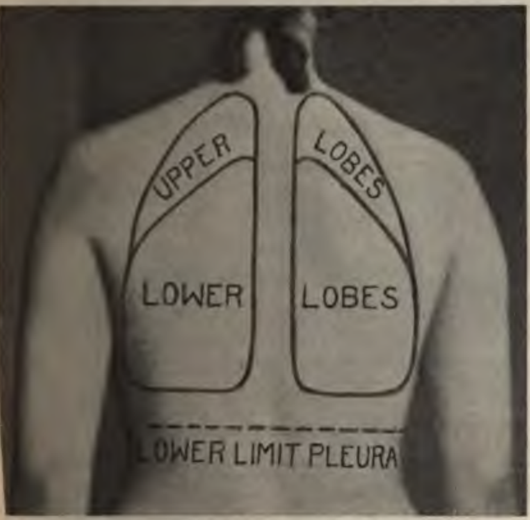


FIG. 135.—Showing the lobes of the lung and the lower limit of the pleura posteriorly.

In front, on the *right side*, upper lobe as far down as the 4th rib; from the 4th to the 6th, middle lobe; on the *left side*, upper lobe alone.

Laterally on the right, in the midaxillary line, upper, beginning of middle, and the lower lobe.

Laterally on the left, in the midaxillary line, upper and lower lobes.

Pleuræ.—The pleural sacs extend below the lower border of the lungs to a very considerable extent. In the mammillary line they lie 2 inches, in the midaxillary line 3½ inches, in the scapular line 1½ inch lower than the edges of the lungs (Fig. 135). The anterior edge of the left pleural sac below the 4th rib lies nearer the midsternal line than the corresponding part of the anterior border of the left lung. The surfaces of the reflected pleura forming this complementary pleural space or sinus are in contact except when separated by fluid in the pleural cavities, or thrust apart by the edges of the lungs as the latter advance and retreat during inspiration and expiration.

II. THE PHYSIOLOGY OF THE LUNGS

The lungs may be looked upon as large airbags minutely subdivided into lesser sacs—infundibuli—the walls of which are bulged outward by numerous concavities averaging $\frac{1}{100}$ of an inch in diameter, the air-vesicles or alveoli. The total area of the latter is about 200 square metres. The air enters the vesicles through the trachea, the bronchi, the bronchioles, the infundibuli, and is separated from the blood in the abundant pulmonary capillaries by an exceedingly thin and delicate membrane, through which oxygen passes to the blood and carbon dioxide to the air in the vesicles. For clinical purposes it is important to study the movements of respiration with reference to their physics and the muscular mechanism concerned; and the nervous mechanism which controls these movements.

Muscular Mechanism and Physics of the Respiratory Movements.—Each *respiration* consists of two acts, *inspiration*, an increase in the 3 diameters of the thorax, and *expiration*, a sequent lessening of the same diameters. During inspiration the transverse diameter is increased by the elevation and rotation outward and upward of the ribs; the antero-posterior is lengthened by the upward, outward, and forward movement of the ribs and costal cartilages, thus carrying the sternum also forward and upward; and the vertical diameter gains by the descent of the diaphragm.

The lungs themselves are extremely elastic, and after inflation tend to collapse very promptly. *In situ* they can not collapse, because they fit snugly in the air-tight thoracic cage, but if air is admitted to the pleural cavity the corresponding lung shrinks at once to a much smaller bulk. It is obvious that, as under normal circumstances the lungs exert a constant tractive force upon the diaphragm and thoracic walls, a pressure less than that of the atmosphere (negative pressure) must exist in the thorax outside of the lungs (intra-thoracic pressure), which is to be carefully distinguished from the pressure of the air in the trachea, bronchi, and lungs (intra-pulmonary pressure). As the thorax enlarges during inspiration the lungs follow its boundary walls in every direction, i. e., expand, thereby producing a diminution of the intra-pulmonary pressure, and in order to restore the equilibrium between this and the outside atmospheric pressure there is an inflow of air into the trachea and lungs—practically a bellows-action.

Inspiration is active, and accomplished by the diaphragm and other muscles to be subsequently enumerated. Expiration is passive, and is due mainly to the natural elasticity of the lungs, costal cartilages, and abdominal walls, as well as to the negative intra-thoracic pressure, which tends to draw the relaxed diaphragm upward and to bring the thoracic cage into the resting form.

Coincidently with the movements of the chest the nostrils dilate and the glottis widens during inspiration, both narrowing during expiration. If the respiration is difficult or laboured the mouth opens, the facial muscles work spasmodically, and the larynx descends with each inspiration, presenting the familiar clinical picture of dyspnœa.

Nervous Mechanism of the Respiratory Movements.—The nervous mechanism required to initiate and regulate the co-ordinated action of the numerous muscles which execute the respiratory movements consists of a respiratory centre, with afferent and efferent nerves (Fig. 136).

(1) The *respiratory centre* lies in the lower part of the medulla (Reichert). This centre is divided into 2 halves so closely connected that they form functionally a single centre. The right half is connected with the right lung and the respiratory muscles of the right side, and *vice versa*. Each half is physiologically again subdivided into 2 portions: an *inspiratory* or *accelerator centre*, connected with the muscles of inspiration, and an *expiratory* or *inhibitory centre*, controlling the expiratory muscles in forced expiration.

The power of rhythmic action is inherent in the nerve cells of the centre, but it requires a continuous excitation, which is supplied by the oxygen and carbon dioxide in the circulating blood and the stimulus received from the lungs by way of the pneumogastrics.

(2) The main *afferent* or *sensory* nerves are the pneumogastric, glosso-pharyngeal, trigeminal, and cutaneous.

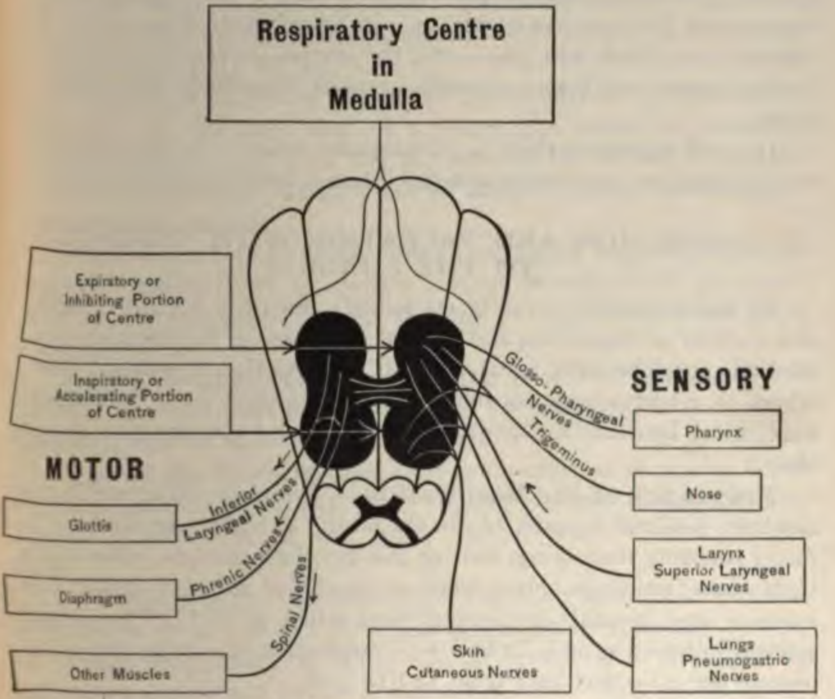

Respiratory Centre
in
Medulla
Expiratory or Inhibiting Portion of Centre
Inspiratory or Accelerating Portion of Centre
MOTOR
Glottis
Diaphragm
Other Muscles
Inferior Laryngeal Nerves
Phrenic Nerves
Spinal Nerves
SENSORY
Glosso-Pharyngeal Nerves
Trigeminus
Pharynx
Nose
Larynx Superior Laryngeal Nerves
Skin Cutaneous Nerves
Lungs Pneumogastric Nerves

Fig. 136.—Diagram of the respiratory centre.

The *pneumogastric* nerve contains fibres some of which convey impulses to the inspiratory, others to the expiratory centre. These impulses are derived from the movements of the lungs, lung expansion giving rise to expiratory, lung collapse to inspiratory, impulses, one alternating with the other. The *superior laryngeal* and *glossopharyngeal* act upon the inhibitory centre—e. g., arrest of respiration from foreign body in larynx, or during the act of swallowing. The *trigeminal nerves* usually arrest, less frequently increase the rapidity of, the respiratory movements—e. g., inhalation of irritating gases; and the *cutaneous* nerves cause primarily accelerated and deep breathing—e. g., a dash of hot or cold water on the skin. Other (psychic) impulses arise from the centres of the brain.

(3) The *efferent* or *motor* nerves are: the inferior or recurrent laryngeal branch of the pneumogastric, which widens the glottis during inspiration; the very important phrenic nerve, innervating the diaphragm; and the various spinal nerves which control the other respiratory muscles.

The muscles which act in *quiet inspiration* are the diaphragm, quadrati lumborum, serrati postici inferiores, scaleni, external intercostals, and the levatores costarum. In *forced inspiration* additional muscles are called into play—viz., the sterno-cleido-mastoids, infrahyoids, greater and lesser pectorals, trapezii, rhomboids and erectores spinæ.

In *quiet expiration* there is no muscular action. In *forced expiration* the abdominal muscles and the internal intercostals are at work.

III. INSPECTION AND PALPATION WITH REFERENCE TO THE LUNGS

By sight or touch (usually conjoined) the rate, rhythm, amount and manner of expansion, and general character of the breathing are studied, together with vocal, rhoncal, and friction fremitus. Elsewhere (*q. v.*) have been considered the shape, pulsations, tenderness, anatomical landmarks, topographical areas, and mensuration of the thorax.

Frequency of the Respiration.—The respiration rate in the newborn is 44; at 5 years, 26; in the adult, 16 to 20. In health it is faster standing than lying, during the day than at night, after meals than when fasting, in spring than at the end of summer, and during exercise and mental excitement than when at rest. The normal pulse-respiration ratio is 1 : 4; i. e., 1 respiration to 4 pulse beats. In disease the ratio may vary from 1 : 1 to 1 : 8.

To count the respirations, one may watch the rise and fall of the chest or, better, the upper abdomen; lay the hand lightly on the lower thorax; or perhaps hear the breathing. In many individuals the consciousness of being watched will cause involuntary alterations in the frequency and rhythm of the respiration, and it is best, if practicable, to rate the breathing while ostensibly counting the pulse or taking the temperature. The effects of exertion or mental activity should be allowed to pass off. In children, when possible, the breathing should be taken while asleep.

In disease the respiration rate may be *increased* or *diminished.*

(1) **Rapid Respiration.**—Increased frequency of breathing may exist without the presence of dyspnœa (difficult or laborious breathing), although the two are frequently associated. Dyspnœa (*q. v.*) is considered separately.

Muscular exertion causes rapid breathing because of the influence upon the respiratory centre of certain substances which enter the blood from the muscles as the result of metabolic changes. Mental excitement is at times a potent factor in the same direction. The respiration is accelerated in fever, especially in children, by the influence of the heated blood upon the medulla. The breathing may be much more rapid in lobar pneumonia than is warranted by the extent of the local lesions, suggesting a special action of the pneumotoxine upon the respiratory centre.

Hysteria is occasionally responsible for an extremely high respiration rate. In one case, that of a young girl under my care, the breathing varied from 90 to 100 per minute for several hours, and the only subjective complaint was of fatigue due to the continued rapid muscular action.

From a diagnostic point of view an increased respiration rate is suggestive primarily of pulmonary disease, secondarily of the conditions already mentioned as well as those to be considered under the head of dyspnœa.

(2) **Slow Respiration.**—The breathing is slowed in many of the varieties of coma, also in collapse, and by poisoning with aconite, antimony, chloral, chloroform, and opium.

Type of the Respiration.—Upon observation it may be found that the movement of the upper thorax during respiration predominates over that of the lower thorax and abdomen or *vice versa.*

(1) **Thoracic Type Predominating.**—Costal or thoracic respiration is normal in women at and after the age of puberty, largely because of heredity and modes of dress. Costal breathing in a man or its presence to excess in a woman is indicative either of dyspnœa, real or subjective (*q. v.*), or of some condition limiting the mobility of the diaphragm, abdominal respiration depending upon the full and free action of the latter. The action of the diaphragm is recognised by its causing protrusion of the epigastric region during inspiration. If this protrusion is absent, or if the epigastrium and upper abdominal walls suddenly recede or are sucked in during inspiration, it is

good evidence, in the absence of any form of stenosis of the upper air passages, that the diaphragm is not working.

Excessive upper costal or diminished abdominal breathing may be caused by pressure upon the diaphragm, by ascites, meteorism, or abdominal tumours or enlargements. A very large pericardial effusion by its weight may act similarly. Inflammation of the diaphragmatic pleura or peritoneum is a cause of increased thoracic breathing. Paralysis of the diaphragm affords a typical example of absent abdominal and excessive thoracic respiration. In some cases it is possible by careful palpation to determine that the paralysis is unilateral, one side still acting. In emphysema, although the actual expansion of the chest is much less than normal, there is an exaggerated up-and-down (vertical) movement of the thorax. Finally, the marked superior costal breathing of the hysterical patient is familiar.

(2) **Abdominal Type Predominating.**—Abdominal breathing normally predominates in children of both sexes and the adult male. If this type of respiration is exaggerated in a man, or if it is present together with diminished or absent thoracic breathing in a woman, it is an abnormal condition.

The causes of excessive respiratory action of the abdomen—i. e., of the diaphragm—are found in conditions which render movement of the thorax painful, such as pleurisy, pleurodynia, or fracture of a rib, or which mechanically hinder thoracic expansion, as in double pleural effusion, calcification of the costal cartilages, emphysema (permanent inspiratory form of the thorax throwing extra work upon the diaphragm), the rare scleroderma of the chest wall, and ossifying myositis. Inaction of the thorax may also be due to paralysis of the muscles of inspiration, as in injury or disease of the cervical portion of the cord or bulbar paralysis, or spasm of the same muscles in strychnine poisoning or tetanus.

Degree of Respiratory Expansion.—The amount of the respiratory movement of the thorax should be investigated by inspection, palpation, and mensuration. The chest should be inspected in a good light from the front, side, back, and, last but not least, from above, watching carefully for defective or excessive motion, either general or local. The warmed hands should be laid flat, first upon the front of the chest, then one upon each side, in order to confirm or modify the results of inspection.

Mensuration (*q. v.*) should be employed to determine with accuracy the amount of expansion. If the expansion is less than 2 inches in men and 2½ inches in women, it is below the normal average.

In this connection the spirometer, if at hand, is a useful apparatus, of which there are several varieties. By this instrument one can measure the number of cubic inches of air exhaled. It consists essentially of a bell jar or capped cylinder submerged in water and counterpoised, with a tube to admit the expired air under it. As the jar rises, a properly proportioned scale indicates the volume of air admitted. The only diagnostic fact of importance to be obtained from the spirometer, and it is of value, is a knowledge of the vital capacity of the lungs—i. e., the volume of air which can be expired after taking the deepest possible inspiration. The normal vital capacity of a man 22 years old and 5 feet 8 inches in height averages from 230 to 240 cubic inches, 3½ cubic inches for each inch in height. In a woman of 19 years of age and 5 feet 2½ inches in height it averages 145 to 150 cubic inches, 2.3 cubic inches for each inch in height. It decreases somewhat with age.

(1) **Deficient Expansion.**—The defect in expansion may be general, affecting the thorax as a whole, or localized on one side or portion of the chest.

If the vital capacity is from 10 to 70 per cent below what it should be for the particular individual, the existence of pulmonary tuberculosis, or at least a strong predisposition to the disease, may justly be suspected. Similar conclusions may be drawn if the general expansion is found by measurement to be much below 2 inches. It is claimed that the expansion is diminished in exophthalmic goitre, but the statement lacks confirmation.

A *general* poor expansion may be due to the limiting effect of the chest pain incident to pleurisy, pneumonia, pleurodynia, fractured ribs, angina pectoris and intercostal neuralgia; obstructed upper air passages, as in pressure on the trachea (by mediastinal tumours or aneurism), laryngeal stenosis, tumour, paralysis, or spasm; paralysis or tetanic spasm of the respiratory muscles; and asthma and emphysema, the lungs being distended to an extent which will permit but slight additional expansion. Shallow breathing may be simply a part of the general muscular weakness of adynamic conditions—e. g., collapse, syncope, or the typhoid state in general.

Unilateral deficiency of expansion, one or the other lateral halves of the chest exhibiting an obvious lack of inspiratory movement, is indicative of some diseased condition which prevents inflation of the corresponding lung. It is a diagnostic symptom of importance, and may find its explanation in a mechanical hindrance to expansion upon one side by the presence of fluid or air in the pleura, or extensive pleural adhesions, or (on the right side) by an enlarged liver; in obstruction of a main bronchus by a foreign body or the pressure of an aneurism or tumour, or in disease confined to one lung—tuberculous, fibroid, pneumonic, cancerous, hydatid, or atelectatic.

A *local* deficiency in expansion, or a *lagging* of one portion of the chest wall behind the remainder during inspiration, is suggestive of circumscribed disease of the lung or pleura. Deficient expansion under the clavicle is seen in phthisis; of the upper or lower portion of the thorax in an apical or basal pneumonia; of the pericardial space in pericardial adhesions, of various localities with pleural adhesions, and, especially in a child, in local atelectasis or collapse of the lung.

(2) **Increased Expansion.**—Increased *general* expansion occurs after exercise or mental excitement, and in hysteria and some forms of dyspnœa. *Local* or *unilateral* increase of expansion may be compensatory—i. e., a portion or the whole of one lung may expand beyond its usual limits in order to perform the additional work thrown upon it by disease in the remaining portion, or in the opposite lung.

Respiratory Bulging and Retraction.—Retraction (during inspiration) or bulging of the intercostal spaces (during inspiration or expiration) may be either general or circumscribed. In judging of their extent it must be remembered that the ribs are prominent and the interspaces marked in a thin or emaciated thorax, while the converse condition is found in stout or muscular persons.

(1) Retraction of the interspaces is usually associated with deficient expansion and inspiratory dyspnœa (difficult entrance of air). General retraction is most marked in obstruction of the upper air passages, and extensive bilateral broncho-pneumonia (in children). *Unilateral* or *local* recession indicates obstruction of a bronchus or circumscribed interference with expansion, as in the pulmonary collapse of infants, the affected portion not distending, pleuritic adhehesions, and other similar conditions.

(2) Bulging of the interspaces *during expiration* is usually conjoined with expiratory dyspnœa (difficulty in emptying the lungs), as in asthma and emphysema. In these diseases there is also inspiratory dyspnœa, and the interspaces alternately bulge and retract. Bulging *during inspiration* is seen only above the clavicles, and is an evidence of emphysema, the apex of the hypertrophied lung protruding above its normal level.

(3) **Litten's Phenomenon.**—The movements of the diaphragm may be seen if the lateral walls of the chest are exposed and properly illuminated. The patient, lying upon his back, is placed so that his feet point toward a window, or, if that is impracticable, a lamp or movable electric light is held at the foot of the couch. In either case it is essential that all side lights should be excluded. The observer then sits or stands at the side of the patient, and desires

him to breathe deeply. During inspiration a narrow shadow moves downward, in the mid-axillary line, from the 6th to the 9th or 10th rib, for a distance normally of from 2½ to 3½ inches. The shadow may be seen, but less distinctly, to move upward during expiration. In normal subjects the shadow is always present, except in the obese, or in those who can not, or will not, breathe deeply. In the latter voluntary coughing may elicit it. Weakness or extreme prostration may lessen, but not entirely abolish, this phenomenon. The existence of the moving shadow is due to the fact that, at the end of expiration, the lower portion of the highly arched diaphragm is in contact with the chest walls as far up as the 6th rib; during inspiration the diaphragm flattens, recedes from the chest wall, and permits the lower borders of the expanding lungs to interpose between it and the chest wall; the lungs thus enter the complemental pleura, and fill out the corresponding intercostal spaces. The extent of movement of the shadow is therefore a measure of the excursion of the diaphragm. In this respect Litten's method is also a fairly good substitute for the fluoroscope.

A *normal excursion* and a proper respiratory action of the thorax constitute good evidence of satisfactory pulmonary capacity. A *diminished excursion* is present in the early stage of pulmonary tuberculosis, or may occur in conditions of great prostration from any cause. *Absent excursion* is seen in pleural effusions, pneumonia of the lower lobes, and in emphysema, as the diaphragm in such cases is held away from contact with the chest wall. It may be due to pleuritic adhesions between the chest wall and the diaphragm, whereby the descent of the latter is prevented. A contracted (fibroid) or adherent lung may be unable to descend into the complementary space. A very large ascites also may abolish the phenomenon, although abdominal tumors, either solid or fluid, provided they are not of great size, do not hinder the movement of the diaphragm and the production of the shadow. This fact may afford a valuable differential sign in deciding between a right pleural effusion or enlargement of the liver on the one hand, and a subphrenic abscess on the other. The finding of the Litten phenomenon speaks for the subphrenic abscess.

Rhythm of the Respiration.—Normally inspiration passes into expiration without an observable pause. Inspiration is rather more rapid and shorter, in the ratio of 5 : 6, than expiration. If the breathing is unusually slow, there is a pause at the end of expiration. While quiet, the successive respirations are regularly rhythmical, but this regularity is subject to variations, sometimes purposive, sometimes involuntary. The breathing is very irregular in children while awake, or during sleep if restless. The disturbances of rhythm which possess some diagnostic and prognostic value are:

(1) **Sighing.**—An occasional slow, deep inspiration, followed by a somewhat rapid expiration, is normal in many persons while at rest, in order to more fully oxygenate the blood. It is seen as the result of emotion, and in the hysterical, hypochondriacal, and melancholic patient. The sighing respiration of hemorrhage, collapse, or syncope is characteristic. An overdistended stomach, by causing pressure on the diaphragm, may be responsible for frequent sighing; it is seen in cases of dilated heart, Addison's disease, meningitis, and lesions of the medulla, and is very common in typhoid fever.

(2) **Simple Irregularity.**—A more or less constant irregularity in the time intervals and the depth of the respirations may be due to collapse, sudden and overwhelming cerebral apoplexy, meningitis, brain tumours, especially lesions of the medulla, and chorea (involvement of the respiratory muscles). A pause at the end of inspiration is very characteristic of acute pneumonia.

(3) **Cheyne-Stokes Breathing.**—This is a peculiar form of rhythmical irregularity which is not infrequently encountered. The patient ceases to breathe (period of apnœa), then a slight, slow respiration occurs, followed by others which progressively increase in depth and rapidity, until an acme of deep and hurried breathing is reached (period of dyspnœa), after which follows a corresponding gradual diminution in rate and depth until the respiration again ceases. The duration of the cycle (from apnœa to apnœa) varies from 30 seconds to 2 minutes. The Cheyne-Stokes type of respiration is particularly noticeable when the patient is quiet, asleep or comatose. If awake and talking, its presence may be overlooked. It is not uncommon for the patient to be unconscious during the apnoic period, and to wake, move, or speak with the onset of the hurried breathing. The supervention of this curious variety of breathing is always a grave omen and usually foretells a fatal issue, although cases are occasionally seen in which recovery follows, especially if occurring in the course of an acute and not a chronic disease. It may last for hours, days, and, very rarely, even for months. There are various minor and allied forms—e. g., sudden deep breathing, following a cessation (cerebral respiration), but these are better classed under (2) preceding.

In the majority of instances it is associated with apoplexy (toward the end), chronic nephritis (uræmia), tumour of the brain, tuberculous meningitis, or degeneration of the heart muscle. Less frequently it is observed as a result of cardiac valvular defects and consequent embolism, diabetes, and certain acute diseases, notably typhoid fever, pneumonia, pertussis, cerebro-spinal fever, scarlet fever, and septicæmic conditions.

Other Characters of the Respiration.—Certain other points of some clinical importance may here be noted:

(1) *Jerking Respiration.*—A spasmodic performance of either inspiration or expiration or both is occasionally seen. *Inspiration* may be jerking in asthma, hysteria, and hydrophobia; jerking *expiration* is seen in pleurodynia, fractured rib and the early stage of acute pleurisy, because of the sudden stab of pain in the side which arrests inspiration and calls for prompt relief by expiration; *both* mainly in cases where thoracic breathing predominates.

(2) *Stertorous Respiration.*—Snoring—the sounds arising from the vibration of the soft palate when breathing through the mouth and nose at the same time—usually requires unconsciousness or muscular relaxation for its production. Aside from its frequent occurrence during sleep in healthy individuals, especially if overtired, it is observed as a symptom in profound coma (apoplectic, uræmic, diabetic, etc.), narcotic poisoning, and paralysis of the soft palate. It is also a prominent and continuous attendant of postnasal adenoids and enlarged tonsils in children, because of the necessary existence of mouth breathing during sleep.

(3) *Stridulous Respiration.*—When the air in passing through the larynx, particularly during inspiration, is accompanied by a sound variously described as whistling, harsh, shrill, creaking, or screechy, it is termed stridor or stridulous breathing. It is always of laryngeal origin, and is due either to a mechanical obstruction in the larynx (inflammatory swelling, œdema, membrane, tumour, foreign body) or to spasm or paralysis of certain laryngeal muscles with a consequent defective opening of the glottis—e. g., spasmodic croup, laryngismus stridulus, strychnine poisoning, tetanus, hydrophobia, pressure upon the inferior laryngeal nerve by aneurism, mediastinal tumour or enlarged bronchial glands, and in rare cases displacement of the heart or the trachea. In certain forms, because of the peculiar character of the breathing, it is spoken of as "choking" respiration.

(4) *Wavy Respiration.*—A lack of evenness in the movement of inspiration or expiration, one part of the chest expanding or contracting before another, giving rise to a peculiar undulating or wavy motion of the chest walls, as if the ribs had lost their firmness, is seen in certain adynamic (typhoid) conditions, particularly pneumonia.

Dyspnœa.—Dyspnœa may be defined as difficult, laborious, or painful breathing. The definition implies in the majority of cases a subjective sense of breathlessness; in patients' parlance, "shortness of breath." The objective symptoms are rapid or laboured respiration and more or less cyanosis (*q. v.*), according to the cause and severity of the dyspnœa. The face wears an anxious expression; the pupils are dilated. The nostrils expand and contract, the mouth opens with each inspiration, the thorax and abdomen heave more or less violently, the skin is cold and wet, and if the dyspnœa is extreme the patient sits upright (orthopnœa) and leans upon the hands, thus supporting the shoulders so that the extraordinary muscles of respiration may work to better advantage. In dyspnœa any of the variations from the normal manner of breathing which have previously been considered may be present in varying number and intensity—e. g., the respiration may be rapid or slow, thoracic or abdominal, deep or shallow, etc.

Primarily, dyspnœa is due, with few exceptions, either to lack of oxygen, excess of carbon dioxide, or the presence in the blood of certain products of muscular activity (dyspnœa of exercise).

The diseased conditions which may be responsible for dyspnœa are in general: obstruction or obstructive disease of the nose, throat, larynx, trachea, and bronchi, causing hindrance to the entrance of air into the lungs; diseases of the lungs which diminish the available air space; diseases of the pleura hindering lung expansion; diseases of the heart which cause pulmonary stasis; pressure upon the diaphragm by fluid, gas or tumour in the abdomen; diminished amount of hæmoglobin in the blood, poisonous materials circulating in it, or an increase of its temperature; paralysis or spasm of the muscles of respiration; and pain in such localities as to interfere with respiration.

The most common causes of dyspnœa are debility or anæmia, cardiac, pulmonary, or renal disease. Therefore always examine the heart, lungs, blood, and urine. The dyspnœa most commonly seen is felt during both phases of the respiratory act. It is helpful to recognise certain clinical varieties of this symptom and their usual disease associations as follows:

(1) *Simple Accelerated Respiration.*—This usually occurs in fever, particularly if the patient is an infant or a neurotic adult. The breathing is shallow and a physical examination does not reveal lesions capable of causing dyspnœa.

(2) *Dyspnœa on Exertion.*—Shortness of breath observed only after physical exertion is characteristic of obesity, debility, anæmia; valvular defects, well, but not perfectly, compensated; moderately weak heart, disease of the blood vessels, severe bronchitis of the larger tubes, emphysema, and incipient tuberculosis or latent pleurisy with effusion.

(3) *Dyspnœa which is Constant.*—Short breathing, even while at rest, may be due to a severer grade of conditions mentioned, (2); or to laryngeal stenosis, fibrinous bronchitis, broncho-pneumonia, lobar pneumonia, fibroid or ulcerative phthisis, acute pleural or large pericardial effusion. Deep, but not difficult, breathing may occur toward the close of diabetes (diabetic dyspnœa).

(4) *Dyspnœa which is Paroxysmal.*—Sudden attacks of dyspnœa may be accounted for by acute indigestion, bronchial asthma, spasm of the laryngeal adductors (spasmodic croup), chronic nephritis (uræmic asthma), disease of heart (cardiac asthma), angina pectoris, or the pressure of a tumour or aneurism upon the pneumogastric.

(5) *Dyspnœa which is Inspiratory.*—When there is difficult entrance of air into the lungs, as evidenced by retraction or recession of the supraclavicular, intercostal and subcostal spaces, with a comparatively easy expiration, one may suspect foreign body in the larynx or trachea, spasm of the adductors (croup, laryngismus stridulus) or paralysis of the dilators of the glottis, œdema, inflammatory swelling of or membrane in the larynx, or a movable tumour over the glottis.

(6) *Dyspnœa which is Expiratory.*—Hindered expiration, proved by expiratory bulging and excessive action of the abdominal muscles, while inspiration is reasonably easy, is due most frequently to pulmonary emphysema. In bronchial asthma the dyspnœa is usually greater than in emphysema, the act of inspiration is short and gasping, while that of expiration is prolonged and wheezy, thus involving a reversal of the normal relative duration. Bronchitis with much viscid mucus, and a movable tumour below the glottis, so placed and attached as to swing up during expiration, are other causes.

(7) *Dyspnœa which is Subjective.*—There is a form of dyspnœa met with in nervous and hysterical women which appears to be purely psychic. The dyspnoic sensation is the subject of bitter complaint; indeed, there may be orthopnœa, but there is no blueness of the lips and finger nails, and a thorough search for cardiac, pulmonary, hæmic, renal, or other possible causal conditions will afford negative results.

Vocal Fremitus.—If a hand is laid upon the chest wall while the patient speaks with ordinary or more than ordinary loudness, a peculiar buzzing or vibrating sensation is perceived by the palpating fingers—the vocal fremitus. The vibrations originate in the vocal cords, and are conducted by the columns of air in the trachea and large and small bronchial tubes through the substance of the lungs to the chest wall. The intensity of the vocal fremitus depends upon two factors: first, the loudness and pitch of the voice; second, the varying conductivity and thickness of the media through which the vibrations must pass. To study the vocal fremitus the patient is told to articulate certain words—e. g., "one, two, three," "ninety-nine"—while one, preferably always the same, warmed hand is laid upon various parts of the thorax. Some experience with different voices and chests is requisite to be assured of variations from what may be considered normal for the individual under examination.

(1) **Marked or Increased Fremitus.**—If the patient has a loud, low-pitched, or harsh voice, especially if the chest walls are thin, the fremitus will be marked. It is consequently much more distinct in men than in women and children. It is normally more intense over the upper half of the right lung, partly because the right bronchus exceeds the left in diameter and therefore affords a larger vibration-conveying column of air, partly because the primary bronchus entering the right upper lobe is given off nearer the main bronchus and at a higher level than that entering the left upper lobe (CAREY).

If the lung substance is consolidated or infiltrated it conducts the sound vibrations with greater facility; consequently the vocal fremitus is increased in pneumonia, tuberculous lung, etc. If there is a solid tumour in the chest, lying between a large bronchus and the chest wall, and of sufficient size to leave little lung tissue between it and them, the vocal fremitus is increased over its site. Pulmonary cavities, if thin walled and near the surface of the chest, may afford a very distinct fremitus, the cavity acting as a resonator for the sound of the voice. If bands of adhesion stretch from lung to pleura the fremitus may be conducted and perceived, even if there is a considerable pleural effusion.

(2) **Diminished or Absent Fremitus.**—The vocal fremitus is slight in women, children, men with feeble, high-pitched voices, and stout persons. It is normally less on the left side than on the right. Absence of vocal fremitus, where it should exist, is significant of air or fluid in the pleural cavity. Air and fluid under these circumstances do not act as good conductors, and will hinder the vibration of the chest wall; or, as another explanation has it, the collapsed lung, having lost its elasticity, does not carry the vocal vibrations to the air or fluid. Moreover, a thickened pleura damps the vibrations. If a main bronchus is obstructed by a foreign body, a plug of mucus, or the pressure of a tumour, the vocal vibrations are not conducted to the corresponding lung and the fremitus is lacking. If a large bronchus leading to a consolidated area—e. g., in pneumonia—is obstructed, the solidified lung will not offer the customary increased fremitus.

Friction Fremitus.—If a fine creaking or rubbing vibration is felt, which is synchronous with the heart beat or the respiration, it is significant respectively of pericarditis or pleurisy.

Bronchial Fremitus.—A somewhat coarse fremitus, synchronous with the respiration, and often heard some inches away from the chest, is a palpable, large, moist or bubbling râle, due to the passage of air through a bronchus partly plugged with viscid mucus, or constricted by swelling of the mucosa; or the bubbling of air through fluid in a cavity. Bronchial (or rhonchal) fremitus is common in infants and children suffering from an ordinary bronchitis, and in asthma, as well as in the chronic bronchitis with bronchiectases of older persons.

IV. PERCUSSION OF THE LUNGS

With reference to the lungs, percussion is employed for four purposes: (1) To determine the boundaries of the lungs; (2) to ascertain whether they contain more or less than the normal amount of air; (3) to aid in determining the presence of cavities in the lungs, or (4) of air or fluid in the pleural cavities, or thickening of the pleura.

(A) Technic of Percussion of the Lungs

The standing or sitting position is preferable, unless, as frequently happens, the patient is ill in bed and greatly prostrated. With the patient facing the examiner, arms hanging easily at the side, face looking straight forward, head turned neither to the right nor the left, in order to avoid unequal tension of the muscles and fascia on either side, the front of the chest may be gone over. To percuss the sides of the chest, the arms should be raised and the hands clasped over the head. To examine the back, let the arms be folded and the patient lean somewhat forward. Before leaving the back, it is well to have the hands again placed upon the head, as this action swings the angles of the scapulæ outward, and, by uncovering, renders accessible a portion of lung in which there is not infrequently found the earliest evidence of tuberculous disease. In percussing the lungs it should be made an invariable rule to compare symmetrical points on each side; moreover, to compare interspace with interspace and rib with rib, never letting the pleximeter finger rest upon two ribs or a rib and an interspace at the same moment.

Beginning above the clavicle (Fig. 137), with a rather strong stroke, note how high above this bone the pulmonary resonance can be elicited—i. e., the height of the apices—and compare the various qualities of the sounds upon each side. Next percuss and contrast successively, with a stroke of moderate strength, the 1st, 2d, and 3d interspaces upon each side, along their length from sternal edge outward. Passing down the right mammillary line, the covered (deep) dulness of the liver becomes manifest by strong percussion at the 4th interspace or 5th rib, and continuing, with gentle percussion, the exposed (absolute or superficial) dulness of the same organ becomes evident at the 6th rib or interspace, thus indicating the usual location of the anterior portion of the lower margin of the lung in this line. In the left mammillary line the normal lung resonance is impaired at the 4th or 5th interspace by the exposed (superficial) cardiac dulness; and a little outside of this line and at about the same level strong percussion elicits a tympanitic quality from the underlying left end of the stomach, which mingles with the lung resonance. From this point downward very gentle percussion finally offers, at or just below the 6th rib, a pure tympanitic sound, indicating the usual position of the lower anterior border of the left lung. Any areas of dulness or hyperresonance which have been found on the front of the thorax may now be more carefully mapped out.

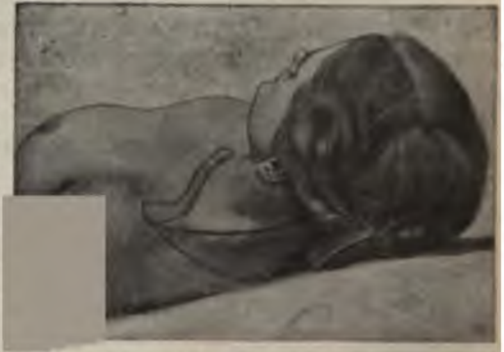


FIG. 137.—Showing the two triangular areas—one anterior, between clavicle and line of trapezius; the other posterior, between trapezius and line of spine of scapula—over which percussion and auscultation are to be performed in order to determine the condition of the apices of the lungs.

The lateral (axillary) regions of the thorax may now be examined, percussing from the axilla downward in the midaxillary line, finding, by gentle percussion, the last trace of pulmonary resonance—i. e., the lower lateral border of the lung—at the 8th rib, if it is in its normal position.

Percuss next the back of the thorax in the scapular line, from the suprascapular space downward. At the base, the liver on the right, and the kidney and spleen on the left, will modify the lung sound, but it may be traced by percussion strokes of moderate strength as far down as the 10th rib or space on both sides.

By implication from the foregoing suggestions it is readily seen that during the examination one endeavours to ascertain the boundaries of the lungs (topographical percussion), and that comparison is made, not only between the sounds from corresponding portions of the individual chest, but that each sound elicited is assigned to its proper place in the descriptive list of percussion sounds.

(B) The Results of Percussion in Normal Lungs

In the normal chest the pulmonary resonance is less clear (dulled or muffled) if its walls are thick (obesity), and is clearer and more resonant if the chest walls are thin or especially resilient, as in children. If the lungs are healthy, the thorax well formed, and the individual neither obese nor emaciated, the results of percussion in the various topographical areas are as follows (Fig. 138):

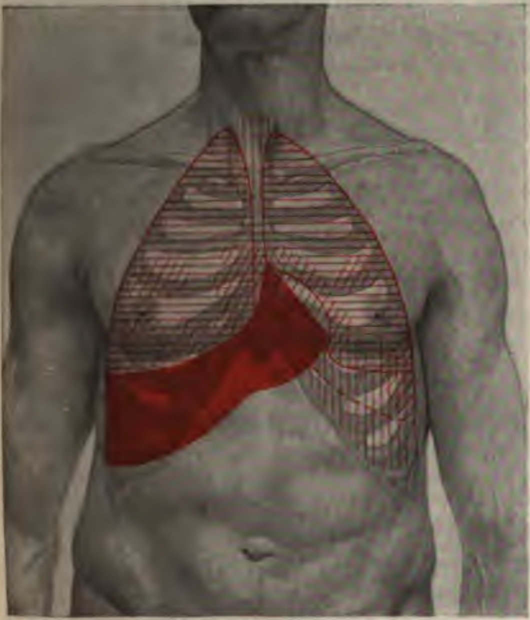


Fig. 138.—Showing the relative resonance of various portions of the anterior surface of the thorax. Horizontal lines = pulmonary resonance. Vertical lines = tympanitic resonance of trachea and stomach. Oblique lines = impaired resonance or moderate dulness due to mammary glands, liver, heart, and spleen. Solid shading = absolute dulness due to liver and heart.

Supraclavicular Spaces.—There is pulmonary resonance of a moderate character, becoming somewhat tympanitic if percussion is carried toward and over the trachea.

Infraclavicular Spaces.—Below the middle of the clavicle (in the mammillary line) there is typical well-marked pulmonary resonance. Percussing toward the median line, osteal and tympanitic resonance tends to be heard, owing to the presence of the sternum and the tracheobronchial air columns, and pulmonary resonance diminishes. From the mammillary line outward there is a slight diminution in the pulmonary quality. The pulmonary quality is slightly less marked in the right than in the left supraclavicular and infraclavicular areas.

Mammary Spaces.—In both mammary spaces the pectoral muscles and, in women, the mammary glands tend to muffle the sound of pulmonary resonance. In the lower portion of the right mammary region the presence of the liver causes partial dulness. In that portion of the left mammary region internal to the mammillary line, and at and below the fourth space, the heart dulness modifies, and, over the area of exposed cardiac dulness, replaces pulmonary resonance. At the lower portion of the left mammary space in the anterior axillary line the percussion sound becomes hyperresonant and, finally, purely tympanitic from the presence of the stomach.

Axillary Space.—The upper axillary spaces, down to the 6th rib on both sides, afford typical pulmonary resonance. The right inferior axillary space is less resonant because of the liver; the left inferior space is partly tympanitic from the stomach, partly dull from the spleen.

Suprascapular and Interscapular Spaces.—These give a moderate pulmonary resonance.

Scapular Space.—The overlying scapula and its muscles, by muffling the sound, cause these spaces to be the least resonant of the pulmonary areas.

Infrascapular Space.—Compared to the upper anterior portions of the chest, the infrascapular regions have only a fair degree of pulmonary resonance, but they are the least muffled and most resonant of the posterior areas.

(C) The Results of Percussion in Disease of the Lungs and Pleuræ, or of Neighbouring Organs

As a consequence of some morbid condition of the respiratory apparatus, or of some contiguous part or organ, one may find the boundaries of the lungs displaced, or an abnormal alteration of the percussion sounds in a given point or area.

(*a*) **Changes in the Position of the Borders of the Lungs.**—(1) *Apices.*—Normally the pulmonary resonance extends 1½ to 2 inches above the clavicles, the right apex being sometimes slightly higher than the left. If one apex is found to be distinctly lower than the other—i. e., shrunken—it is suggestive of past or present tuberculous disease of the lung, a contracting adhesive pleurisy or collapse of the lung. If both apices stand unusually high, and especially if the supraclavicular spaces bulge during inspiration, the lungs are emphysematous. Temporary expansion is present during attacks of bronchial asthma.

(2) *Anterior Borders.*—As the right anterior border lies for its whole length behind the sternum, its position can not be determined, the osteal resonance of the firm bone preventing. The same statement applies to that portion of the left anterior border extending from the apex down to the 4th costal cartilage. At this point it curves outward from under cover of the sternum and downward to the 6th rib, becoming accessible to percussion and forming the left

border of the exposed (superficial) cardiac dulness. While a considerable increase in the exposed cardiac dulness may indicate an enlarged heart or a pericardial effusion displacing the left anterior border outward, it is in the majority of cases due to retraction of the lung from tuberculous or fibroid disease, the whole length of the anterior border sometimes withdrawing to the left of the sternum, or to a pleural effusion with resulting collapse of the lung. On the other hand, if the lower part of the anterior border covers the usually exposed portion of the heart, it is indicative of emphysema or, temporarily, of bronchial asthma.

(3) *Lower Borders.*—In drawing inferences from the position of the lower borders of the lung, allowance must be made for age and respiratory and postural displacement as follows:

In *old age* these borders lie 1 rib below, in *infancy* 1 rib above, the position which is normal for a healthy person of intermediate age. In *quiet respiration* the edges of the lung advance into the anterior and inferior complementary pleura about ⅖ inch. If percussion is made in the mammillary line at the end of inspiration and again at the end of full expiration (*forced respiration*), the excursion of the lower border will be found to be 1¼ to 1½ inch. In a person lying upon the *back*, the lung borders in the mammillary line are ¾ inch lower than when erect. If lying upon the *side*, the border of the lung upon the uppermost side may advance downward as much as 4 inches in the midaxillary line.

If the lower borders of the lung, front, side, and back, are found to be lower than normal, it is significant of emphysema, in which case it may be that pulmonary resonance is elicited as far down as the 9th rib in the right mammillary line, or, temporarily, bronchial asthma. If the lower borders are higher than normal, it may be due to phthisical shrinking, collapse, a distended abdomen pushing the diaphragm and lungs upward, or to paralysis of the diaphragm. The lung may, of course, be pushed up by air or fluid in the pleural cavities, but in such cases pulmonary resonance is absent, being replaced respectively either by a tympanitic or a dull percussion sound.

If the normal respiratory displacement is sought for and not found, its absence may be due to extensive pleuritic adhesions. The movement of the lung borders during respiration in emphysema is slight, as the lungs are already permanently expanded beyond their natural limits.

(*b*) **Decreased Resonance.**—The percussion sound over the lung becomes less resonant in proportion to the diminution in the amount of air underlying the part of the chest percussed. Consequently, exudation into the air cells, collapse or consolidation of the lung, the presence of fluid in the pleural cavities, or the existence of a solid tumour renders the percussion sound over such areas more or less dull (Fig. 140*a*).

Consolidations of the lung are found in pneumonia, phthisis, hemorrhagic infarcts, gangrene, abscess, and tumours. In order to cause appreciable dulness the consolidation must be at least 1½ inch in diameter, and lie just beneath the chest wall. More remote airless portions must be larger and lie not deeper than 2½ to 3 inches, the distance to which the percussion stroke penetrates below the under surface of the pleximeter finger, and require strong percussion to elicit their modifying effect, which, indeed, may be simply a heightening of pitch. Fluid in the pleura (serum, pus, blood), in order to cause flatness, must amount at least to 12½ oz.

Variations in the degree of dulness are recognised by appropriate terms (page 307). The sense of increased resistance, which is very obvious to the skilled finger, should not be forgotten. It is most marked over consolidations, fluid, thickened pleura, high grades of emphysema, and pneumothorax when the air is under great tension.

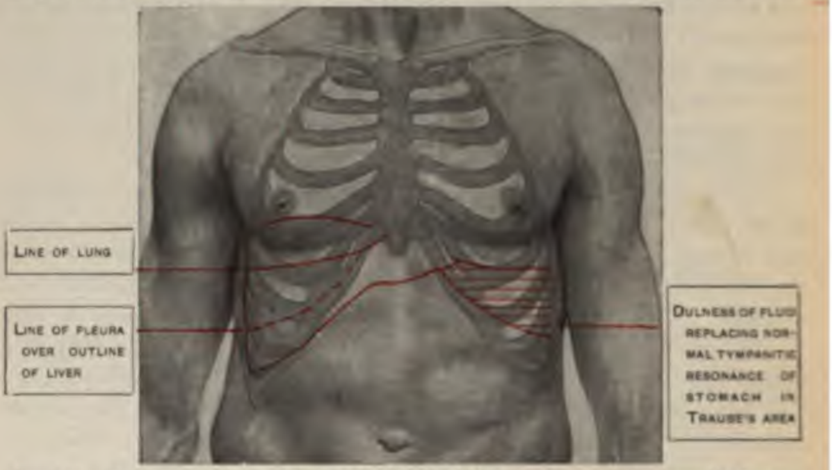

LINE OF LUNG
LINE OF PLEURA OVER OUTLINE OF LIVER
DULNESS OF FLUID REPLACING NORMAL TYMPANITIC RESONANCE OF STOMACH IN TRAUBE'S AREA

FIG. 139.—Showing the dulness due to fluid in the left complementary (reflected) pleura.

(1) Dulness or heightened pitch over *one apex* (sometimes both), with normal resonance elsewhere, is usually suggestive of tuberculosis, but may in somewhat rare cases be due to pneumonia, gangrene, or new growth. Slight impairment at both apices, disappearing after a few forced inspirations, is found not infrequently in persons of sedentary habits who are not accustomed to deep breathing.

(2) Dulness over the *lower lobes posteriorly* may be significant of pneumonia, pleurisy with effusion, œdema, atelectasis, or the hypo-

static congestion which is found in patients with weak heart, as in exhausting fevers, who lie persistently in the dorsal posture. Much less often it is due to infarcts, abscess, gangrene, tumour, or basic tuberculosis, the latter usually sequent to disease of the upper lobes.

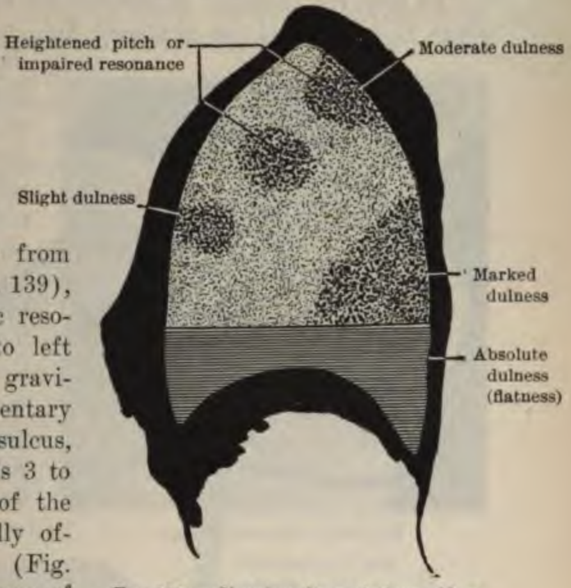

Heightened pitch or impaired resonance
Moderate dulness
Slight dulness
Marked dulness
Absolute dulness (flatness)

Fig. 140*a*.—Showing the variations of lessened resonance due to the presence of consolidations in the lung or fluid in the pleural cavity.

(3) A band of dulness or flatness in the *left anterior axillary line* from the 6th to the 8th rib (Fig. 139), where normally tympanitic resonance is found, is due to left pleural effusion, the fluid gravitating into the complementary (or reflected) pleural sulcus, which at this point extends 3 to 4 inches below the edge of the lung. This region normally offers a tympanitic sound (Fig. 138) because of the presence of the cardiac end of the stomach, and is the so-called Traube's area or "half-moon-shaped space" (Fig. 166). It is bounded above and laterally by the contiguous borders of the liver, lungs, and spleen.

(4) A "wooden" percussion sound, with an unusual sense of resistance, points toward a marked fibroid change in the lungs, such as occurs in chronic fibroid phthisis or chronic interstitial pneumonia. It is also said to be present over solid lung which is overlaid by a thin layer of relaxed pulmonary tissue.

(5) Dulness in the *left suprascapular,* and especially in the *left interscapular,* space may be due to an aneurism of the descending aorta—a fact to be remembered. Over one or both interscapular spaces lack of resonance may indicate enlargement of the bronchial glands (so also with dulness instead of tympanicity over the lower cervical vertebræ) or collapse of the lung.

(6) Dulness at *either base,* the upper line of which shifts with a change in the position of the patient, may occur in pleurisy with effusion, but is most characteristic of pneumohydrothorax, the fluid gravitating more readily in the latter condition.

(7) *Grocco's* (*Paravertebral*) *Triangle.*—Where there is fluid in

one pleural cavity, a triangular area of dulness may be found o opposite side of the spine (Fig. 140*b*).

To map the triangle: Determine the upper limit of flatness above it dulness, of the supposed fluid on the affected side o thorax. Ascertain the limit of pulmonary reso on the unaffected side. ginning well up, percus spinous processes down and mark the point at relative dulness is met, locating the upper end vertical side of the tri Then percuss over the he lung in horizontal lines without toward the spir order to obtain the ol side of the triangle. base of the triangle coir with the lower limit of monary resonance alread termined. The breath- and voice-sounds and vocal fremitus w the triangle are lessened, but not as much as over the affected The base varies from ¾ to 2¾ inches in length. The vertical s usually nearly double the length of the base line.

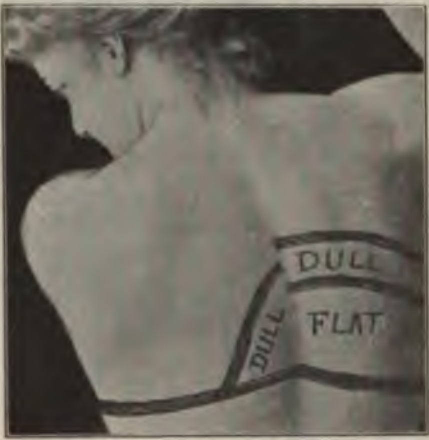


Fig. 140*b*.—Grocco's triangle.

This sign is practically constant in all cases of pleural eff unless the fluid is in very small quantity. The presence of th angle in pleurisy with effusion is reasonably explained by the sure of the contained fluid displacing the mediastinum towar unaffected side, perhaps compressing the sound lung, and as deadening the vertebral resonance. The greater the effusion larger is the triangle.

This sign is very reliable if further tested (Ewart). Thus suspected right effusion and left triangle turn the patient upo right side. Unless the effusion is extremely large the triangl lessen in size, or, with small effusions, disappears. Conversely, on the left side, the dull triangle will not become smaller, and increase in size. Such changes, if observed, prove definitely th dulness is due to free fluid.

Grocco's triangle is not pathognomonic of pleural effusion cause it may be found with subphrenic abscess, large abdominal plasms, large ascites (double triangle), and (left side) the pre uterus (Smithies). Triangles due to nonpleural causes are fo by the upthrust of the fluid or tumour against the diaphragm pushing the lung away from the spine.

(*c*) **Increased Resonance.**—Taking again pulmonary resonance as the standard, the percussion note may depart from it in the direction of greater resonance. The presence of a tympanitic sound usually but by no means always implies a greater amount of air under the point at which percussion is made. The general conditions which are responsible for this sound and its modifications are (Fig. 141):

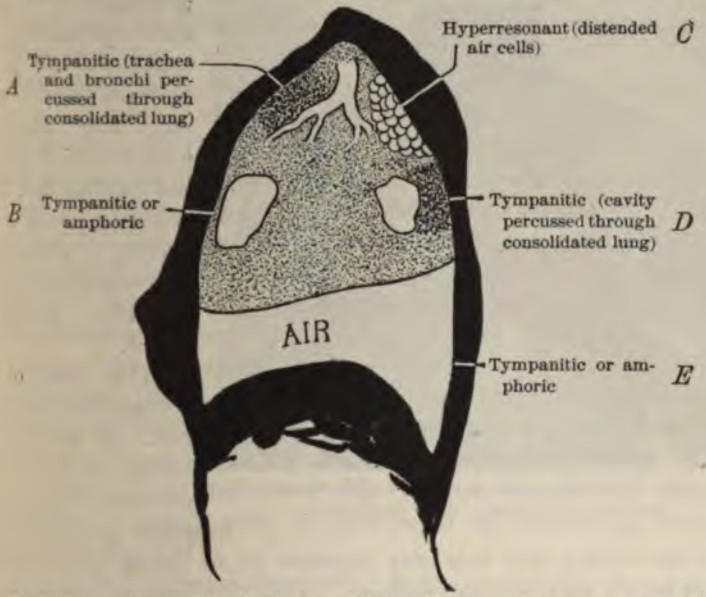

A
Tympanitic (trachea and bronchi percussed through consolidated lung)
Hyperresonant (distended air cells)
C
B
Tympanitic or amphoric
Tympanitic (cavity percussed through consolidated lung)
D
AIR
Tympanitic or amphoric
E

Fig. 141.—Diagram showing the physical conditions which cause hyperresonance and tympanitic or amphoric percussion sounds.

(1) Distention of the air cells (increased amount of air), as in emphysema (*C*).

(2) The presence of air-containing cavities in the lung, as in phthisis (*B* and *D*). If the cavity becomes filled with fluid the tympanitic quality disappears.

(3) The presence of air in the pleural cavity (*E*), as in pneumothorax, if not under too great tension.

(4) The presence of a shaft of consolidated lung leading from the trachea and main and larger bronchi to the surface, through which the percussion stroke is conducted directly to the column of air in these large tubes, practically normal cavities (*A*), or when percussion is made immediately over the trachea and main bronchi.

(5) The presence of a large pleural effusion or pulmonary consoli-

dation not infrequently causes a tympanitic percussion sound in that portion of the lung which lies above the fluid or solid exudate (Fig. 142). The explanation of this fact, which is not altogether satisfactory, infers that the part of the lung from which tympanitic resonance may be elicited is in a state of relaxation or atony, the walls of the alveoli—as their tonus or tension is in abeyance—permitting the contained air to vibrate as if in a loose sac, the numerous cells in which the air lies having lost their ordinary power of imparting a characteristic pulmonary quality to the percussion sound.

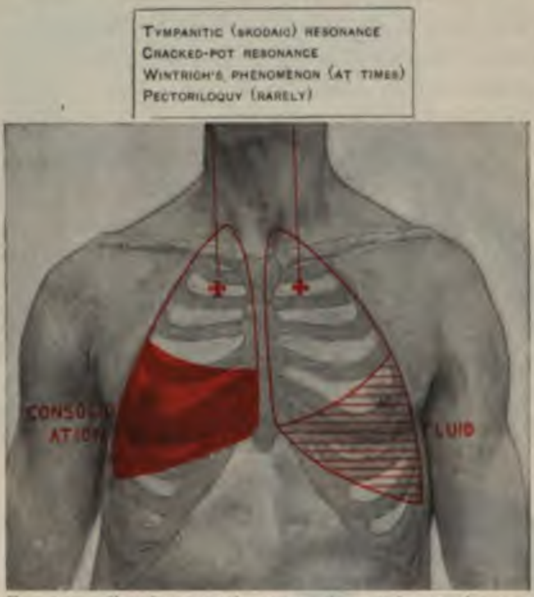

TYMPANITIC (SKODAIC) RESONANCE
CRACKED-POT RESONANCE
WINTRICH'S PHENOMENON (AT TIMES)
PECTORILOQUY (RARELY)
CONSO
ATIO
LUID

FIG. 142.—Showing certain percussion and auscultatory findings above consolidations or effusions.

The *pitch* of the tympanitic sound varies. If the air in a cavity is under considerable tension the pitch becomes higher, and the volume and intensity decrease to an extent (dull tympanicity) which may suggest dulness. This fact may be demonstrated by percussing the cheeks while inflated under varying pressure by buccal contraction. If it is a cavity communicating with the air which furnishes the tympanitic sound, the wider the channel of communication the higher the pitch.

For the *terminology* of the variations of increased resonance see page 307. There are certain peculiar percussion sounds which, as their resonance exceeds that of the normal lung, are classed as varieties of tympanitic sound—namely, *amphoric*, *cracked-pot*, and *coin percussion*. There are other minor alterations in the extraresonant percussion sounds which will be noted.

The **diagnostic significance** of increased resonance may now be pointed out as follows:

(1) A hyperresonant, almost tympanitic, note on *both sides* of the chest is indicative of emphysema, which, if extreme, may afford a dull tympany on account of the tension of the chest walls.

(2) Tympanicity over the greater part of *one side* of the chest may be due to compensatory emphysema of one lung, the other having been disabled by disease, or to pneumothorax. If the air tension in the latter disease is very great, the tympany may be so raised in pitch as to be mistaken for dulness.

(3) If there is great increase of cardiac dulness and a tympanitic quality of sound is discovered above and to the left in front, and posteriorly on the left side as well, it is probably due to an unusually large pericardial effusion displacing the lung. In a case under my care, which presented this combination of signs, the pericardium contained 90 oz. of serum.

(4) Dulness at *one or both bases*, with hyperresonance ("skodaic") over the upper portion of the chest, usually most marked in the infraclavicular space (Fig. 142), may be caused by pleurisy with effusion, basic pneumonia (1st and 3d stages of lobar pneumonia particularly), large pulmonary infarctions, and pulmonary œdema.

(5) If dulness is discovered over *one or both apices*, and a tympanitic note is found in the 1st and 2d interspaces close to the edge of the sternum, it is probably due to the conduction of the tracheobronchial percussion sound by consolidated lung (*A*, Fig. 141) in apical or upper lobe pneumonia or tuberculosis, rarely tumour.

(6) A localized tympanitic sound may be due to a cavity (phthisis, bronchiectasis, pulmonary actinomycosis, gangrene, abscess). In order to produce a hyperresonant sound the cavity must be at least the size of a walnut (1 × 1¼ inch), and either lie just beneath the chest wall or be put in touch with the pleximeter by intervening solid lung (*B* and *D*, Fig. 141).

(7) It must be reiterated that, owing to the presence of the stomach, tympanitic resonance of varying perfection, in the left anterior axillary line, from the 5th rib downward, is a normal finding. If the stomach is greatly distended with gas the tympanicity may begin as high as the 4th or even the 3d interspace.

(8) Tympany over the usual area of exposed liver dulness, from the 6th rib downward in the axillary line, may occur in great gaseous distention of the intestines as well as from the presence of air in the peritoneal cavity, so that the transition in this case is from pulmonary resonance to tympanitic resonance, instead of the normal dull sound.

(9) *Amphoric resonance* is a tympanitic sound with an added metallic clanging or echoing quality. It is rather higher in pitch but of longer duration than ordinary tympany. It may be imitated by percussing an empty vessel. With reference to its diagnostic significance, it should be remembered that it is due either to pneu-

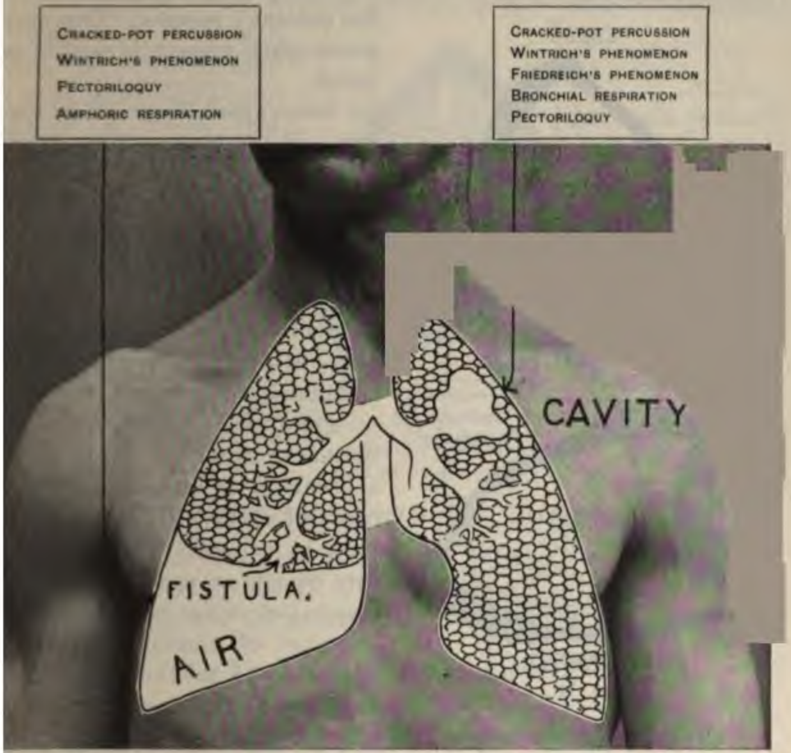
mothorax, provided that the air is under a certain degree of tension (not too great), or to a cavity (*B* and *E*, Fig. 141). If present over a cavity, it may be inferred that the latter is at least 2½ inches in diameter, close under the parietes, and with smooth, firm walls. If amphoric resonance is suspected, a pleximeter and percussion hammer may be used, these instruments tending to develop this peculiar quality of sound better than finger percussion alone, especially if simultaneous auscultation is made. If due to pneumothorax and not to a cavity, the discrimination may be made by noting the much greater extent over which it is heard in the former.

(10) The *cracked-pot sound* is tympanitic, but has a superadded peculiar hissing, chinking quality. The hissing component of the sound resembles and, indeed, is sometimes due to an outrush of air from under the percussed surface through the upper air passages. The chinking element resembles the sound heard on tapping cracked metal vessel. The sound as a whole may be roughly imitated by clasping the hands loosely together and striking them over the knee. If the presence of this sound is suspected the percussion stroke should be sharp and rather strong, and the plexor finger, after its descent, allowed to rest upon the pleximeter finger, thus adding a species of push to the stroke. The patient's mouth should be open and percussion made during expiration.

The diagnostic value of this sign is somewhat varied. It may be obtained from the chest of a healthy infant (especially while crying) and in some normal adults. If occurring (Fig. 143) over one apex in a thin chest wall, it may be due to a cavity having a communication with a bronchus; if over the lower half of one side of the chest, to pneumothorax opening by a fistula into a bronchus, or communicating with the external air by an opening through the chest wall. It may also be elicited from the lung above pleural effusions, and in the congestive stage of pneumonia (Fig. 142).

(11) There are certain alterations of pitch—sound mutation or change—found in hyperresonant percussion notes over cavities, which are detected by and partly dependent upon special postural or other disposition of the patient. These changes are:

Wintrich's Phenomenon.—If a tympanitic note becomes loude and raised in pitch when the patient opens his mouth, protrudes hi tongue, and breathes quietly, it is suggestive of a cavity having fr communication with a bronchus. It can be simulated by percussi over the trachea while opening and closing the mouth. It may obtained in cases of pneumothorax with a large fistulous open into a bronchus. In both of these instances (Fig. 143) the per sion vibrations over the cavity are directly transmitted to the nn of air in the bronchi and trachea, and the opening of the mouth ows the pharynx to act as a resonator, thus causing the change of ınd. It may occur over the upper lobes, if the latter are consoli- ted by inflammation or compressed by pleural effusion (Fig. 142).



. 143.—Showing certain percussion and auscultatory findings over cavity or pneumothorax, provided each communicates freely with a bronchus.

A similar change of sound, normally absent, will occur when per- ssing over the *upper sternum*, provided that the tissues lying be- een the manubrium and the trachea are of sufficiently increased nsistence to conduct the percussion vibrations to the tracheal air lumn (HOOVER). Thus, in lymphosarcoma of the mediastinum, neurism of the ascending arch of the aorta, and in some cases of ericardial effusion, this sign, when elicited by percussion over the nanubrium, has been of much value in diagnosis, as it is not present in cavity or consolidation of the upper lobes, or in pleural effusion. Certain precautions are necessary. The chin should be elevated, the mouth opened, and the tongue protruded. As this sign may be found at the end of inspiration only, the patient should be instructed to inspire deeply, and continue the effort so as to preve the instinctive closure of the glottis, which usually takes place whe the breath is held at the close of inspiration. The manubriu should be gently percussed, and the examiner's ear placed close t the patient's mouth. The metalli sound when present is readily recognised.

Interrupted Wintrich's Phenomenon.—If the change just described is present while the patient is in one position—e. g., recumbent—and disappears in another posture—e. g., erect—it is rare but reliable evidence of a fluid-containing cavity, the secretion in one position occluding, in another leaving open, the communicating bronchus (Fig. 144).

Friedreich's Phenomenon.—The resonance over a cavity opening into a bronchus is higher in pitch during and at the end of inspiration than in expiration. This respiratory change is probably due to the inspiratory widening of the glottis (channel of communication), perhaps also to the increased tension of the walls of the cavity.

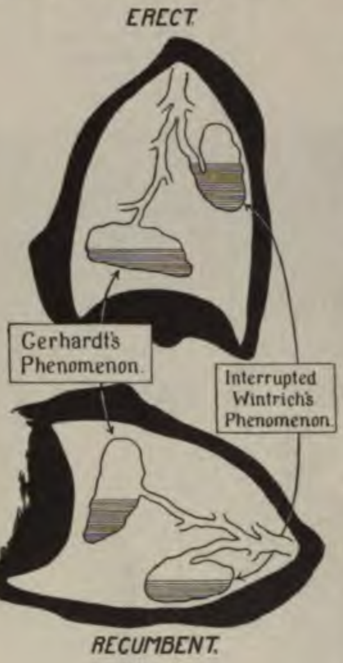


Fig. 144.

Gerhardt's Phenomenon.—If the pitch of the percussion sound over a cavity varies with change of position, it is indicative of a partly filled vomica, one diameter of which is considerably longer than the others. It is a very infrequent finding, but is good proof of a cavity. The change in pitch is due to the alteration in the shape of the air-containing part of the cavity caused by the mobility of the fluid. When the long diameter of the cavity is horizontal the pitch is lowest (Fig. 144).

Biermer's Phenomenon.—In pneumohydrothorax the percussion note is lower when the patient is recumbent, as in the horizontal position the fluid gravitates upon the posterior chest walls, thus increasing the long diameter of the air-filled portion of the pleural cavity. The explanation of the change in pitch is similar to that of the preceding (Gerhardt's) phenomenon.

(*d*) **Coin Percussion.**—This is employed where pneumothorax is suspected. The mouthpiece of the stethoscope is placed upon the back of the chest and an assistant percusses the front of the chest, using two coins (25 or 50 cent pieces preferably), one as a pleximeter, the edge of the other as a plessor (Fig. 145). If there is air in the pleural cavity, a sound (*bruit d'airain*) variously described as metallic and echoing, a distant soft musical chiming, as of a bell (bell tympany) or a far-off ring of a hammer on anvil, is heard—a most characteristic sign.

In lieu of coin percussion the chest may be "flicked" with the finger. The fillip is heard through a normal chest as a dull thud; through a pneumothorax as a ringing or chiming sound.

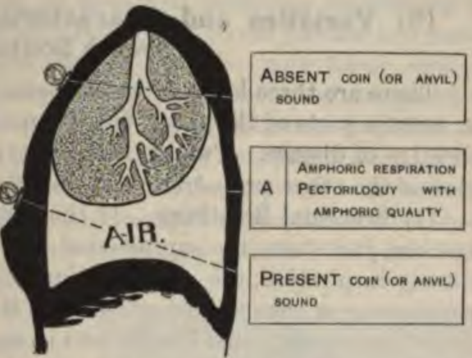

ABSENT COIN (OR ANVIL) SOUND
A
AMPHORIC RESPIRATION PECTORILOQUY WITH AMPHORIC QUALITY
AIR.
PRESENT COIN (OR ANVIL) SOUND

FIG. 145.—Showing the utility of coin percussion, also amphoric respiration and pectoriloquy over an open pneumothorax.

V. AUSCULTATION OF LUNGS

Auscultation of the lungs has for its objects the determination of the character of the breath sounds, the presence or absence of adventitious sounds, and the amount and character of vocal resonance.

(A) Technic of Auscultation

Certain points with reference to auscultation of the lungs in particular may be stated here.

The patient should, if practicable, be examined in the sitting posture. If too ill for this, he may be gently turned from one side to the other; or a flattened, disklike chest piece (SMITH) may be used for the back. In diseases of the lung which confine the patient to bed and require more or less frequent examinations of the back (e. g., pneumonia), it is a great convenience to have the nightdress put on wrong side forward, or slit down the back.

In the majority of cases it is necessary to direct the patient how to breathe, as it is of great importance that the respirations should be deep, regular, quiet, and not too rapid, so far as such qualities can be voluntarily secured. If the respirations are shallow, valuable evidence, such as distant bronchial breathing, fine râles, etc., may be overlooked. If irregular, noisy, and rapid, it is difficult to make certain fine discriminations which are desirable. The desired end is most conveniently accomplished by a personal demonstration on the part of the examiner. It is also desirable, when possible, to cause a temporary cessation of moaning, grunting, or noises in the pharynx or nares; or, if this be impracticable, to guard against mistaking such sounds for those of intrathoracic origin.

(B) Varieties and Characteristics of the Normal Breath Sounds

There are three kinds of breath sounds which are heard normally in certain parts of the chest, but if found over other portions are indicative of disease. Two of these are types, the third is a combination of the other two—*bronchial*, *vesicular*, and *broncho-vesicular*.

(1) **Bronchial Breathing.**—If the stethoscope is placed over the trachea, just above the suprasternal notch, two sounds are heard, one during inspiration, the other during expiration, separated by a distinct pause or silence, just previous to the end of inspiration. They are of practically equal length, and if any difference exists it is usually that the expiratory element is the longer. The quality of both sounds is that which is descriptively termed blowing, tubular, hollow, or bronchial; the pitch of both is higher than that of vesicular respiration. The expiratory element is more intense and frequently of higher pitch than the inspiratory sound.

This breath sound is caused by the air, which, when passing through the chink of the glottis, is thrown into rotary eddies, and imparts its motion to the air columns in the trachea and bronchi. It may be imitated by placing the mouth and tongue in position to sound the soft German *ch*, and expiring slowly.

(2) **Vesicular Breathing.**—If one listens to the breathing over a portion of the lung which lies some distance away from the trachea and larger bronchi—e. g., in the axillary or infrascapular spaces—a sound will be heard, the character of which is variously described as soft, breezy, sighing, or resembling the rustling of leaves in a gentle wind—vesicular respiration. This sound is audible during the whole of inspiration, and is immediately followed, without an interval of silence unless the breath is held, by a short expiratory sound. The inspiratory element is moderately intense, low in pitch, possesses the true breezy, vesicular quality, and is 3 times the length of the expiratory element; while the expiration, which may be inaudible, is less intense, somewhat lower in pitch, has a slight blowing as well as rustling quality, and is only one third as long as the sound of inspiration. To avoid confusion it may be recalled that the *movements* of inspiration and expiration are to each other in point of duration as 5 to 6 respectively, while the *sounds* of (vesicular) respiration stand in the ratio of 3 to 1. Vesicular breathing may be imitated by placing

the lips and teeth in position to articulate the letter *f*, and then inspiring slowly and steadily.

The exact mode of origin of the vesicular sound is still somewhat uncertain. By one hypothesis it is conceived to arise from the passage of air into and out of the infundibuli and alveoli; by the other it is considered to be the sound of bronchial breathing originating in the trachea and larger bronchi, transmitted to the surface and modified by passing through the spongy vesicular lung tissue, certain acoustic elements of the sound having been absorbed or dissipated in transit. Without question the latter hypothesis best explains the modifications of the breath sounds which are heard in disease. Thus bronchial breathing is heard over a consolidated portion of the lung, because the mitigating effect of the spongy lung tissue is abolished, and the tracheo-bronchial breath sounds are transmitted with little or no restraint through the well-conducting solid lung. While the major portion of vesicular breathing is unquestionably a modification of the tracheo-bronchial sound, it is possible that a minor portion may be conceded to arise from the entrance and exit of air into and from the terminal bronchioles and alveoli.

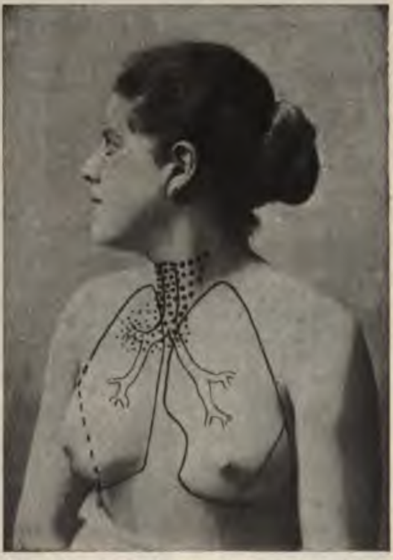


FIG. 146.—Dotted area shows where bronchial (larger dots) and broncho-vesicular (smaller dots) breathing normally exist. Note higher origin of bronchus to right upper lobe compared with that to left upper lobe. Shows also line of demarcation between upper and lower lobes of left lung anteriorly (see page 412).

(3) **Broncho-vesicular Breathing.**—This is a form of breathing which is neither distinctly bronchial nor distinctly vesicular in its characteristics, and is therefore sometimes spoken of as "indeterminate." It may be heard in the healthy chest over the lower portion of the manubrium, and over the interscapular regions at the level of the 3d dorsal vertebra—i. e., over portions of the chest where the larger bronchi are within auscultating distance, but a certain, not great, thickness of spongy lung intervenes between the ear and the main bronchi, thus affording a breath sound with commingled bronchial and vesicular characters.

It is necessary not only to become thoroughly familiar with these three varieties of breathing, and the special characters which serve to differentiate one from the other, but to know as well in what parts of the chest they may normally be heard. In many cases of pulmonary disease the principal and important physical sign consists in finding one kind of breath sound in a place where another variety should normally exist—e. g., bronchial, instead of vesicular, respiration over an infraclavicular space. It should be remembered, therefore, that in the normal individual vesicular respiration should be found in all parts of the chest, except over and in the close neighbourhood of the trachea and the main and larger bronchial tubes.

Consequently (see Fig. 146), *anteriorly*, bronchial breathing is found in the anterior (and antero-lateral) portions of the neck, from larynx down to the suprasternal notch and over the upper portion of the manubrium. Broncho-vesicular respiration is normal over the lower portion of the manubrium, and over the right and left sternal edges at the same level. The breathing in the right infraclavicular space is normally somewhat harsher and the expiration more prolonged than in the corresponding left space, because of the greater size and higher origin of the large bronchus going to the right upper lobe. *Posteriorly* (Fig. 147), bronchial respiration exists over the lower two or three cervical vertebræ; broncho-vesicular breathing below the vertebra prominens as far down as the 3d or 4th dorsal, and for a short distance to right and left of the spine at this level, more marked on the right side.

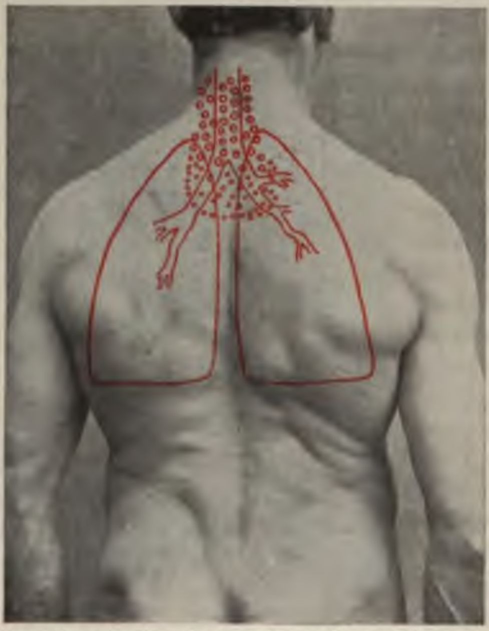


FIG. 147.—Showing the site of normal bronchial (large dots) and broncho-vesicular (small dots) respiration posteriorly.

The vesicular breathing over the remainder of the chest does not vary in quality, but does vary in intensity, according to the thickness of the chest wall (pectoral muscles, mammary gland, scapula); and the amount of lung substance underlying the point of auscultation (small amount at apices and over the thin lower, especially anterior, borders).

(C) The Breath Sounds in Disease

In all cases the examiner should carefully note at each point the *relative length* of inspiration and expiration after having distinguished them; and the *character*, whether soft and vesicular, harsh, blowing, or uncertain, both of inspiration and expiration (Fig. 148).

(*a*) **Bronchial Breathing.**—In the majority of cases bronchial breathing signifies consolidation (exudate, compression) of the lung, the solidified tissue acting as an excellent transmitter of the breath sounds (*G*, Fig. 148); in other cases a large cavity having free communication with a bronchus is responsible (*A* and *B*, Fig. 148).

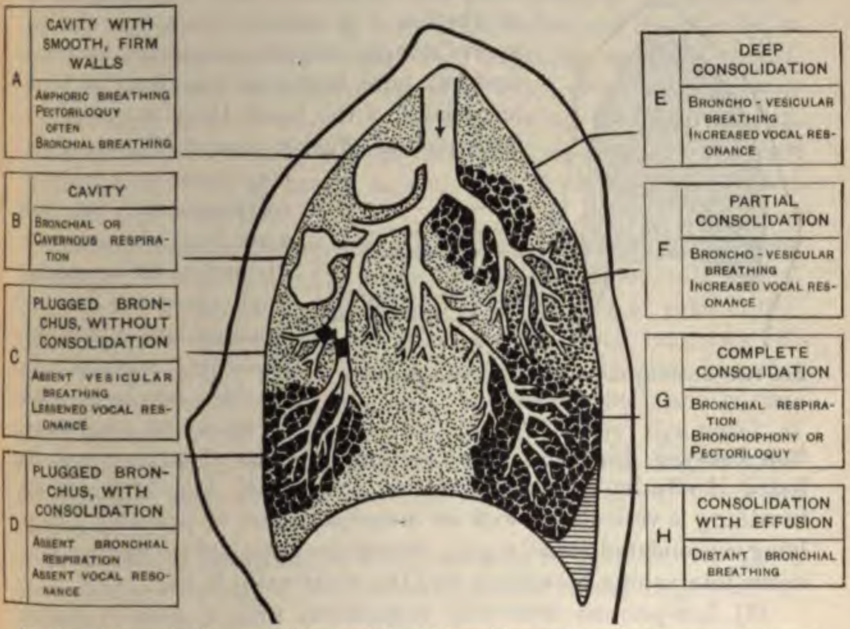

A
CAVITY WITH SMOOTH, FIRM WALLS
AMPHORIC BREATHING
PECTORILOQUY OFTEN
BRONCHIAL BREATHING
B
CAVITY
BRONCHIAL OR CAVERNOUS RESPIRATION
C
PLUGGED BRONCHUS, WITHOUT CONSOLIDATION
ABSENT VESICULAR BREATHING
LESSENED VOCAL RESONANCE
D
PLUGGED BRONCHUS, WITH CONSOLIDATION
ABSENT BRONCHIAL RESPIRATION
ABSENT VOCAL RESONANCE
E
DEEP CONSOLIDATION
BRONCHO-VESICULAR BREATHING
INCREASED VOCAL RESONANCE
F
PARTIAL CONSOLIDATION
BRONCHO-VESICULAR BREATHING
INCREASED VOCAL RESONANCE
G
COMPLETE CONSOLIDATION
BRONCHIAL RESPIRATION
BRONCHOPHONY OR PECTORILOQUY
H
CONSOLIDATION WITH EFFUSION
DISTANT BRONCHIAL BREATHING

FIG. 148.—Schematic diagram of the varieties of breathing and vocal resonance in disease.

Bronchial respiration in diseased conditions varies in pitch and to some extent in quality. If the sound is transmitted directly from the larger bronchi by consolidation or cavities it is low pitched, and may have the qualities known as cavernous or amphoric. On the other hand, if it arises from consolidation around the medium and smaller tubes, it is high pitched and whiffing, as in pneumonia involving the bases of the lungs. A practical point to be remembered is that bronchial breathing may be present and yet not heard, unless the patient takes a full and deep breath, a precaution which should never be omitted. The clinical varieties and significance of this form of breathing are as follows:

(1) If heard at the *base* of the lung, it signifies most commonly pneumonia, exceptionally tuberculosis; at the *apex*, tuberculosis, or (above the level of the fluid) compression of the lung by pleural effusion, less frequently pneumonia. Much more rarely it results from gangrene, beginning abscess, a septic or non-septic infarction, or compression of the lung by aneurism or tumour.

(2) In certain cases *distant* bronchial breathing is heard—i. e. the sound does not appear to emanate from the surface of the chest. In such a case one must endeavour further to determine whether it is a pure but far-away bronchial respiration, or whether it is mixed with a vesicular element (broncho-vesicular respiration). If distant but pure bronchial breathing is heard (especially at the base), there is probably a large pleural effusion present (Fig. 149), and corroborative evidence (displacement of the apex beat, characteristic history) must be sought. Such cases are not uncommon, and exploratory puncture, especially in children, may be required to make the discrimination between fluid and consolidation. Similar breathing may be heard if effusion and consolidation coexist (*H*, Fig. 148). If the breathing is vesicular as well as bronchial, there is probably a deep-lying consolidated area—e. g., a central pneumonia, with spongy lung tissue intervening between it and the chest wall (*E*, Fig. 148).

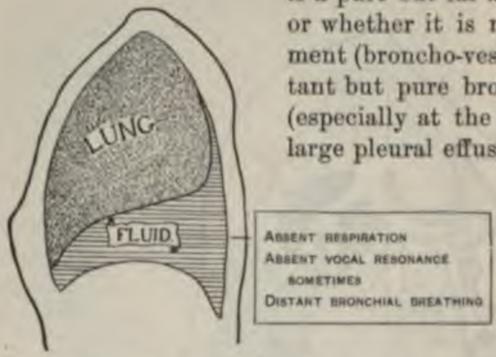


FIG. 149.—Showing the results of auscultation over a pleural effusion.

(3) Low-pitched bronchial respiration, with a notably hollow quality, constitutes *amphoric* breathing. It may be imitated by blowing partly into, partly across, the mouth of an empty bottle. Not infrequently cavernous is mistaken for amphoric breathing. The latter can be said to exist only when the peculiar musical, hollow intonation is present.

Amphoric respiration is never found in the normal chest, and when discovered indicates one of two things: a superficial cavity with smooth, firm walls, communicating with a bronchus (*A*, Fig.

148); or an open pneumothorax—i. e., one in which there is a large patent bronchial fistula (Fig. 143, also *A*, Fig. 145). The pitch of this variety of breathing varies with the size of the cavity.

(5) The absence of bronchial breathing, when it might reasonably be expected, or its rather abrupt disappearance when previously found, may be due to the plugging of a large bronchus leading to the consolidated area, thus preventing the transmission of the sound from the trachea, and coughing may make it manifest (*D*, Fig. 148). The filling up of a cavity with secretion may abolish cavernous or amphoric breathing, which will return when the fluid is raised and expectorated.

(*b*) **Broncho-vesicular Breathing.**—One of the common mistakes of the physical diagnostician in his days of pupilage is to call broncho-vesicular "bronchial" breathing. There are, indeed, so many variations between pure bronchial and pure vesicular respiration in diseased conditions that such an error is excusable, and the alternative names, "indeterminate" or "transition" breathing, are well deserved. The term is applied to a breath sound of which either inspiration or expiration has more or less of the harsh, blowing bronchial quality, with an expiration usually as long, rarely longer, often shorter, than the inspiration; or, as nearly as can be judged, midway between bronchial and vesicular.

Broncho-vesicular respiration is in general indicative of the same pathological conditions, but of lesser amount and degree, as bronchial breathing—i. e., partial consolidations, small patches of solid or collapsed lung, or consolidations overlaid by unaffected lung (see (2) under (*a*) preceding, also *E* and *F*, Fig. 148). If harsh broncho-vesicular respiration, with prolonged expiration, is heard permanently at one apex, especially the left, it is very significant of tuberculosis. With reference to this form of breathing it must be confessed that at times one may remain doubtful as to its exact meaning.

(*c*) **Vesicular Breathing.**—While retaining its characteristic qualities, vesicular respiration presents certain variations of diagnostic importance. It may be—

(1) *Weak or Absent.*—In old persons the vesicular breathing is normally of slight intensity (senile respiration), so also in some younger individuals when breathing quietly. The respiratory murmur is weak in emphysema, as the air cells are permanently much distended, comparatively little air enters, and conduction of the tracheal sound is impaired. Weakness may be due also to fat chest walls, thickened or adherent pleura, and a moderate pleural effusion. Absence or weakness of vesicular breathing may be found over the

area of lung supplied by an obstructed bronchus (foreign body, pressure, or secretion, *C*, Fig. 148); over a closed pneumothorax (one not communicating with a bronchus); over large pleural effusions (Fig. 149), in which case either the relaxed lung fails to vibrate, or the fluid acts under such circumstances as a poor transmitter of sound; over portions of a fibroid lung; and sometimes over the earliest state of a tuberculous apex.

(2) *Increased or Puerile.*—Loud vesicular breathing is normal in infants and children, diminishing in intensity up to the age of 12 years. It is sometimes confounded with bronchial respiration by the inexperienced auscultator, and closely resembles broncho-vesicular breathing; but no matter how intense and apparently harsh it may be, the normal ratio between the length of inspiration and expiration is retained. In the adult the respiration over the right infraclavicular space and apex normally approaches this type. It is heard in many cases of dyspnœa, especially from cardiac causes, because of the increased vigour of the respiratory movements. Puerile breathing over one lung is usually compensatory for disease of the other lung (extensive consolidation, effusion, or congestion), or in a portion of one lung for disease in the remainder of the same lung.

(3) *Harsh or Prolonged Expiration.*—Under this name are included certain forms of vesicular respiration which differ from the type by having an unusually harsh or prolonged expiration. When it is heard over both sides of the chest, and is rather soft and low-pitched, it is usually due to asthma or emphysema with bronchitis. The most important diagnostic occurrence of prolonged expiration, especially if high pitched, is its discovery over an apex of the lung as an early sign of tuberculosis. When found over the left apex it is very significant of disease; but at the right apex, in the absence of other signs (râles, elevated temperature, etc.), its diagnostic value is by comparison slight, because of the normal existence at that point of puerile or moderately broncho-vesicular breathing.

(4) *Cog-wheel Breathing.*—Jerky, wavy, or interrupted breathing—vesicular respiration, with short, irregular intermissions, especially during inspiration—is by itself of little diagnostic value. It is observed in healthy but nervous individuals, and is due in this case to the lack of smooth muscular respiratory action. In disease it is caused either by the air forcing its way by a series of efforts through the small bronchioles, the air column being broken and delayed by tenacious mucus, or by the expansion of different lobules at different times—for the same reasons—or by both. It may be confused with the cardio-pulmonary whiff due to rapid and excited action of the heart. The conditions for its production are present in early and late phthisis.

(D) The Voice Sounds in Health and Disease

Having applied the stethoscope, if the patient is asked to speak aloud with his face turned away from the examiner, a buzzing noise without any semblance of articulate sounds is perceived. It varies in intensity according to the loudness and depth of the subject's voice, and, following a suggestion by Rainy, seems to originate at the surface of the chest. The nearer the stethoscope is placed over the main bronchi, the more decided is the buzzing sound—the vocal resonance. Consequently, it is well marked over the right upper chest. Just as the vocal fremitus consists of the vocal vibrations transmitted from the larynx through the tracheo-bronchial air columns and the substance of the lung to the chest wall, so the vocal resonance consists of the same vibrations appreciated by the sense of hearing, and it varies in intensity under similar conditions. The variations in vocal resonance and their significance are as follows:

(1) **Lessened or Absent Vocal Resonance.**—If the sound is indistinct, and seems to come from a point more or less below the surface of the chest, or no vibration whatever is perceived, one may infer that there is a pleural effusion (Fig. 149), thickening of the pleura, or obstruction of a large bronchus by pressure or a mucous plug (*C* and *D*, Fig. 148). It is also weakened in emphysema.

(2) **Increased Vocal Resonance.**—If the sound is insistent, and appears to be produced not far from the examiner's ear, the vocal resonance is increased. If the increase is very well marked it is *bronchophony*. Its most common cause is consolidation of the lung in the neighbourhood of the larger or medium bronchi (*E*, *F*, and *G*, Fig. 148). The causes of increased vocal fremitus (page 425) are also the causes of bronchophony.

(3) **Pectoriloquy.**—If the sounds become articulate, the uttered words being apparently spoken directly into one's ear, it is *pectoriloquy*, and denotes either a consolidation connecting a large bronchus with the chest wall, and acting as an unusually perfect conductor (*G*, Fig. 148); the presence of a cavity having a free communication with a bronchus (Fig. 143, also *A*, Fig. 148); an open pneumothorax, one having free communication with a bronchus (Fig. 143); or, more rarely, is observed over the compressed lung above a large pleural effusion (Fig. 142). When pectoriloquy is extremely distinct it is fairly reliable evidence of a cavity.

(4) **Whispering Pectoriloquy.**—If, instead of speaking aloud, the patient is required to whisper, nothing but a slight distant expiratory whiff is heard in the normal lung with each syllable of the whispered words. But when the conditions which cause bronchophony

and pectoriloquy are present, the whiffing sound is nearer and more distinct, according to the increase in the conductivity of the lung; and if the conductivity is perfect, or a cavity exists, the words themselves are heard with curious distinctness and Lilliputian dimensions. The use of the whispered voice is a much more accurate and discriminating method of estimating the state of the vocal resonance, than to have the patient speak aloud. Its employment should be routine.

Here may be mentioned "*Bacelli's sign*"—i. e., that the whispered voice is transmitted readily and distinctly through a pleural effusion, provided the fluid is serous; while if the effusion is purulent and therefore denser, the whispered voice is not transmitted, thereby permitting a differential diagnosis between a simple serous pleurisy and an empyema. It is a sign which is unquestionably of some service, but if the serous effusion is large the whisper may fail of transmission.

(5) **Egophony.**—When the voice has a peculiar nasal, tremulous, or quavering intonation, resembling somewhat the bleating of a goat, it is termed egophony. It is heard at and just below the upper limit of moderate pleural effusions, and is usually best perceived at the angle of the scapula, or the space between the angle and the posterior axillary line. No very satisfactory explanation of the mechanism of its causation has as yet been offered. It is variously attributed to collapse of the bronchial tubes or to the interception by the effusion of the fundamental tones of the voice, the overtones remaining.

(6) **Amphoric Vocal Resonance.**—The voice has a metallic echoing quality in pneumothorax, similar in all respects to the breath sounds under the same circumstances.

(E) Adventitious Sounds or Accompaniments

In normal lungs nothing is heard but the breath sounds, but in diseased conditions various additional sounds may be perceived, which originate either in the lungs or the pleura—as follows:

(*a*) **Râles.**—This is the generic name given to adventitious sounds produced either in the bronchial tubes or the air cells. Râles are divided, according to the impression made upon the mind of the observer, into *dry* and *moist* (Fig. 150).

(1) *Dry Râles* (*Rhonchi*).—These are whistling, squeaking sounds caused by the passage of air through bronchial tubes which are partly occluded by swelling of the mucosa, or by spasm, or a lining of tough, viscid mucus. If high pitched, whistling, and heard in greatest abundance toward the end of inspiration, they are called *small* or *sibilant* râles, and originate in the smaller tubes. If low pitched and

greater volume, purring, almost snoring, in quality, beginning rly and continuing during inspiration, and perhaps during expira- on also, they are *large* or *sonorous* râles, having their seat in the rger tubes. Tubes of medium size afford râles of medium pitch.

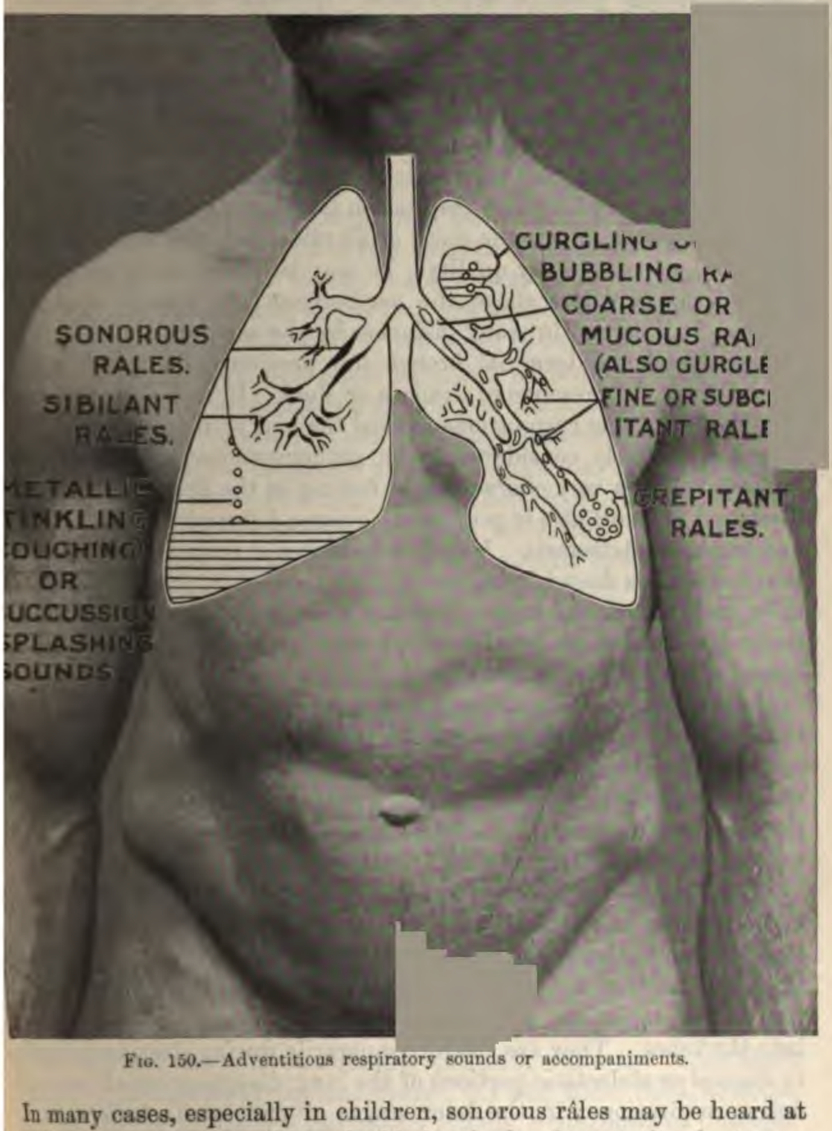


Fig. 150.—Adventitious respiratory sounds or accompaniments.

In many cases, especially in children, sonorous râles may be heard at a distance, and are readily palpable by the hand upon the chest.

These râles are significant of bronchitis in the early stage when the bronchial mucosa is congested and swollen, with perhaps a small amount of thick secretion; are heard abundantly and typically in bronchial asthma ("nest of kittens"); and isolated sonorous râles are frequently found in pulmonary phthisis, due to the partial blocking of a bronchus by a plug of tenacious muco-pus.

(2) *Moist Râles.*—Crepitations or moist râles are produced either by the expansion of previously closed alveoli, or the passage of air through fluid in the bronchi or in a cavity. Some crepitations originate in the pleural sacs. Moist râles vary in size and character, according to the calibre of the tube or the dimensions and nature of the cavity in which they are created. The absence of râles can not be affirmed until after deep inspiration or the act of coughing.

The *crepitant râle* is the finest of all râles, and, although classified as moist, possesses a dry quality. It may be imitated by rubbing a lock of the hair just above the ear between the thumb and forefinger. It is caused in many instances by the separation of the walls of the alveoli and terminal bronchioles previously collapsed or adherent from the presence of exudate or fluid. It is heard typically as a shower of fine crepitations toward the end of inspiration. This râle was formerly considered to be pathognomonic of pneumonia; but, while it is a very characteristic finding in the first stage of this disease, it is heard also in pulmonary œdema, hemorrhagic infarction, and localized atelectasis. Mixed with larger crepitations it may be heard, during a deep inspiration, over the *apices*, in those who habitually underinflate the lungs; over the *bases*, in those who have been lying for some time in the dorsal position, especially during the course of an exhausting disease.

It is further to be noted that a fine pleural friction may simulate the crepitant râle so closely that the two are indistinguishable by the ear, and the discrimination must be made by the preponderance of the associated symptoms and signs. Indeed, it is a question whether the crepitant râle should not be defined as a fine crepitation of a dry quality produced either in the air cells *or* the pleura.

The *fine moist* or *subcrepitant* râle is next in size to the crepitant, and originates in the small bronchi. It is heard both during inspiration and expiration. Râles of this character indicate the presence of fluid, and are therefore suggestive of bronchitis in the stage of secretion (moist replacing dry râles), pulmonary œdema, or hemorrhage into the tubes. They are heard temporarily during deep inspiration in disused or atelectatic portions of the lung, disappearing after a half dozen respirations; in the later stages of pneumonia (*râle redux*); and around the borders of patches or areas of consolidation from any cause. A number of small, moist "crackling" râles at one apex is very significant of beginning phthisis. A few moist râles at the base of the lungs usually indicate slight œdema, hypostatic congestion, or disuse of the lungs.

Coarse, *large*, or "mucous" râles, existing during inspiration, expiration, or both, originate in the larger bronchi, and arise from the same causes as do fine or subcrepitant râles. Large râles of a gurgling or bubbling character may be due to the interrupted passage of air through a considerable amount of fluid in a cavity or a large bronchus, and are best developed by coughing or forced breathing. They are most commonly heard in the softening stage of phthisis, more rarely in bronchitis with profuse secretion and bronchiectases.

Ringing or *metallic* râles derive their peculiar character from the acoustic properties (due to size, shape, character of walls, contents) of the cavities or tubes in which they originate. Such râles are usually found over the upper half of the lungs in conjunction with amphoric respiration and percussion note, and accordingly denote cavities, consolidation around large bronchi, or compressed lung.

A single metallic râle (*metallic tinkling*), occurring perhaps in a series of 3 or 4, after coughing or deep respiration, is due to pneumohydrothorax, rarely to a large, partly filled cavity. It sounds like and, indeed, is the falling of a drop of fluid from the surface of the lung upon the surface of fluid in the pleural cavity (Fig. 150). It has been compared to the dropping of water into a cistern, and derives its peculiar characteristics from the resonating qualities of the air-containing space.

It is sometimes difficult to decide whether a given sound is a fine moist râle or a fine friction sound. It will aid in the discrimination to remember that râles may be caused to disappear by coughing (removal of fluid from the tube); that they are less superficial; not strictly localized; are heard mainly during inspiration; and have a moist quality.

Two other clinical points may here be noted. First, that when bronchitis, asthma, and pleural effusion coexist, the râles may be so numerous and plainly heard that the presence of the effusion may readily be overlooked. In a personal case of this kind the lessened resonance and the displaced heart led to a correct diagnosis, and aspiration took away 80 oz. of fluid. Second, that if the temperature is elevated and the patient evidently quite ill, while the whole chest is filled with numerous fine and coarse, dry and moist râles, so abundant as utterly to obscure the breath sounds, a diagnosis of bronchopneumonia may safely be made.

(*b*) **Succussion Sounds.**—If, when the upper part of the body is somewhat vigorously shaken from side to side, a metallic splashing sound is heard, audible at a distance, it is almost certain evidence of the presence of air and fluid in the pleural cavity (Fig. 150). A similar sound originating in the stomach or colon may be a source of error. Very exceptionally it is due to a large, partly filled lung cavity.

(*c*) **Friction Sounds.**—Sounds variously described as rubbing, creaking, grating, rasping, crepitating, or leathery are due to the friction of apposed inflamed and fibrin-coated pleural surfaces. the pleural surfaces are subsequently separated by effusion the sounds disappear, and may return when the fluid is absorbed. Friction sounds are superficial—i. e., give an impression of nearness to the ear; are quite strictly localized and do not tend to shift their position; are not removed or perceptibly modified by cough or forced respiration; can sometimes be intensified by pressure with the stethoscope, which is painful; are heard especially during inspiration, may continue during expiration; and at times become palpable (friction fremitus). Friction sounds, according to the impression made upon the observer, may also be classified as fine, medium, and coarse. The finer frictions may very closely simulate the small or crepitant râle, but the points noted in the preceding sentence will in most cases serve for differentiation.

Friction sounds over the thorax signify pleurisy or pericarditis, more rarely (over lower thorax) peritonitis. They are absent in beginning hydrothorax, as the pleura is not inflamed. If found at the base of the right lung posteriorly, or over the 7th, 8th, or 9th right interspace anteriorly, it may indicate hepatic abscess, or cancer, or subphrenic abscess (subdiaphragmatic peritonitis), because of the involvement either of the peritoneal covering of the liver (perihepatitis) or of the complementary pleura from its contiguity to the inflamed peritoneum (Fig. 179).

Subpleural friction (RIESMAN) is a soft rubbing or fine, soft crepitation, without pain or evidence of consolidation, which has been observed in cases of miliary tuberculosis, the tubercles not causing inflammatory roughening of the pleura but lying just beneath and projecting sufficiently to produce the sounds observed.

SECTION XXXIII

THE ABDOMEN. METHODS AND RESULTS OF ITS GENERAL EXAMINATION

It is desired to consider here the *topographical anatomy of the abdomen*, and the methods and results of its *general physical examination* by inspection, palpation, percussion, and auscultation.

I. TOPOGRAPHICAL MARKS, AREAS, AND ANATOMY OF THE ABDOMEN

Anatomical Landmarks of the Abdomen.—At the upper portion of the abdomen, the ensiform appendix and down-curving arches of the ribs are important landmarks which are readily identified. At the lower part of the abdomen, the iliac crests and especially the anterior superior spines of the ilium are easily found, even in the obese. The symphysis pubis marks the lower anterior limit of the abdomen in the median line. These constitute the *bony landmarks.*

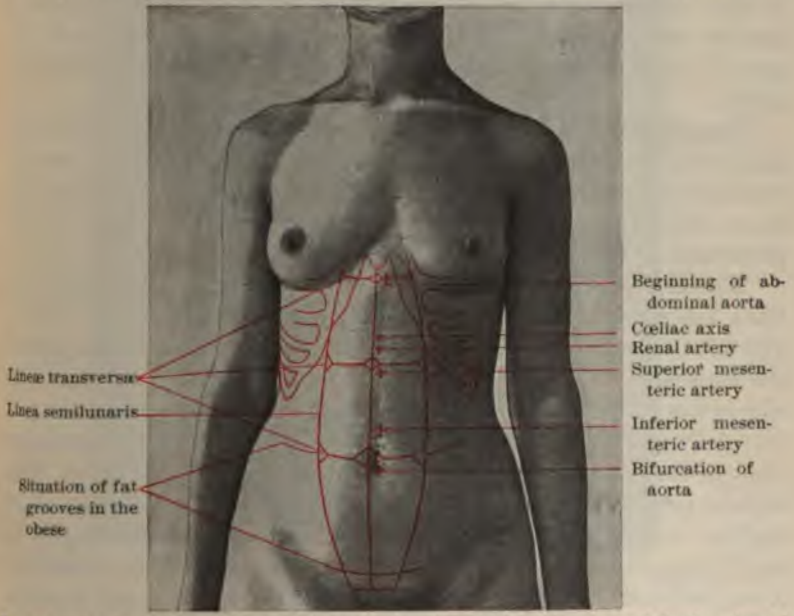

Lineæ transversæ
Linea semilunaris
Situation of fat grooves in the obese
Beginning of abdominal aorta
Cœliac axis
Renal artery
Superior mesenteric artery
Inferior mesenteric artery
Bifurcation of aorta

Fig. 151.—Showing the surface and bony landmarks of the abdomen and the location of the abdominal aorta and its more important branches.

There are certain more or less distinct *surface markings* which it is useful to bear in mind (Fig. 151). The *linea alba*, lying between the recti muscles, and running from ensiform appendix to pubic symphysis, is visible as a groove only above the umbilicus. The *umbilicus* itself is a most important point of departure, although its position is somewhat variable. It lies usually about 2 inches above the bispinal line (drawn between anterior upper spines of ilium), and corresponds to the tip of the spinous process of the 3d lumbar vertebra, on a level with the disk between the 3d and 4th lumbar vertebræ. On either side of the linea alba lie the recti muscles, bounded externally by the *lineæ semilunares*, which run with an outward curve from the lowest part of the 7th rib down to the pubic spines. The semilunar lines lie on either side about 3 inches from the umbilicus farther away if the abdomen is distended. The muscular segments of the recti are separated by the *lineæ transversæ*, one of which is at the ensiform appendix, one at the umbilicus, and one midway between the other two on a level with the costal cartilages of the 10th ribs. Rarely there is an additional line below the umbilicus. If the abdominal walls are thick with fat, two deep wrinkles or grooves cross the abdomen, varying in depth according to the degree of obesity. One is at the umbilicus, the other just above the pubes.

Topographical Areas of the Abdomen.—The surface of the abdomen is divided arbitrarily into certain regions in order to describe intelligibly the situation of organs or lesions. Two sets of areas, one more elaborate than the other, are in use, according to the personal preference of the examiner.

The *first set* divides the abdomen into 9 regions, by the use of 4 lines, 2 horizontal and 2 vertical. The *horizontal lines* are: the *infracostal* or *subcostal*, drawn across at the level of the lowest part of the 10th ribs; and the *bispinal*, a line connecting the anterior superior spines of the ilia. The *vertical lines* are drawn through the centre of Poupart's ligament, and are practically downward prolongations of the mammillary lines of the thorax. Fig. 152 shows these regions (after Joessel, somewhat modified), and the names by which they are known. It may be noted here that as the lumbar region extends from the vertical line in front to the median line posteriorly, it may be further subdivided, by prolonging the anterior and posterior axillary lines downward, into anterior, lateral, and posterior portions (Fig. 93). The boundary lines between the epigastric region and the hypochondriac regions on either side are made (as they should be) to correspond with the costal margin. The iliac spaces are frequently referred to as right and left inguinal.

The *second set* of areas requires 2 lines, 1 vertical, 1 horizontal, drawn through the umbilicus. The abdomen is thus subdivided into quadrants, right upper and lower, left upper and lower (Fig. 153). This arrangement has the desirable merit of simplicity.

Topographical Anatomy of the Abdomen.—The *abdominal aorta* (Fig. 151) begins just above a point midway between the upper border of the sternum and the umbilicus, lying to the left of the spinal column. It commonly bifurcates about ¾ inch to the left of and the same distance below the umbilicus, corresponding to the body of the 4th lumbar vertebra. One inch above the umbilicus arises the *inferior mesenteric* artery; 3½ to 4 inches, the *renal* artery; 4 to 4½ inches, the *superior mesenteric* artery; 4½ to 5 inches, the *cœliac axis*, corresponding to the body of the 12th dorsal vertebra.

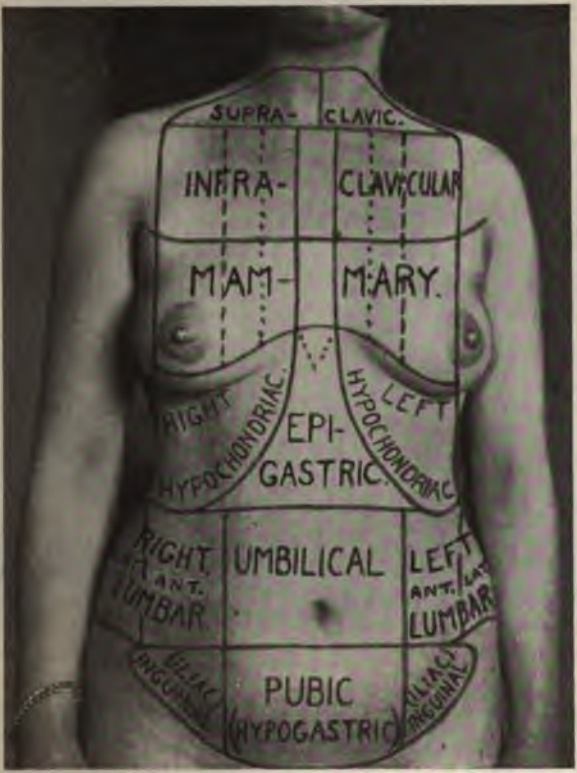


Fig. 152.—Showing nine topographical areas of abdomen. (After Joessel. Redrawn and modified.) See also Fig. 98.

The exact surface relations of the different abdominal viscera are stated in connection with the detailed examination of each organ. The contents of the various regions are indicated in Fig. 154. See also Plates I and II.

Practically there are various methods of indicating the location of abdominal lesions and physical signs.

(1) *The findings may be roughly stated* to lie in one or more of the 9 areas or regions marked out by the older system; or in one of the quadrants of the simpler method.

(2) *To localize more minutely* (Fig. 153): Suppose a vertical and a horizontal line, intersecting the umbilicus, to be already drawn. Then measure the distance of the point (centre of tumour, tender spot) above (or below) the umbilical horizontal line, and to right (or left) of the median line. These measurements will accurately orient the desired spot, according to the position of the lesion or physical sign.

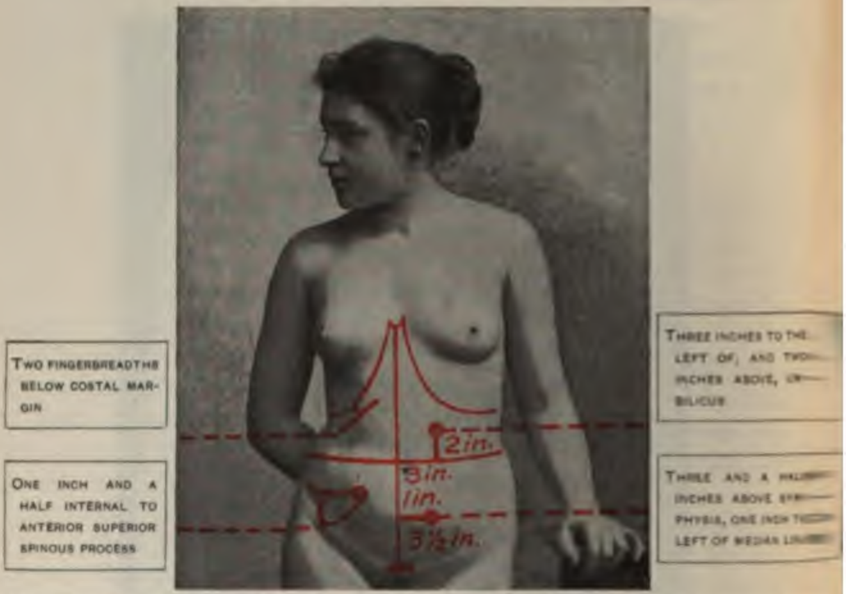

TWO FINGERBREADTHS BELOW COSTAL MARGIN
ONE INCH AND A HALF INTERNAL TO ANTERIOR SUPERIOR SPINOUS PROCESS
THREE INCHES TO THE LEFT OF, AND TWO INCHES ABOVE, UMBILICUS
THREE AND A HALF INCHES ABOVE SYMPHYSIS, ONE INCH TO LEFT OF MEDIAN LINE
2 in.
3 in.
1 in.
3½ in.

FIG. 153.—Showing the quadrants (right and left upper and right and left lower) of the abdomen; also different methods of describing with accuracy the location of abdominal signs or lesions.

Measure its distance above the symphysis pubis in, and, if to one side, to the right (or left) of, the median line.

Measure its distance above, and also inward from, the right (or left) anterior superior iliac spine.

It is sometimes convenient to state that the lower border of an enlarged liver or spleen lies so many fingerbreadths or a handbreadth below the costal margin. One fingerbreadth is ¾ inch; two, 1½ inch; three, 2¼ inches, and the width of the broadest part of the flat hand is from 3½ to 4 inches.

The Normal Abdomen.—In infants and young children the abdomen is normally large and prominent relatively to the size of the chest. It is larger in women (especially multiparæ) than in men, and in the former frequently presents a bulging of its lower half as a result of corset wearing. The upper abdomen may be temporarily distended by a hearty meal.

The normal abdomen on *inspection* is seen to be moderately arched. Its upper portion moves easily and quietly with the movements of respiration. By *palpation*, it is found to be soft, its walls are readily depressed, without causing pain, and the abdominal muscles are not contracted unless the patient is "ticklish" or nervous. No swellings or hard masses can be perceived unless there happen to be large portions of fæcal matter in the colon. On *percussion*, it is everywhere tympanitic except over the liver and spleen. *Auscultation* is negative except for occasional gurgling or sonorous sounds.

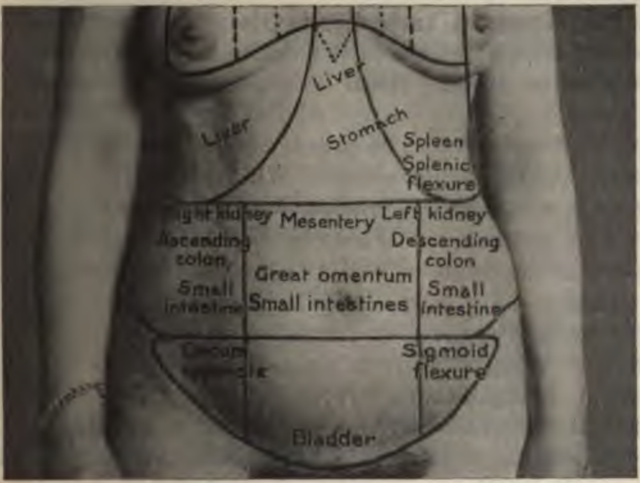


FIG. 154.—Showing roughly the contents of the nine topographical areas of the abdomen.

II. METHODS AND GENERAL RESULTS OF ABDOMINAL INSPECTION

Technic.—With the patient in bed the abdomen should be exposed by turning well down all the bed clothing, thick quilts or blankets particularly, except the sheet; then, under cover of the sheet, drawing the nightdress as far up as the lower sternum, and afterward folding the sheet down from a point a short distance above the pubes. The position of the patient should be as symmetrical as possible, the trunk being turned neither to right nor left (i. e., the bispinal line horizontal), and a good illumination secured. While the recumbent position is necessary for an examination of the abdomen, it is sometimes of service, when practicable, to make an observation in the standing or sitting posture, as pendulosity or prolapse of the abdominal walls or viscera may thus be more clearly manifested. Inspection of the abdomen should be made from various directions—front, side, and back, and obliquely as well—in order to detect pulsations or vermicular movements. Owing to the very intimate correlation of inspection, palpation, and percussion, as applied to the abdomen, some of the results of inspection will be rehearsed in connection with palpation and percussion.

Results of Abdominal Inspection in Disease.—The abdomen should be inspected with reference to its cutaneous surface, nutrition of walls, shape, size, bulging (local or general), retraction, and movements.

(1) *Skin of Abdomen.*—The skin is smooth, shining, and stretched in excessive abdominal distention. Whitish streaks or striæ (*lineæ albicantes*) are significant of previous long-continued distention, as in ascites, fat, or pregnancy, and may be seen on the buttocks and upper portions of the thigh, as well as upon the abdomen. A deposit of pigment, especially in the middle line (the *linea alba* becoming the *linea nigra*), is observed in pregnancy and in chronic abdominal enlargements. Copper-coloured, scaly, somewhat circular spots upon the abdomen are significant of secondary syphilis. The brownish or yellowish macular areas of chloasma may also be found here. Enlarged glands in the groins or retracted cicatrices may be indices of present or past specific or other venereal infection.

(2) *Enlarged Superficial Abdominal Veins.*—A number of enlarged veins radiating from the umbilicus constitutes the *caput Medusæ.* This is significant of portal obstruction (hepatic cirrhosis or tumour), and represents an effort to establish a freer communication between the portal veins and those of the abdominal parietes by way of the round and falciform ligaments, the small veins of the latter becoming enlarged.

General enlargement of the superficial abdominal veins occurs from similar conditions—e. g., cirrhosis or tumour of liver; ascites of long duration and greatly dilated stomach; or pressure upon the inferior or superior cavæ by abdominal or mediastinal tumours. If there is obstruction to the inferior cava, a dilated lateral vein may be present, running up the right midaxillary line, connecting the tributaries of the superior cava with the enlarged inferior epigastric veins. An effort should be made, by emptying the vein and watching it refill, to determine the direction of the blood flow. In obstruction of the portal vein and inferior cava the current is upward, the most usual finding; but if the superior cava is pressed upon, the direction is downward, the blood from the superior cava endeavouring to enter the inferior cava through the medium of the azygos veins, which communicate with many of the tributaries of the inferior cava. If the veins in the pubic region alone are distended there is probably some obstruction (pressure or thrombosis) below the liver.

(3) *Enlarged* and visible epigastric *arteries* in the abdominal walls constitute a sign of the rare condition of obstructed aorta or iliac arteries.

(4) *Umbilicus.*—The navel is deeply retracted in stout people; if projecting, it may be due to portal obstruction, pregnancy, or hernia; it is flattened, or protruding and stretched, in excessive ascites or other abdominal distentions; or it may be eczematous or inflamed.

(5) *Absent respiratory movement* of the abdomen is a somewhat significant sign of peritonitis, the great pain inducing tonic contraction and rigidity of the abdominal muscles. This symptom, together with inspiratory retraction and excessive abdominal breathing, has been elsewhere considered.

(6) *Visible Peristalsis.*—The vermicular movements of the intestines are sometimes visible if the abdominal walls are not too thick. The peristalsis may be made more active by manipulation; by sharply tapping the surface with the finger; flicking with a wet towel; the laying on of a cold hand; or the application of faradism. It presents itself as a series of rolling, rounding elevations, which increase and subside, accompanied or not by borborygmi. At their height the protuberances are noticeably resistant and tympanitic. It is sometimes possible to differentiate peristalsis of the transverse colon from peristalsis of the stomach by the fact that the waves of motion run in the former from right to left, in the latter from left to right.

While visible peristalsis may occur as a normal event in persons with extremely thin and relaxed abdominal walls, especially multiparæ, its principal diagnostic association is with intestinal obstruction and stenosis. At times an inference may be drawn as to the site of the obstruction by the location and character of the moving distention. If the obstacle is at, or a short distance proximal to, the ileo-cæcal valve, the swollen and mobile coils of intestine lie one above the other (ladder pattern) in the central portion of the abdomen; but if the constriction is low down in the large intestine, in the neighbourhood of the sigmoid flexure, the distention is observed mainly in the circumferential portion of the abdomen—i. e., the course of the colon. If there is a persistently recurring excessive protuberance at one point, which disappears with a loud sound, it may be conjectured to be at the point of stenosis.

Visible peristalsis in the upper left quadrant with the waves of motion running from left to right may be due to a much-dilated stomach.

III. METHODS AND RESULTS OF GENERAL ABDOMINAL PALPATION AND PERCUSSION

Technic of Abdominal Palpation.—The abdomen being exposed as for inspection, the warm hand is laid flat upon the surface, letting it remain quiet until the skin has become somewhat accustomed to its presence. Palpation should then be made mainly by somewhat circular pressing movements, sliding the skin over subjacent parts, and passing smoothly and steadily from one portion of the surface to another. A sudden poke with the finger tips will often defeat the examiner's object by causing immediate contraction of the abdominal muscles. After tolerance is established, deeper and localized palpation may be made with the pulps of the fingers to determine more exactly the existence of tender spots, or the size, shape, and mobility of existing masses or swellings. As in all examinations to detect pain on pressure, one should pay attention to the face of the patient rather than to verbal expressions of suffering. If it requires firm pressure to elicit tenderness the latter is apt to be real and deep seated rather than a superficial hyperæsthesia or surface lesion. If malingering or hysterical exaggeration is suspected, it is helpful to divert the attention of the patient by pressure with one hand upon a widely different portion of the surface, conjoined with suggestion, while the other explores the original point of complaint—a procedure which not infrequently reveals a significant absence of tenderness with greater pressure than that of which bitter complaint was originally made.

If the abdominal muscles are contracted to an extent which interferes with proper palpation, the patient may be encouraged to relax them voluntarily; or the knees may be flexed, and a pillow placed under the head and shoulders to diminish the tension; or he may be directed to take several deep respirations; or to breathe as rapidly as possible. Toward the end of expiration the muscles are usually in a momentarily relaxed condition. Deep breathing, moreover, determines, by the occurrence of relaxation, whether a mass felt is a contracted belly of the rectus or ridge of the lateral and posterior abdominal muscles, and whether a tumour found is movable with respiration. If it is desired to get deep down into the abdomen, one hand, re-enforced by the other laid upon it, should exercise increasing pressure during a series of forced respirations, following the sinking abdominal walls with each expiration, and maintaining during each inspiration the ground which has been gained. The re-enforcement of the palpating hand by the other preserves the perceptive delicacy which the former would otherwise lose on account of the very considerable muscular power required to be exerted. If there is fluid in the peritoneal cavity and one wishes to palpate an organ obscured by its presence, a sudden deep pressure with the finger tips ("dipping") may successfully displace the fluid and allow the desired end to be attained. Finally, if the diagnosis is of sufficient importance, a general anæsthetic may be given in order to eliminate the difficulties due to pain or contraction of the muscles.

The various regions of the abdomen should be systematically explored, never forgetting to examine the umbilical, inguinal, and femoral sites of hernia. To examine the lateral portions of the abdomen, both hands should be employed, one being slipped under the body so as to make forward pressure in the space between the last rib and the iliac crest, thus pushing the deeper structures up against the other hand placed over the abdomen in front, which can then appreciate the consistence and character of the intervening tissues. It is sometimes serviceable to examine in the knee-elbow position, or with the patient standing and leaning forward, supporting the body by the hands upon a table or chair. If the patient has a large, fat abdomen, it is advantageous to have him turn partly over to one side or the other, thus "spilling" the intestines and thick abdominal walls away from the area under investigation. The utility of a digital rectal and vaginal examination in tracing the origin of abdominal lesions, especially those which are situated in the lower third of the abdomen, should not be forgotten. *Mensuration* of the abdominal circumference at the level of the umbilicus, and the length of its anterior wall from ensiform to symphysis, are useful in keeping track of the increase of ascitic fluid or the rate of growth of a large tumour.

Technic of Abdominal Percussion.—Pulmonary resonance, except in defining the upper limits of the liver and stomach, can be eliminated, and the distinctions to be made are between degrees of dulness and tympanicity.

Barring hepatic and splenic dulness, the abdomen normally is tympanitic, the pitch of the resonant note varying with the size of the air space and the degree of tension of the containing cavity. The smaller the air space and the greater the tension the higher is the pitch. Consequently the stomach and colon afford a lower pitched note than the small intestines, although the great variations in size and tension which take place from time to time greatly minimize the value of this sign.

Auscultatory percussion has proved a very disappointing method of outlining the contiguous air-containing abdominal viscera. "Flicking" percussion has many advocates, and is claimed to be very useful in detecting slight degrees of dulness. In this method the left forefinger is placed nail downward upon the surface, and the nail of the middle finger of the right hand having been pressed against the palmar surface of the end of the thumb, is suddenly allowed to escape, so as to strike sharply against the palmar surface of the left forefinger. If the percussion note of a deep-lying mass in the abdomen is to be elicited, the pleximeter finger must be pressed slowly and firmly down in order to push away or compress air-containing coils of intestine, which would otherwise afford a masking tympanitic sound. If dulness is found in any part of the abdomen where it should not exist, it is of great importance to ascertain whether it disappears or shifts with changes in the position of the patient (fluid, sometimes air).

Results of General Abdominal Palpation and Percussion.—Observation should be directed to ascertain the condition and resistance to pressure of the abdominal walls, general or local retraction or distention, the shape and consistence of palpable organs, the presence of tumours, thrill, fluctuation, abnormal dulness or tympanicity and other points, as follows:

(1) **Abdominal Walls.**—The thickness of the abdominal wall may be estimated by endeavouring to grasp it in the hand, or better by placing one hand on either side and approximating them. Increased thickness is due to fat or œdema. If the latter, there is pitting on steady pressure, and the swelling is more marked in the lower and lateral portions of the abdomen. Thickening may also be due to extensive suppuration in the abdominal walls, in which case the signs of inflammation will be present. Lax and thin walls, with wrinkled skin, are due to long-standing distention from ascites, tumour, repeated pregnancies, old age, or wasting disease. Induration and infiltration around the umbilicus are caused by periomphalitis, said to be a significant sign of tuberculous peritonitis. Fixation of the umbilicus may be present as an evidence of malignant disease of the liver, a sign comparable to the similar condition of the nipple in mammary cancer.

(2) **Rigid Recti Muscles.**—A persistent tonic contraction of one or both recti, amounting perhaps to extreme rigidity, if not due to nervousness or "ticklish" sensitiveness, is very significant of peritoneal inflammation, intra-abdominal hemorrhage, and rupture or perforation of hollow viscera of the upper abdomen especially, to thoracic inflammations, e. g., pneumonia, diaphragmatic pleurisy; of the right rectus alone, of appendicitis in particular; of both recti and all the abdominal muscles, of general peritonitis. In the slighter degrees there is merely a sense of resistance, the muscles contracting only when pressure is made. Rigidity may be absent when the abdominal muscles have undergone atrophy from overdistention, as in women who have borne children; or may vanish with the oncoming of an acute toxæmia and its resulting muscular atony.

(3) **General Retraction of the Abdomen.**—The abdomen as a whole may be sunken in wasting diseases. It is very marked in inanition from stricture of the esophagus or pyloric stenosis and gastric dilatation; in the violent vomiting and purging of cholera and severe gastro-enteritis; and in the pernicious vomiting of pregnancy. If tenderness and muscular rigidity is associated with the retraction, one may suspect the early stage of peritonitis. The scaphoid (boat-shaped) abdomen is also seen as a symptom of meningitis (basal especially), tumour of the brain, and lead colic, because of the irritative tonic spasm of the abdominal muscles.

(4) **General Distention of the Abdomen.**—As a rule, general enlargement of the abdomen is due to one of four things: *fat* in the abdominal walls, *fluid* in the peritoneal cavity, an excessive amount of *gas* in the stomach and intestines, rarely in the peritoneal cavity, or the presence of a large abdominal *tumour*.

Fat.—Enlargement due to adipose tissue is usually easy to determine, as it is associated with general obesity and the bulk of the extremities is proportionate to the dimensions of the abdomen. In some cases fat abdominal walls constitute a very serious obstacle to the discovery of small neoplasms. A possible source of error arises from the occasional accumulation of fat in the great omentum in the middle-aged, which at first glance simulates a pregnant uterus or median tumour, but careful palpation will reveal its nature.

Fluid.—If the distention is due to ascites—an accumulation of fluid in the peritoneal cavity—the centre of the abdomen, in the dorsal decubitus and provided the amount of fluid is not excessive, is flattened, and the lateral and dependent portions bulge outward from the weight of the fluid. If the amount is very great the entire abdomen is arched and prominent, the umbilicus is stretched or bulging, and the shape of the abdomen does not change when the posture is altered. If ascites occurs in a previously lax and pendulou men, the latter may protrude in an oddly conical form. On sion the flanks are dull and the centre of the abdomen resona air-containing intestines floating upward toward the highest (Fig. 155). Unless the fluid is encapsulated or its amount ex

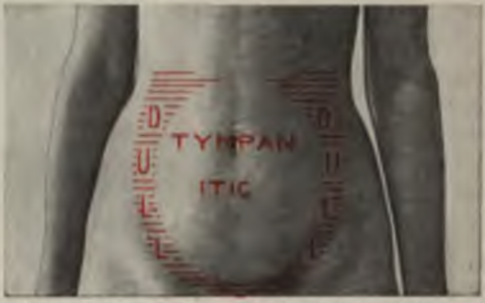


FIG. 155.—Showing the central tympanicity and lateral dulness of an abdomen c free fluid. Compare with Fig. 156.

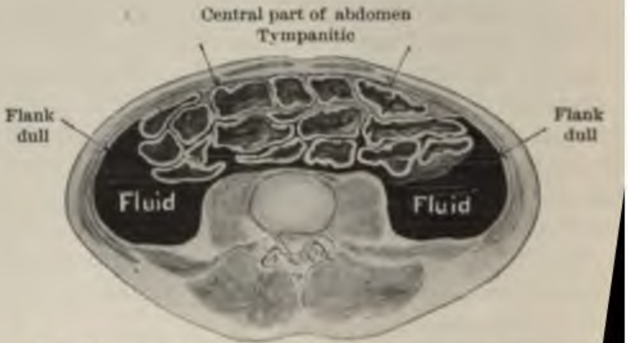


FIG. 156.—Showing both flanks dull in ascites, dorsal posture.

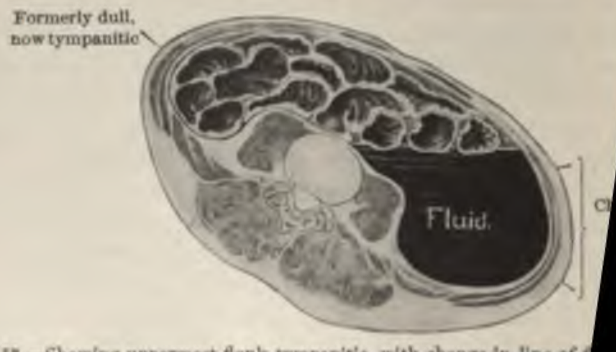


FIG. 157.—Showing uppermost flank tympanitic, with change in line of flank in ascites, after assuming the latero-dorsal posture. Compa

the line of dulness changes position as the patient is turned to one side or the other, the fluid gravitating to the lowest point and being replaced by the tympanitic intestine. The uppermost flank, previously dull, is now resonant (Figs. 156 and 157). If the presence of a small amount of fluid is suspected, the patient may be put into the knee-hand position, when, if the suspicion is correct, an area of dulness may be made out in the umbilical region, due to the subsidence of the fluid to that point. If the amount of fluid is considerable, fluctuation may be elicited. To obtain fluctuation, the ulnar edge of an assistant's hand should first be pressed firmly upon the linea alba in order to cut off the vibrations of the abdominal wall which may closely simulate fluctuation. One hand of the examiner is then laid upon one lateral wall of the abdomen, while the fingers of the other hand tap rather smartly upon the opposite side. If fluid is present, a transmitted wave, which may also be visible, is distinctly felt by

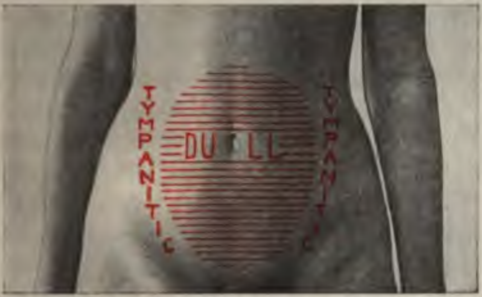


Fig. 158.—Showing central dulness and lateral tympanicity of abdominal cystic or solid tumours. Compare with Fig. 159.

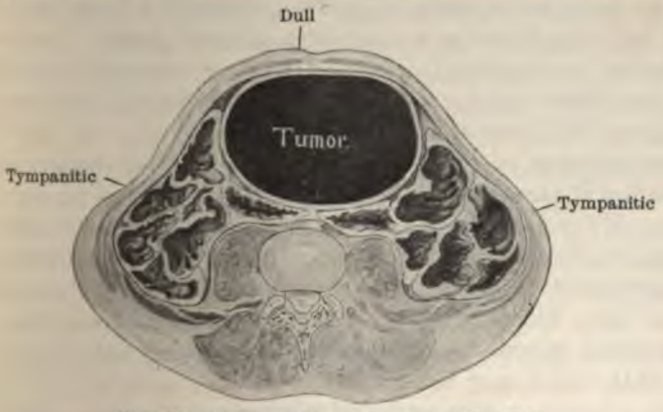


Fig. 159.—Cross section explanatory of Fig. 158.

the palpating hand. Very light percussion is often sufficient t cause it. This sign may fail in very large effusions under high pre sure.

It is necessary to discriminate ascites from pregnancy, ovarian cystoma, distended bladder, and rarely hydatid cyst of the liver o cyst of the pancreas. Aside from the history, vaginal examination catheterization and aspiration, these sources of error can be rule out if there is dulness on percussion of the flanks. The first thre conditions mentioned give rise to dulness in the centre of th abdomen and tympanitic resonance in the lateral regions, sign directly contrary to those of ascites, as the fluid (amniotic, ovarian urine) is not free in the peritoneal cavity and can not seek the low est points of the latter (Figs. 158 and 159).

Uncomplicated ascites—i. e., without general œdema—is most commonly due to cirrhosis of the liver; less frequently to chronic peritonitis. Other causes of the accumulation of fluid in the peritoneal cavity are obstruction of the thoracic duct, compression of the portal vein by abdominal tumours, portal thrombosis, or obliterating pericarditis. Hemorrhage into the abdominal cavity, due to ruptured tubal pregnancy, may, if sufficiently large, give rise to the physical signs of free fluid. In any case of ascites there may be a secondary œdema of the lower extremities, but the history will show that the abdominal distention antedated the swelling of the legs.

Ascites *plus* general œdema constitutes a part of the latter, and is due to renal, more rarely to cardiac, disease. A large accumulation is not often found as a result of a diseased heart. Very rarely it is due to the rupture of a cyst of the ovary or broad ligament, in which case there may be a history of a slow-growing localized (pelvic, right or left) tumour.

Some evidence of the causative disease may be obtained by an examination of the ascitic fluid withdrawn by puncture (*q. v.*).

Gas.—If the distention is due to gas in the intestines (meteorism, tympanites), the abdomen is arched and tense, universally tympanitic upon percussion (Fig. 160), and fluctuation can not be obtained—physical signs which render it an easily determined condition. If the distention is extreme, the diaphragm is pressed upward, and the action of the heart may be seriously impeded.

With reference to the causes of meteorism, it may be noted that, in moderate degree, it is often due to acute or chronic gastro-intestinal disorders, and is a frequent source of complaint in such cases when occurring in neurotic women. It is almost always present in typhoid fever, sometimes to an extreme degree, and in the typhoid state in general. A high grade of tympanites conjoined with great abdominal tenderness is significant of acute general peritonitis. Great and rapid gaseous distention of the stomach and intestines (*pneumatosis*) is an occasional symptom of hysteria. In all cases of excessive meteorism the possibility of intestinal obstruction should be borne in mind. Intestinal paresis, due to sepsis, to quick removal of pressure from intestines previously constricted — e. g., strangulated hernia—or compressed—e. g., by tumour or fluid—or to deranged or defective innervation, may also be a cause of meteorism and obstinate constipation (WOOD).

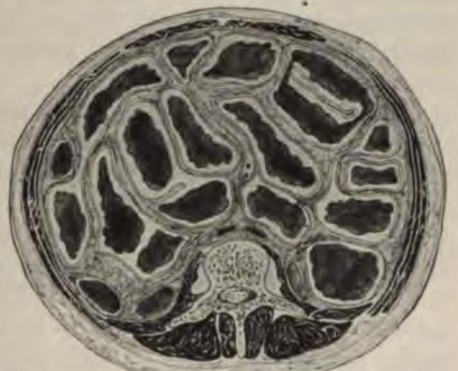


FIG. 160.—Showing abdominal meteorism, percussion note universally tympanitic. Frozen section from Pirigoff, redrawn.

In comparatively rare instances there is *free gas* in the peritoneal cavity, due to perforating ulcer of the stomach or intestine, a perforated appendix, or the presence of a gas-forming micro-organism in the cavity of the peritoneum. If, from the sudden onset of collapse, meteorism, and severe abdominal pain, a perforation peritonitis is suspected, one may endeavour to determine that the distention is extra-intestinal by finding whether lateral liver dulness is present in the dorsal position, and if so, whether it disappears when the patient is turned well over on the left side; and similarly with the splenic dulness. If this change takes place, one may infer that the air is in the peritoneal cavity, and that, being free, it has in each case risen to the highest point, thus interposing itself between the liver (or spleen) and the abdominal walls. The alteration with change of position is the significant sign, as liver dulness may nearly disappear as a result of ordinary meteorism, marked emphysema, hepatic cirrhosis, and a disease of rare occurrence—acute yellow atrophy of the liver.

Tumour.—Abdominal distention from tumours may, if the latter are very large, closely simulate enlargement due to the previously mentioned causes. It may be said that in general a large neoplasm gives a greater increase in the antero-posterior than in the transverse diameter as compared to the manner of increase caused by fat, fluid, or gas. Moreover, in the majority of large tumours the enlargement of the abdomen is not quite symmetrical; percussion does not show the uniform resonance of gas nor the lateral dulness and central tympanicity of free fluid; and palpation may declare the solidity of the mass. The tumours or organs which may grow to such a size as to cause general abdominal tumidity or distention are: of the *liver*, hydatid cyst, cancer, syphilitic and amyloid disease; of the *spleen*, malarial, leucæmic, and amyloid enlargement; of the *kidney*, sarcoma and cysts; cancer of the peritoneum and intestine; dilated stomach; ovarian cystoma and uterine fibroids; finally, as a source of error, the pregnant uterus.

The abdomen may be generally protuberant because of the presence of a considerable number of enlarged mesenteric glands, either tuberculous or a part of the glandular hyperplasia of Hodgkin's disease; so also in the chronic gastro-intestinal catarrh or greatly dilated colon of children; and in cretinism, rachitis, and pseudo-hypertrophic paralysis.

(5) **Local Bulgings, Swellings or Tumours.**—One can not exercise too much care in searching, first by inspection and then by painstaking palpation, for the presence of local swellings or small tumours in all cases presenting symptoms referable to the abdominal viscera. The following remarks apply equally to inflammatory swellings or new growths:

(*a*) *Sources of Error.*—There are certain difficulties or sources of error in the search which are worthy of mention. The occasional and sometimes almost insuperable obstacle presented by excessive thickness of the abdominal wall may be partly overcome by strong palpation, one hand being re-enforced by pressure with the other, and by spilling the fat, thick walls to one side or the other by position; ascites by "dipping" (page 463); and "ticklishness" by slow, equable movement and moderately firm pressure. The segments of the recti muscles when contracted may very exactly simulate a small tumour, and one may endeavour to eliminate this not uncommon element of doubt by insinuating the tips of the fingers under the edge of the apparent tumour, and then asking the patient to raise his head, upon which the muscle, if it be such, is felt to contract and thicken.

A localized spasmodic contraction of the abdominal muscles or persistent gaseous distention of a knuckle of intestine ("phantom tumour") may be extremely deceptive. An ostensible tumour of this kind occurs as a rule in an hysterical woman, is dull or dull tympanitic, may disappear during rapid and forced respiration, and—a crucial test—vanishes during full anæsthesia. It should, however, be stated that some "phantom" tumours are probably congenital or acquired dilatations of the colon (Fitz).

(*b*) *Points to be Observed.*—If one is satisfied of the presence of a tumour, the following points remain for determination:

Is it intra-abdominal or extra-abdominal; is it freely movable, and does it move with respiration; what is its size, shape, consistence, the nature of its surface; does it fluctuate; in what region of the abdomen does it lie; and from what organ, if any, does it spring?

(1) If situated in the abdominal wall, it is usually possible to gather up, either in one hand or between both, that portion of the abdominal wall overlying the tumour, when the latter can be distinctly felt to lie in the grasp of the hand. An intra-abdominal growth, on the contrary, can not thus be elevated and seized, the abdominal wall slipping easily over it unless it has contracted firm parietal adhesions.

(2) The mobility of the tumour should be tested by moving it in various directions, observing the extent of movement and the line in which it is most readily pushed — e. g., floating kidney, which is most easily carried upward and backward.

If, when the hand is laid upon the tumour, the latter is found to move up and down with each respiration, it may be inferred that it springs from organs in close relation with the diaphragm —i. e., liver, spleen, and, to a less extent, the kidney. This is a sign which possesses considerable diagnostic value, but it must be remembered that the tumour may have contracted adhesions in such a manner as to produce the same effect. On the other hand, tumours which would ordinarily move with respiration may be hindered from so doing by interference with the contraction

WITHOUT DEFINITE LOCATION

- Fæcal masses (in course of colon)
- Large gallstones or fæcal concretions (in intestine)
- Floating kidney (usually remains on its own side, but may be found anywhere between ribs and pelvis)
- Floating spleen (left side, but may descend into pelvis)
- Tumour of intussusception
- Pyloric tumour, usually cancer (very movable)
- Phantom tumour
- Masses of tuberculous or carcinomatous peritonitis
- Enlarged glands (tubercle, cancer, Hodgkin's disease)

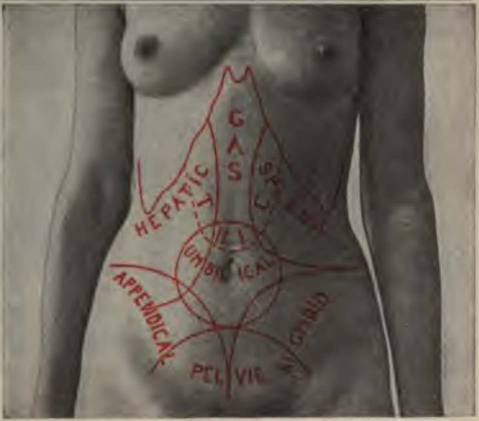


Fig. 161.—Showing the clinical areas of the abdomen with reference to the diagnostic value of the situation of circumscribed swellings or tumours; also lesions without definite location.

APPENDICAL AREA

- Acute appendicitis (tumour rarely palpable until 48 hours after onset)
- Chronic appendicitis (sausage-shaped tumour, palpable?)
- (Fæcal impaction in cæcum with sequent typhlitis, perityphlitis, paratyphlitis?)
- Fæcal abscess (perforating ulcer of colon)
- Tumour of intussusception
- Foreign bodies (gallstones, fæcal impaction, enteroliths)
- Cancer of cæcum or ascending colon
- Retroperitoneal sarcoma
- Floating or enlarged kidney
- Ovarian cyst or abscess
- Cyst of broad ligament
- Pyosalpinx
- Hæmatoma or hæmatocele (ruptured ectopic gestation)
- Psoas abscess
- Inguinal hernia
- Fibroid tumours

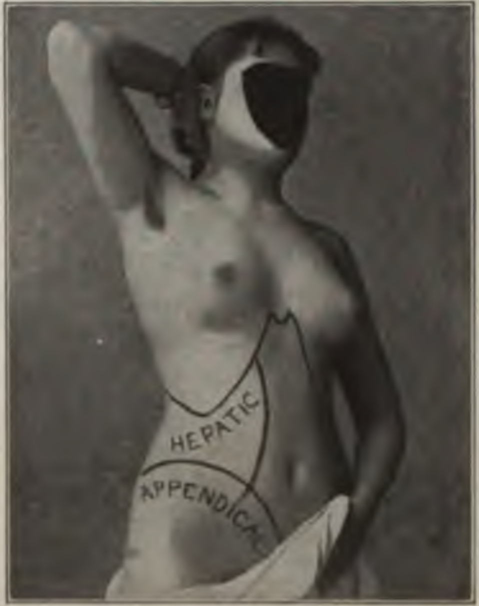


Fig. 162.—Showing possible findings in the hepatic and appendical areas.

HEPATIC AREA

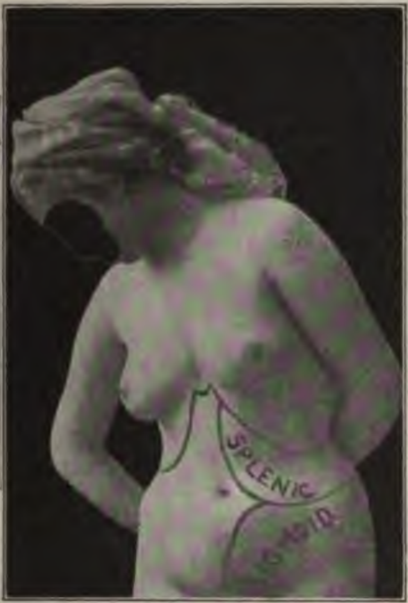
- Enlarged liver (passive congestion, hypertrophic cirrhosis, atrophic cirrhosis (early stage), gumma, amyloid disease, hydatid cyst, abscess, "lacing liver")
- Movable and prolapsed liver
- Gall bladder (pear-shaped mass) distended with bile, pus, stones, or enlarged by cancer
- Movable or enlarged (hydronephrosis or pyonephrosis, cancer) kidney
- Perinephritic abscess
- Subphrenic abscess (rarely palpable)
- Abscess due to caries of vertebræ
- Cancer or fæcal mass at or below hepatic flexure of colon
- Enlarged retroperitoneal glands
- Mesenteric cyst

liaphragm consequent upon pleurisy, emphysema, or a greatly d liver or spleen.

tumours which are *readily movable* by palpation, and which l when the patient is in the erect position, are floating liver, and kidney; tumour of the stomach (especially pyloric) or ie; fæcal masses or concretions, and gallstones. *Slightly* are tumours of the gall bladder and omentum above; uterus ries below. *Immovable* are tumours of the pancreas, aneurism bdominal aorta, abscess or inflammation due to disease of the ix, tumour of bone or abscess resulting from caries, and en-

NIC AREA

. OR MOVABLE
LAPSED SPLEEN
, OR MOVABLE
LAPSED KIDNEY
RITIC ABSCESS
STOMACH
CCUMULATION
OLITHS
TONES
IN LESSER PERI-
CAVITY
IC ABSCESS
LY PALPABLE)
DUE TO SPINAL



SIGMOID AREA

CANCER OF SIGMOID FLEXURE OR DESCENDING COLON
FÆCAL ACCUMULATION
ENTEROLITHS
GALLSTONES
PSOAS ABSCESS
FÆCAL ABSCESS
ENLARGED GLANDS
TUBERCULOUS PERITONITIS
CANCEROUS PERITONITIS
INTUSSUSCEPTION
HERNIA
MOVABLE SPLEEN
MOVABLE KIDNEY
OVARIAN TUMOUR
OVARIAN ABSCESS
CYST OF BROAD LIGAMENT
HÆMATOMA OR HÆMATOCELE
FIBROID TUMOURS

IG. 163.—Showing possible findings in the splenic and sigmoid areas.

retroperitoneal glands or abscess. Tumours of the stomach tine may change position with the peristaltic movements.

Note also its size, approximately or by measurement; its -round, ovoid, or irregular; its surface, whether smooth or ; and its consistence—soft, doughy, and indentable (fæcal moderately hard or stony. Can fluctuation be obtained—i. e., a cystic nature, with fluid or semifluid contents (hydrone- or pyonephrosis, ovarian cystoma, distended bladder, hydatid regnant uterus, ectopic gestation, or encysted abscess)? If fluctuation is present, test for the "hydatid thrill," by placing three fingers over the fluctuating mass and percussing strongly upon the middle one of the three, letting the plessor or striking finger rest at the end of each stroke, when, if the thrill is elicited, it will be perceived by the two lateral fingers.

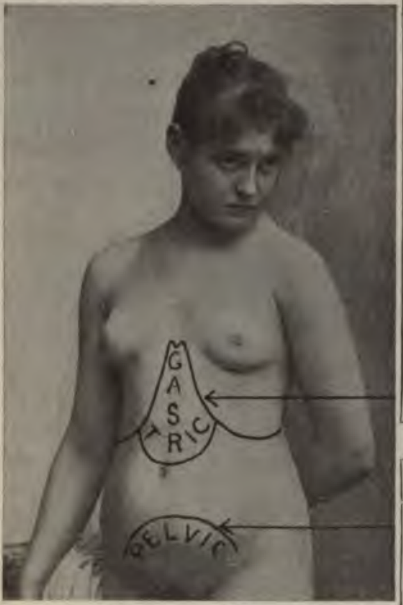


Fig. 164.—Showing possible findings in the gastric and pelvic areas.

GASTRIC AREA

FATTY TUMOUR OR ABSCESS OF ABDOMINAL WALL
DISTENDED STOMACH (GAS, FLUID, FOOD)
DILATED STOMACH (RARELY)
TUMOUR OF PYLORUS OR ANTERIOR WALL OF STOMACH (USUALLY CANCER)
INDURATION OF CHRONIC GASTRIC ULCER (RARE)
CYST, CANCER, OR SCLEROSIS OF PANCREAS, OR ACUTE HEMORRHAGIC PANCREATITIS
TUMOUR (CANCER, HYDATID CYST) OR ENLARGEMENT (PART OF GENERAL INCREASE) OF LEFT LOBE OF LIVER
DISTENDED (BILE, PUS, CONCRETIONS) OR CANCEROUS GALL BLADDER (RIGHT SIDE OF AREA)
CANCER OF TRANSVERSE COLON
TUMOUR OF INTUSSUSCEPTION
TUBERCULOUS OR CANCEROUS OMENTUM (TRANSVERSE, CORDLIKE TUMOUR)
ENLARGED POSTERIOR MEDIASTINAL, MESENTERIC OR RETROPERITONEAL GLANDS (TUBERCULOUS, CANCEROUS, HODGKIN'S DISEASE)
TUBERCULOUS ABSCESS
SUBPHRENIC ABSCESS (RARELY PALPABLE)
ANEURISM OF ABDOMINAL AORTA (MIDDLE LINE)
EFFUSION INTO LESSER PERITONEAL CAVITY (TO LEFT)

PELVIC (PUBIC) AREA

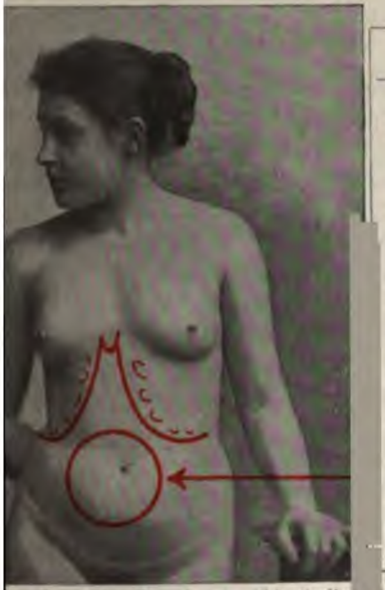
IN MEDIAN LINE: DISTENDED BLADDER; UTERUS, PREGNANT, DISTENDED (IMPERFORATE HYMEN); OR FIBROID TUMOUR
LATERALLY: OVARIAN TUMOUR, ABSCESS OF OVARY, MASSES DUE TO PYOSALPINX, RUPTURED ECTOPIC GESTATION (HÆMATOMA), TUBERCULOUS PERITONITIS, OR AN UNUSUALLY LONG INFLAMED APPENDIX LYING IN THE PELVIS

(4) Observe carefully in what part or region of the abdomen the swelling or tumour lies.

(5) Determine as accurately as possible whether it is entirely of abdominal origin, or whether it springs from the pelvis. Careful deep palpation, just above the brim of the pelvis, together with a rectal or vaginal examination, will usually determine this point, but cases occur in which errors are quite possible—e. g., an abscess of the ovary rising out of the pelvis sufficiently high to be diagnosed as an appendical abscess.

A decision as to the particular organ or structure from which a tumour springs, or a diagnosis of the nature and seat of the disease causing local swelling or bulging in various parts of the abdomen, depends not only upon the location and characters of the tumour or swelling, but also, and often to a large extent, upon the history of nd the results of chemical and microscopical examinations tum, gastric contents, blood, urine, or fæces.

lications Derived from the Situation of Abdominal Swell- mours.—For the sake of clinical convenience in describing icance of swellings or tumours according to the part of ıen in which they are found, one may recognise 7 areas or ıch named, with 2 exceptions (pelvic and umbilical), after mportant organ or part underlying it. These areas (Fig. oundaries of which necessarily overlap to some extent, are dian line *gastric*, *umbilical*, and *pelvic*; to the right, the d *appendical*; to the left, the *splenic* and *sigmoid*. Fur- as certain bulgings or tumours may occupy almost any he abdominal cavity, it is practicable to form, according stribution but with some necessary repetition, 8 groups of bdominal lesions. It is helpful from a diagnostic view- ave in mind the possible findings when palpating and per- ecial regions or areas of the abdomen. It is to be remem- a tumour or an enlarged organ in one of these areas may



UMBILICAL AREA

- UMBILICAL HERNIA
- DILATED AND DISTENDED (GAS, FLUID) STOMACH
- LARGE CANCER OF STOMACH
- MOVABLE AND PROLAPSED OR ENLARGED KIDNEY, SPLEEN, OR LIVER
- ENTEROPTOSIS (BULGING)
- CANCER OF INTESTINE OR OMENTUM (TUMOUR)
- PROLAPSED COLON (TRANSVERSE CORD IN LOWER PORTION OF AREA)
- ENLARGED MESENTERIC GLANDS (TUBERCLE, CANCER, ETC.)
- TUBERCULOUS OR CANCEROUS PERITONITIS
- PROJECTING VERTEBRÆ (SIMULATING A TUMOUR)
- UMBILICAL METASTASES FROM CANCER OF LIVER

FIG. 165.—Showing possible findings in umbilical area.

ıch dimensions that it underlies several of these areas, or ıy occupy nearly the entire abdominal cavity—e. g., liver, ırian tumour—but careful palpation, aided perhaps by the ables it to be traced to its origin in a particular region.

(1) SWELLINGS OR MASSES WITHOUT DEFINITE LOCALIZATION.—Here are included: *Fæcal masses* (impacted fæces) at some point in the course of the colon, most commonly at the cæcum or at one of the flexures: Large *gallstones* in the intestine or *fæcal concretions*: Floating *kidney*, which usually remains on its own side, but may be found anywhere between ribs and pelvis: Floating *spleen* (left side), but which may descend into the pelvis: The tumour of *intussusception*: *Pyloric tumour*, usually cancer, extremely movable, but commonly found in the upper and central portion of the abdomen.

(All of the preceding swellings, unless bound by adhesions, are more or less movable by the palpating hand.)

Phantom tumour (contracted abdominal muscles or distended knuckle of intestine): or the masses of tuberculous or cancerous *peritonitis*, or enlarged abdominal *lymph-glands* (tuberculosis, cancer, Hodgkin's disease).

(2) SWELLINGS OR MASSES IN THE GASTRIC AREA.—In the gastric area, as the term is here employed (Fig. 164), one may find:

Fatty *tumour*, or *abscess* of abdominal wall: Distention of the stomach from gas, or blood: Dilated stomach rarely; the epigastrium is, as a rule, depressed and hollow: Malignant or non-malignant *tumour of pylorus*, but in the majority of cases a distinct tumour in the gastric area is a cancer of the pylorus or anterior wall of the stomach: Palpable induration may possibly be due to a chronic gastric *ulcer*: Cyst, cancer or sclerosis of the *pancreas*, or acute hæmorrhagic pancreatitis: *Tumour* of the left lobe of the *liver* (cancer, hydatid cyst) or its enlargement as a part of a general increase in the size of the liver: A *gall-bladder* distended with bile or pus, packed with concretions, or affected by cancer, in the right-hand portion of this area, as a pear-shaped, perhaps nodular mass: Cancer of transverse colon and the tumour of intussusception.

A firm, elongated cordlike tumour lying transversely across this space is usually a tuberculous, rarely a cancerous *omentum*. Masses felt here may be enlarged posterior mediastinal, mesenteric, or retroperitoneal *glands* (tuberculous, cancerous, Hodgkin's disease). A swelling (fluctuation perhaps obtained) may be a tuberculous abscess; or a subphrenic abscess may contain gas, resulting from perforation of stomach or disease of pancreas, or, toward the left of this space, may be an effusion into the lesser peritoneal cavity from disease of the pancreas; and a pulsating tumour nearly in the middle line may be an aneurism of the abdominal aorta.

(3) SWELLINGS OR MASSES IN THE UMBILICAL AREA.—One may find here (Fig. 165): A somewhat tense and tympanitic protrusion at the navel—an umbilical *hernia*: Bulging due to a *dilated stomach*

(fluid, gas) or prolapsed; or a large *cancer* of the *stomach*: A prolapsed or enlarged spleen, to be traced upward and to the left under the costal margin and further identified by its notch: A prolapsed or enlarged liver, to be traced upward and to the right under the border of the ribs, to be further identified by its notch: A floating or enlarged *kidney* on one side or the other: Enteroptosis (bulging) and cancer of intestine or omentum (tumour): prolapsed *colon*, felt as a transverse cord in the lower portion of this area: and enlarged mesenteric glands. Unusually projecting vertebræ may simulate a tumour-mass.

(4) SWELLINGS OR MASSES IN THE PELVIC AREA.—In the pelvic (or pubic) area (Fig. 164) in the median line may be found a distended bladder; uterus, pregnant or distended by retained menstrual fluid (imperforate hymen), or fibroid tumour; and the bulging due to gastroptosis or enteroptosis. On one or the other side may be discovered an ovarian tumour, abscess of the ovary, pelvic masses due to pyosalpinx, ruptured ectopic gestation, tuberculous peritonitis, or inflammation of a long appendix lying in the pelvis.

(5) SWELLINGS AND MASSES IN THE HEPATIC AREA.—This area (Fig. 162) includes portions of the epigastric and anterior lumbar regions. The most common enlargements found in this area belong to the liver. These and other conditions are: Increased size of liver due to passive congestion, hypertrophic cirrhosis, cancerous, gummatous or amyloid liver, hydatid cyst, hepatic abscess and "lacing liver." The liver may be carried downward by the pressure of a right-side pleural effusion or be movable and prolapsed. A small pear-shaped mass may be a gall-bladder distended with bile or pus (empyæma), stuffed with stones or enlarged by cancer. Exceptionally it may be felt below the level of the umbilicus.

Here also may be felt a movable kidney or one which is the seat of hydronephrosis, pyonephrosis or cancer, as well as a perinephritic abscess, subphrenic abscess (rarely palpable) or one caused by caries of the spinal column.

Cancer or fæcal mass at and below the hepatic flexure of the colon, and enlargement of the retroperitoneal glands.

(6) SWELLINGS AND MASSES IN THE APPENDICAL AREA.—The diseases of the appendix and other conditions which may cause swellings or tumours in this area are (Fig. 162): Acute appendicitis—tumour rarely to be felt until forty-eight hours from its onset: Chronic appendicitis—the enlarged and thickened appendix may, at times, but by no means always, be felt as a sausage-shaped tumour.

Fæcal accumulation in the cæcum which may give rise to a typhlitis (inflammation of the cæcum), perityphlitis (inflammation of the serous covering of the cæcum, and paratyphlitis (suppurative inflammation of the connective tissue in the neighbourhood of the cæcum). The latter condition simulates the tumour of appendicitis exactly, but by comparison is very rare. The existence of these lesions apart from an appendicitis is extremely doubtful.

Fæcal abscess from perforating ulcer of colon: Three-fourths of the tumours due to intussusception are in this area in the neighbourhood of the cæcum (Fowler): Obstructing foreign bodies (gall-stones, fæcal impaction, enteroliths) are usually found in this area: Cancer of the cæcum or of the lower part of the ascending colon, and retroperitoneal sarcoma: A distended gall-bladder (which may descend below the level of the umbilicus): Floating or enlarged kidney: Ovarian cyst, cyst of broad ligament, pyosalpinx or ovarian abscess, or hæmatoma or hæmatocele from a ruptured ectopic gestation: Psoas abscess, enlarged glands, hernia (at the inguinal rings): and as unique occurrences (Fowler) a lithopædion escaped from the right Fallopian tube, and a fibromyoma of the ileo-cæcal region.

It will be noted that lesions originating in the hepatic and pelvic areas may descend or rise into the appendical area, and the former two are consequently represented (Fig. 161) as encroaching upon the latter.

(7) Masses and Swellings in the Splenic Area.—Enlarged spleen from various causes, traced upward under the ribs, or the same organ movable and prolapsed (Fig. 163): Enlarged kidney—from various causes—or the same organ movable and displaced, and perinephritic abscess: Dilated stomach, distended with food or gas; or cancer of the stomach: Fæcal accumulation in the splenic flexure of the colon: and subdiaphragmatic abscess, or effusion into the lesser peritoneal cavity.

(8) Masses and Swellings in the Sigmoid Area.—One may find here (Fig. 163): Cancer of the sigmoid flexure or descending colon: Accumulation or impaction of fæces, or foreign bodies: Psoas abscess and fæcal abscess (from ulcer of colon): Enlarged glands and the masses of tuberculous or cancerous peritonitis: The tumour of intussusception, or in the inguinal rings the tumour of hernia: Floating spleen and kidney may be present here: and coming from the pelvis upward—ovarian tumour, cyst of broad ligament, abscess of ovary, fibroid tumour, and hæmatocele or hæmatoma from ruptured ectopic gestation.

Auscultation of the Abdomen.—Auscultation is only occasionally useful in the examination of the abdomen.

Over the stomach one may hear the sounds of swallowed fluid entering the viscus, and splashing or bubbling sounds with movement of the body (succussion), which are of little or no diagnostic value; or an unusual intensity or quality of heart sounds, the hollow air-containing organ acting as a resonator to re-enforce or alter their normal characters.

Over healthy intestine one can always, at least with patient waiting, hear bubbling, gurgling, ringing, sonorous, or cooing sounds, due to peristaltic contraction forcing gas through fluid or through coiled or knuckled intestine. If a diagnosis is to be made between mechanical obstruction and intestinal paresis (sepsis, defective innervation, etc.), both having tympanites and obstinate constipation as symptoms in common, the entire absence of sound during prolonged auscultation is significant of paresis. On the other hand, in mechanical obstruction the sounds are usually increased in intensity and number by the greater vigour of the peristaltic movements in endeavouring to overcome the stenosis.

In paralysis of the muscular coat of the intestine, usually due to septic peritonitis, the heart and breath sounds may be audible over the entire abdomen, especially in children. They are not audible in tympanites due to other causes than intestinal paralysis, and the distinctness with which they are heard bears a direct relation to the degree of atony (PETERS).

Soft rubbing or crepitating friction sounds are sometimes heard in peritonitis, especially over the right hypochondrium (perihepatitis) or an enlarged spleen (perisplenitis) or a systolic bruit over the latter. One may hear at times a venous hum, or the bruit of aneurism (or pressure) affecting the abdominal aorta; rarely, a venous hum over the liver (pressure on vena cava); and if pregnancy exists, the fœtal heart sounds (funic souffle, uterine souffle, *choc fetal*). *Auscultatory percussion* is dealt with in the examination of special organs.

SECTION XXXIV

EXAMINATION OF THE DIGESTIVE SYSTEM

THE digestive system comprises the mouth, salivary glands, pharynx, tonsils, esophagus, stomach, intestines, liver, pancreas, and, for convenience, the peritoneum.

The subjective symptoms of disease of these organs are summarized elsewhere (Synopsis of Examinations, *q. v.*), with page references to their detailed consideration. The objective examination of some portions (*q. v.*) of the digestive apparatus has already been described—viz., Mouth, Salivary Glands, Pharynx, and Tonsils.

I. THE ESOPHAGUS

Anatomy of the Esophagus.—The esophagus is a muscular tube varying from ¾ of an inch to a scant inch in diameter. It is about 10 inches (25.5 centimetres) long, beginning at the upper border of the cricoid cartilage opposite the disk between the 5th and 6th cervical vertebræ, and, after passing through the diaphragm, ends at the cardiac orifice of the stomach opposite the 10th dorsal vertebra.

Of its relations the following are of special clinical importance: *In the neck*, in front and at the sides with the thyroid gland; at the sides with the recurrent laryngeal nerves. *In the thorax,* in front with the aorta, the left bronchus (between 4th and 5th dorsal vertebræ), and the pericardium; laterally, the pleura and, except at its lower portion, the pneumogastric nerves.

Examination of the Esophagus.—The cardinal symptom of esophageal disease is dysphagia, with or without regurgitation. The object of physical examination is mainly to determine the presence of a stricture or diverticulum.

(1) *Palpation of the esophagus* is possible only in the neck, usually on the left side, behind the trachea. A distinct tumour found here may be a diverticulum distended with food or fluid. A brawny swelling, perhaps with subcutaneous emphysema, is indicative of rupture or perforation of the esophagus with resulting inflammation of the periesophageal tissues which may proceed to suppuration. An abscess in this locality may be due to caries of the cervical vertebræ.

(2) *Auscultation of the esophagus* is occasionally of some diagnostic value. The stethoscope should be placed posteriorly just to the left of the spine about the level of the 6th dorsal vertebra while the patient, at signal, swallows a mouthful of water. At the instant of swallowing a sound (deglutitory) is heard, followed in 6 or 7 seconds by the esophageal *bruit*, a sound like that heard in one's own ears when swallowing saliva. Three to 5 seconds later ensues a secondary sound caused either by the fluid entering the stomach or by regurgitation of air bubbles. If the primary esophageal *bruit* is weak, or delayed longer than 7 seconds, or replaced by a loud splashing or gurgling noise; or if the secondary sound is delayed longer than from 5 to 12 seconds, partial stenosis may be suspected. If the 1st or 2d sound is absent complete stenosis may be suspected. If loud gurgling or bubbling sounds lasting for some minutes are heard at any point along the left side of the spine they may be a result of contractions of the esophagus upon fluid contained in a diverticulum or in the dilated portion of the canal immediately above a stricture.

(3) *Instrumental examination of the esophagus* is made either by the passage of the somewhat flexible but solid bougie or sound, or by the perfectly flexible stomach tube. The technic of introduction will be more conveniently described in connection with the examination of the stomach (page 488). Inspection of the interior of the esophagus has been practised, but at present this method is mainly of academic interest.

The following average measurements are to be remembered in connection with the use of the sound: From the incisor teeth it is 6 inches to the beginning of the esophagus (at the cricoid cartilage); 9 inches to the crossing of the left bronchus; and 16 inches to the cardiac orifice of the stomach. The esophagus is normally somewhat constricted at its beginning, at the crossing of the bronchus, and at its cardiac end.

Obstruction to the passage of the tube may be due, especially in a nervous patient, to spasm of the esophagus, but by waiting a moment the spasm will subside. If the tube passes readily at one sitting and refuses to pass at another it is significant of a diverticulum, which in the first case was empty and allowed the tube to slip by it, and in the second sufficiently distended with food or fluid to engage the end of the instrument and prevent its passage. If the obstruction is found to be permanent, it is necessary to decide whether it is due to the most common cause, narrowing (stricture) of the tube, or to pressure upon it from the outside (aneurism, mediastinal tumour, enlarged bronchial glands).

If the presence of a stricture has been ascertained, one must determine its locality, calibre, and permeability. The *locality* is found by passing the tube to the strictured point, nipping it between the fingers close to the incisor teeth and measuring the distance from the fingers to the end after withdrawal. It is thus possible to decide the practicability of surgical relief. The *calibre* is discovered by using sounds or tubes of varying diameters, finding one which will pass with moderate resistance. Its *permeability* is evidenced by hearing the esophageal bruit as previously described, and by the passage of a somewhat rigid sound, as a very flexible instrument may curve upon itself in the upper dilated portion of the canal. If the stricture is cancerous, which is usually the case in elderly patients, and the tube is fenestrated, small particles of the new growth may be found in the openings. If the tube is bloody, ulceration or erosion is present.

The presence of aneurism of the thoracic aorta is an absolute bar to the use of the sound or tube, and it should rarely, if ever, be employed if there has been recent vomiting of blood.

II. STOMACH

Anatomy and Surface Relations of the Stomach.- stomach lies in the epigastric and left hypochondriac regions. sixths of its bulk is to the left of the median line, one six the right. The larger end, the fundus, fits into the concave left vault of the diaphragm. The important points to be remembered are as follows (Plate I and Fig. 166):

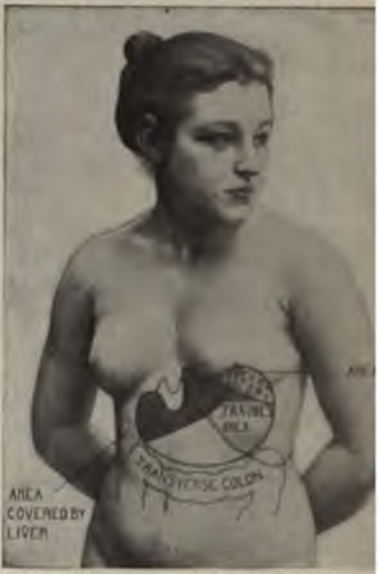


FIG. 166.—This cut shows the shape and t graphical relations of a normal stomach portion of the stomach overlapped by t is indicated by the dotted area, and t tion covered by the liver by the shad Traube's area is that portion which is by the ribs alone.

The *cardiac orifice* (or cardia) is situated directly behind or a little to the left of the sternal junction of the left 7th cartilage, on a level with the body of the 11th dorsal vertebra. It lies 4½ inches distant from the anterior surface of the abdomen. The cardia occupies a relatively fixed position because of its attachment to the esophagus.

The *pylorus* lies between the right sternal and parasternal lines on a level with or slightly below the tip of the ensiform appendix, and corresponds to the body of the 1st lumbar vertebra. It is quite freely movable, passing ward and somewhat to the right, as the stomach becomes dist by air or food.

The *lesser curvature* descends from the cardia, passes transv to the right and then ascends to the pylorus. The two orific closer together, and the lesser curvature is shorter and sharper is sometimes realized.

The *greater curvature,* when the stomach is moderately dist lies 2 to 3 fingerbreadths (1½ to 2½ inches) above the umbilic a level with the infracostal line connecting the lowest points o costal margins. The lower border of a normal but much-dist stomach may be found at the level of the navel. If below the umbilicus, the condition is pathological.

The *fundus* rises as high as the lower border of the left 5th rib in the mammillary line—from 1 to 2 inches higher than the cardia—above and behind the apex of the heart.

The *anterior surface* is for the most part overlapped by the liver, left lung, and certain of the left ribs, leaving a comparatively small extent of its surface in direct contact with the anterior muscular wall of the abdomen. The pyloric end, the lesser curvature, and the cardia lie behind and beneath the quadrate and left lobes of the liver; the fundus and a portion of the body are overlaid by the lower anterior portion of the left lung, and the anterior parts of the 7th, 8th, and 9th left ribs. Traube's space is the area over which the stomach is in direct contact with the ribs just mentioned, and is bounded above by the liver and lung, externally by the spleen, the remaining boundary being formed by the left costal margin. Over this space, as well as the exposed area of the stomach in the epigastric region, pure gastric tympany may be elicited.

The *diameters* of the stomach when moderately distended (SAPPEY) are, between fundus and pylorus, 10 to 12 inches; between lesser and greater curvature, 4 to 5 inches; and between the anterior and posterior walls, 3½ inches. The distance between the cardia and pylorus varies from 3 to 6 inches. The long diameter lies in general transversely, but with a downward inclination from left to right. As the stomach distends (food, fluid, gas) it rotates in such a manner that the lesser curvature is directed toward the spine, the greater curvature moves forward, the anterior surface looks upward and forward, and the pylorus moves to the right.

Physical Examination of the Stomach.—The stomach and its diseases (omitting the anamnesis) are investigated by direct physical examination, and by chemical and microscopical analysis of the stomach contents (Index—Stomach, Examination of contents of).

The principal object of physical examination of the stomach is to determine its size and position, although there are certain other physical signs to be observed which do not bear directly upon the dimensions and location of the organ. The methods are as follows:

(*a*) **Inspection.**—A distinct bulging in any part of the abdomen except the epigastric region may be due to a dilated stomach. A swelling due to this cause is most frequently seen in the hypogastric or the umbilical region, while the epigastrium is notably hollow and transversely depressed. As the cardia is the comparatively immobile point of attachment of the stomach to the diaphragm through the medium of the esophagus, the descent of the enlarged or prolapsed organ increases toward the pylorus. With the patient in the recumbent p ture, a marked concavity between the costal arches, extending from t ensiform appendix to or below the umbilicus, with perhaps a vertic median sulcus wider above than below, the abdomen being as a wh flattened in the central portions and bulging in the lateral regio is significant of prolapse of the stomach (gastroptosis). In the ere position the epigastric region becomes still more depressed, while t umbilical and in particular the pubic regions bulge outward (Fig. 168 Inspection may also show peristaltic waves passing from left to right (rarely reversed) along the dilated viscus (Fig. 167).

(*b*) **Palpation.**—This determines the existence of tenderness, swelling or tumour, and succussion or splashing.

Tenderness (*q. v.*), more or less general, is common in acute and chronic inflammations of the stomach. It is a sign of gastric ulcer only when permanently and very strictly localized.

Tumour of the stomach, if found in an elderly person, is usually cancerous, most commonly affecting the pyloric extremity and generally situated a little to the right of the median line between the ensiform appendix and the umbilicus. It may be extremely mobile and capable of displacement into almost any one of the abdominal regions. But if it has contracted adhesions to fixed organs or tissues it may be quite firmly moored; or, if adherent to the liver or diaphragm, may move with respiration. In younger persons a tumour here may be the result of hypertrophy of the pylorus. A palpable diffuse thickening (perigastritis) of a portion of the stomach wall may be found as a consequence of chronic gastric ulcer. If the abdominal walls are thin and relaxed and the stomach prolapsed, it may be possible but not likely for the pancreas to be felt and mistaken for a tumour of the stomach (EWALD), and a similar mistake may arise from the finding of a small lymphatic gland in the gastrocolic ligament, at the middle of the greater curvature. If the stomach is dilated and the abdominal walls are thin and relaxed, it may be practicable to feel the lower border of the enlarged viscus.

If fluid and air are present in the stomach, splashing or succussion sounds or sensations may be elicited by placing one hand over the lower ribs posteriorly, while with the other hand a series of sudden, quick pressures are made over various points of the abdomen. If splashing is found as long as 3 hours after a meal, particularly if it exists below the umbilicus, one may suspect the presence of a dilated or prolapsed stomach. There is, however, one source of error in that similar sounds may be elicited from the transverse colon or other portion of the intestine. If this question arises the colon should be emptied by a high enema or a laxative.

(*c*) **Percussion.**—As a rule, this is not trustworthy, mainly because of the fact that the percussion note of the surrounding intestines, in particular the colon, so closely resembles gastric tympany as to be indistinguishable from it. Moreover, the colon, if distended, may overlie the adjacent edge of the stomach and thus render it impracticable to determine with any accuracy the position of the lower border. The degree of distention of the stomach, whether the distention is due to fluid or solid material (dulness), or gas (tympany), and the fact that the stomach is overlapped by the liver (which may be enlarged) and left lung are additional sources of uncertainty. If the stomach is considerably distended by gas, percussion may be of some service.

The position of the lower border of the organ may be determined by causing the patient to drink measured quantities of water (Dehio), 7 or 8 ounces at a time, while in the erect position, and then percussing. The fluid in the stomach causes a line of dulness, indicating the position of the lower border, and each additional quantity of water ingested broadens the line of dulness and lowers the border ¾ to 1¼ inch. Normally the lower border is never found below the umbilicus. If it is discovered below the navel, either dilatation or prolapse, or both, exist. The distinction must be made by ascertaining through inflation whether the pylorus and lesser curvature have or have not shared in the descent.

Ordinary percussion enables one to determine the upper and left borders of the exposed gastric tympany, less perfectly the outline of the fundus and lesser curvature, which are overlapped by the liver and lung. Practically this is accomplished by the delimitation of the lower border of the left lung (*q. v.*), the lower border of the left lobe of the liver (*q. v.*), and of the spleen (*q. v.*).

The average normal area of gastric tympany is shown in Fig. 166. An increase in its extent, mainly in a vertical direction, will be found if the stomach is distended by gas (common in general meteorism), and if the left lobe of the liver is shrunken or the left lung retracted by pleurisy or fibroid changes, thus allowing the stomach to lie in contact with a larger surface of the abdominal or lower left thoracic wall. On the other hand, this area is made smaller by enlargement of the left lobe of the liver or of the spleen, and the tympanicity of Traube's space is abolished by a left pleural effusion.

(*d*) **Auscultation of the Stomach.**—There is little of definite diagnostic value to be obtained from auscultation of the stomach. The sounds which may be heard and the significance which they may possess are as follows:

(1) The sounds caused by the *swallowing of fluid* have been pre-

viously described as heard upon auscultation of the esophagus (*q. v.*). Upon listening over the notch between the ensiform appendix and the left costal margin the same sounds may be heard—viz., at the instant of swallowing, the deglutitory sound (which is not often heard over the stomach); the esophageal bruit, 6 or 7 seconds after the act of deglutition; and the secondary murmur, 3 to 5 seconds later. The absence of the latter two sounds, if demonstrated, may be of some significance as indicating esophageal stenosis, or possibly also a lack of motor power in the deglutitory muscles.

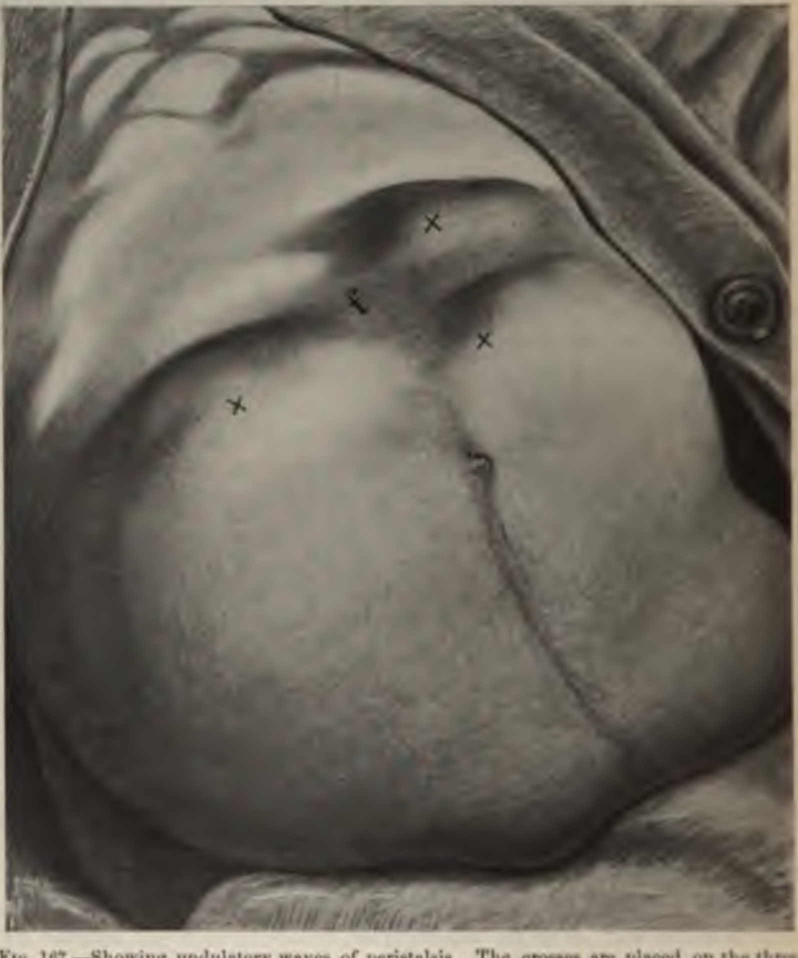


FIG. 167.—Showing undulatory waves of peristalsis. The crosses are placed on the three prominent waves. The letter *f* indicates the depression on the lesser curve. (Redrawn from Osler.)

(2) *Crackling* or *fizzing sounds* may be heard over the stomach, and when detected constitute positive proof of active fermentation and stasis of food. The sounds arise from the bursting of bubbles of gas.

(3) *Succussion sounds* (*q. v.*) have been described as audible at a distance from the patient. The same variety of sounds of less intensity may be heard by direct auscultation upon shaking the trunk from side to side, or upon voluntary change of position of the patient. While these are often audible in the normal stomach, if they are present at times when the organ should be empty it is presumptive evidence of loss of motor power (atony) or dilatation with retention.

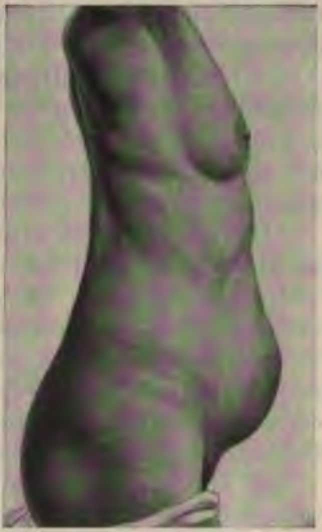


FIG. 168.—Showing marked gastroptosis.

The *splash sound* is similar to the succussion sound except that it is elicited, the patient recumbent, by short, pushing movements of the finger-tips against the stomach region. This sound can, at times, be obtained in a stomach which is quite normal, both in size and position. Particularly is this the case when an unusually large amount of fluid, a quart or more, has been ingested within a short time. In very thin persons, or in those possessing abdominal walls of unusual laxness, the splash sound may often readily be demonstrated. In neither of these instances, however, will the splash sound be found at any point below a line passing horizontally through the umbilicus.

On the other hand, if the stomach has been found to be in its normal position, and the splash sound can be elicited for some distance below the horizontal umbilical line, it must be considered a pathological finding, provided, always, that it occurs three hours after a small meal, or seven hours after a large meal. If it has been demonstrated that the stomach is prolapsed, the splash sound may be found considerably below the horizontal level of the umbilicus, but its presence under such circumstances does not imply an impairment or a deficiency in the power of motility of the viscera. If, finally, it can be elicited by *gentle* tapping over a large area (epigastrium to pubis), motor insufficiency is undoubtedly present.

(4) Loud *rumbling* or *gurgling* sounds or rasping vibrations which are often synchronous with respiration, are not infrequently heard, even at some distance away. They are caused by the respiratory rise and fall of fluid. Such sounds are especially liable to occu in gastric dilatation or prolapse, or in the wearers of tight corsets.

(5) The *sounds of the heart* may be heard over a gas-distende stomach (acting as a resonating chamber), and are encountered particularly in gastric dilatation. In such cases the sounds possess ringing, reverberating quality quite foreign to their usual characte

(*e*) **Inflation or Ballooning of the Stomach.**—Probably the bes method of determining the size, shape, and position of the stomach including an accurate outline of the lesser and greater curvature and pyloric extremity, is that of inflation. There are two ways o inflating the stomach, of which the second is by far the better.

(1) The *first* is to administer in solution 1 drachm of sodiu bicarbonate, followed immediately by the same amount of tartari acid, also in solution. The carbon dioxide evolved promptly distend the stomach. This procedure is open to the very serious objectio that the evolution of the gas is not under control. Thus the stomac may be dangerously overdistended, causing hemorrhage or cardia embarrassment, or there may not be sufficient gas to fully balloo the organ, and its full size and shape fail to be shown.

(2) The *second* manner of inflation is to introduce the stomac tube, and attach to its external end by a bit of glass tubing the bul of a Davidson syringe. Air should then be pumped into the stomach at first vigorously, in order by somewhat sudden distention to caus the pylorus to contract and thereby prevent the passage of the air int the intestines (Pepper, Stengel). The amount of air injected is unde control, and it may be allowed to escape immediately if unpleasan symptoms should appear. If an inflating bulb of known capacit (e. g., a Politzer bag) be employed, the size of the stomach may b roughly estimated by the number of cubic inches of air injected.

As the stomach is distended its outlines become apparent throug the abdominal walls and it is comparatively easy to determine it general size, shape, and position, as well as the more important details relating to the position of the upper and lower borders and th pylorus. If, because of great thickness of the abdominal walls, th stomach does not become manifest to inspection (a rare occurrence i cases requiring this method of examination), inflation greatly facil tates either ordinary or auscultatory percussion.

Inflation should not be employed in very feeble patients, or aft recent hæmatemesis, or where there is a suspicion of gastric ulcer, — in cases of organic cardiac disease.

(*f*) **Gastrodiaphany and Gastroscopy.**—Gastroscopy can be set aside with the bare mention, as the procedure demands unusual tolerance on the part of the patient and special training on the part of the examiner, as well as costly apparatus. Mikulicz was able to demonstrate malignant neoplasms by direct observation, but apart from his personal experience the method has received little attention.

Gastrodiaphany, on the other hand, is comparatively easy. The apparatus devised by Einhorn is a thoroughly practical one, consisting of a small electric light at the end of a rather stiff bougie. In a dark room, with the patient standing, such a light, after being placed in the stomach, produces areas of transillumination (Figs. 169, 170, 171, and 172). Much has been claimed for this method in the differential diagnosis of gastroptosis and gastrectasia, and neoplasm of the anterior stomach wall. There are, however, a number of sources

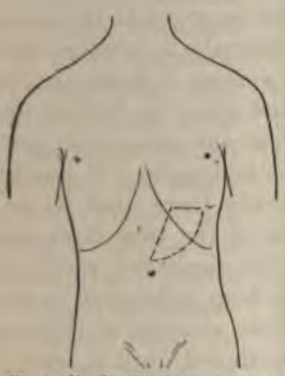


FIG. 169.—Gastrodiaphanic picture in normal stomach (Ewald).

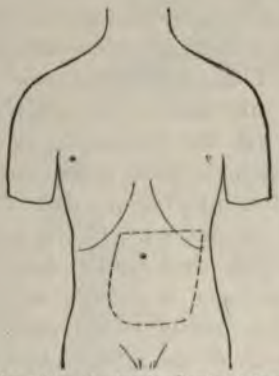


FIG. 170.—Gastrodiaphanic picture in dilated stomach (Ewald).

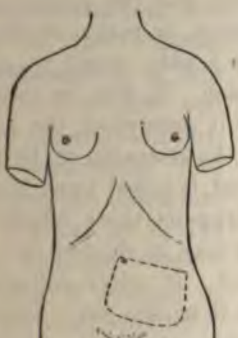


FIG. 171.—Gastrodiaphanic picture in gastroptosis (Ewald).

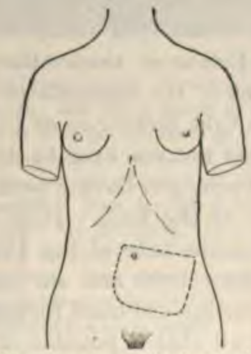


FIG. 172.—Gastrodiaphanic picture in gastroptosis (Ewald).

of error, e. g., intervening coils of intestine, distended colon, and thick abdominal parietes.

(*g*) **Obtaining the Stomach Contents.**—(1) *Technic.*—The method is as follows: Provide a soft-rubber stomach tube having two or more openings in its lower end. Into the upper end a piece of glass tubing, 3 or 4 inches long, is inserted. Have also a pitcher of hot water, a wide-mouthed 6 or 8 ounce glass bottle, a sheet, and a Politzer bag (seldom required). Place the tube in the warm water, let the patient sit upright, pin the sheet around the neck so that it covers the front of the body, and let him hold the bottle in his left hand. Take the tube from the water, request the patient to bend the head slightly forward and open the mouth, but not to protrude the tongue. He should be asked, and admonished at intervals, to breathe steadily and deeply. Then, *without at any time putting a finger in the mouth*, pass the tube back to the pharynx, and when its further progress is arrested, ask the patient to swallow, at the same time pushing the tube farther in, when it will enter the esophagus. It should now be rapidly fed into the mouth. Practically there need be no fear of entering the larynx. If the progress of the tube is arrested, it is probably due to muscular spasm of the esophagus, which will relax after a moment of waiting, or after a deep inspiration or attempt to swallow; or, rarely, to a diverticulum or stenosis of the esophagus. The passage of the tube should be continued until at least 17 or 18 inches of its length, measured from the incisor teeth, and indicated by a ring imprinted upon the tube, has been introduced. The glass tube should then be placed in the bottle and the patient asked to hold his breath and strain as in defecation, or make an attempt to vomit. Either of these efforts will, as a rule, result in successive expulsions of the contents of the stomach. It may be necessary to push the tube farther in or to withdraw it somewhat, the end of the tube either not reaching to the fluid in the stomach, or, by reason of too great a length having been introduced, is coiled upon itself above the level of the fluid. If, as seldom happens, these efforts are unsuccessful, the tube of the Politzer bag may be slipped over the end of the glass tube and suction exercised. If saliva collects in the mouth, incline the head forward and let it trickle out. After having obtained all that is possible of the gastric contents, the finger should be placed firmly upon the end of the glass tube, the rubber tube withdrawn and its contained fluid allowed to run into the bottle.

Until tolerance has been established, the earlier introductions of the tube usually cause more or less violent efforts to vomit. Steady, regular deep breathing is most likely to diminish such attempts. Passing cyanosis, due to the temporary cessation of respiration, is sometimes encountered, but very rarely persists sufficiently to require withdrawal of the tube.

(2) *Contraindications.*—Certain contraindications to the use of the tube, largely because of the strain and effort of vomiting, must be recognised. The conditions to be considered as an absolute bar to its employment, subject in part to the exercise of judgment and the dictates of experience, are as follows:

Marked prostration, continued fevers, or cachectic states; broken compensation of heart lesions, advanced fibrous or fatty degenerations of the heart, and angina pectoris; thoracic aneurism and advanced general arteriosclerosis; last stages of pulmonary tuberculosis, emphysema, bronchitis, and recent hæmoptysis; gastric ulcer with recent hæmatemesis or tarry stools; recent intracranial, renal, vesical, or rectal hemorrhages; and pregnancy and old age.

Interpretation of the Results of the Physical Examination of the Stomach.—The size and position of the stomach having been ascertained and marked upon the abdominal wall, it remains to decide from the findings whether the stomach is normal in size and position, whether it is nearly normal in size but is prolapsed (*gastroptosis*), or whether it is dilated (*gastrectasia*). Some degree of prolapse usually accompanies dilatation.

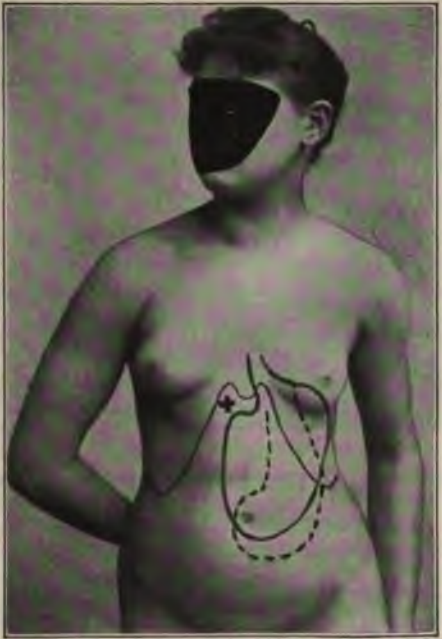


FIG. 173.—Broken line shows prolapse of the stomach (gastroptosis). Note vertical position of stomach; also the situation of the pyloric end at or near the umbilicus as compared to its normal location (indicated by the cross). Solid line shows dilatation of the stomach (gastrectasia). Note descent of lower border to or below the umbilicus, while the pyloric end lies a relatively short distance below the normal point. Note also the much increased distance between pylorus and the most dependent portion of the lower border.

(1) **The Normal Stomach.**—The normal positions of the lesser and greater curvatures and the pylorus have been previously described and are shown in Fig. 166.

(2) **Gastroptosis.**—If the pyloric end is low down (Fig. 173), so that while the lower border is below the umbilicus the lesser curvature and pylorus are *correspondingly depressed* (to a point near the umbilicus), the organ as a whole taking a decidedly vertical position, prolapse of the stomach may be assumed to be present.

(3) **Gastrectasia.**—If, on the other hand, the pyloris and lesser curvature are found to have descended only to a relatively slight extent below their normal position, while the lower border lies below the umbilicus (Fig. 173), dilatation is present. It will be noted that the descent of the greater curvature affects mainly its pyloric (and most dependent) half, and that in consequence the distance between this part of the lower border and the pylorus is greatly increased.

It is quite evident that a determination of the site of the lower border alone is not sufficient to discriminate between prolapse and dilatation, but that a knowledge of the position of the pylorus and lesser curvature is an absolute requisite. It must also be borne in mind that in some individuals the stomach is normally much larger than the average (*megastria*, *megalogastria*), and a diagnosis of pathological dilatation can not be made unless there are coexisting evidences of motor weakness and stagnation of food (Index—Stomach contents, examination of).

III. INTESTINES AND PERITONEUM

Topographical Anatomy of the Intestines.—(1) **Small Intestines.**—The jejunum and ileum occupy in general the central portion of the abdomen. The coils of the *ileum* lie in the left lumbar, left iliac, and left half of the umbilical regions. The coils of the *jejunum* are found in the right half of the umbilical, right lumbar, right iliac, and the pubic regions. As the coils are extremely movable these boundaries are ill-defined and of little value.

(2) **Large Intestine.**—The colon occupies the periphery of the abdomen, and by comparison with the small intestine remains in a relatively fixed position (Fig. 174).

The middle of the lower border of the *cæcum* (its apex) lies at the centre of a line drawn from the anterior spine of the ilium to the symphysis pubis. The ileum joins the cæcum on its inner and posterior aspect, about 3 inches internal to the anterior spine.

The *appendix vermiformis* is found in the right iliac region. Its base lies at the middle of a line drawn from the anterior spine to the umbilicus, corresponding to the right edge of the rectus muscle (McBurney's point). It is attached close to the ileocæcal valve on the inner and posterior side of the cæcum, and the upper portion of the appendix is therefore concealed by the latter. The appendix may lie in any radius of a circle having this point as a centre. In the majority of cases it passes either downward, outward, or inward; in a minority upward. It averages 3½ to 4 inches in length, with the diameter of a goose quill.

The *ascending colon*, running upward on the right side from the comparatively superficial cæcum, sinks back against the posterior abdominal wall and may be partly overlaid by the small intestines. It passes up to the under and concave surface of the liver, by which it is sheltered, and then turns sharply to the left, forming the *hepatic flexure*.

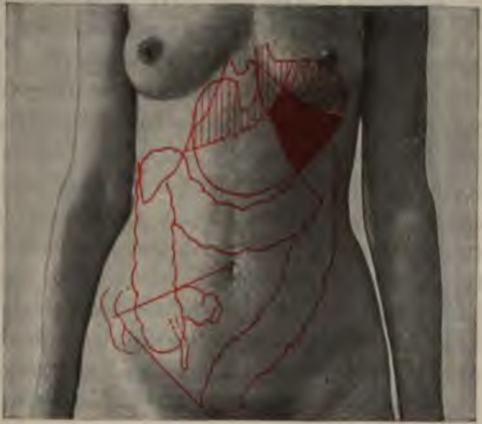


FIG. 174.—Showing the topographical relations of the colon and vermiform appendix (semidiagrammatic). Note (1) the line from the anterior superior spinous process to the symphysis pubis and the relation of the apex of the cæcum thereto; (2) the relation of the ileo-cæcal junction and the base of the appendix to the line running from anterior superior spine to umbilicus; (3) the concealment of the hepatic and splenic flexures of the colon by the costal arches; and (4) the relation of the transverse colon to the stomach above and the umbilicus below.

The *transverse colon* passes forward and downward to the left until its lower border lies at the level of the umbilicus, then curving upward and backward it disappears under the left costal arch and behind the fundus of the stomach to form the *splenic flexure*, which lies at a higher point than the hepatic flexure.

The *descending colon* then proceeds downward on the left, terminating in the *sigmoid flexure* in the left iliac region. It may be noted that the cæcum, the transverse colon, and the upper part of the sigmoid flexure are quite superficial, while the hepatic and splenic flexures lie deeply under the protecting costal arches.

Examination of the Intestines and Peritoneum.—The principal symptoms and signs of disease of the intestines and peritoneum have been already discussed under the heads (*q. v.*) of Vomiting, Constipation, Diarrhœa, Pain, and General Examination of the Abdomen. Certain points, however, may here receive consideration.

(1) **Inspection of Intestines.**—If the colon is distended by gas its course, with the exception of the hepatic and splenic flexures, may be well defined. In such a case the ascending and descending por-

tions are seen as elongated swellings in either lateral region of the abdomen, while its transverse portion becomes prominent at or just above the umbilicus. The cæcum is the largest in diameter, and therefore the most distensible, of any portion of the large intestine. The junction of the sigmoid flexure and the rectum is the narrowest and the most liable to stricture. The visible "patterns of tumidity" in intestinal obstruction have been considered (page 461).

(2) **Palpation of Intestines.**—By touch one discovers an accumulation of fæcal matter in the intestine, known by its doughy feeling and the fact that the mass may be indented by pressure. It is stated that under certain circumstances a crepitating or "sticky" sensation may be perceived as the palpating fingers relax their pressure. This is due to the separation of the intestinal wall from a fæcal lump, but, as this sign requires for its production that the intestine should be sufficiently distended with gas to effect such a separation, as well as the proper degree of adhesiveness in the fæcal mass, it must be of limited availability. Fæcal accumulation occurs most frequently, perhaps, in the cæcum and ascending colon, often also in the sigmoid flexure. Gallstones and enteroliths are sometimes encountered. Their hardness and mobility and the history of the case must be depended upon for differentiation. It is to be remembered that, owing to the close relation of the hepatic flexure to the liver and gall bladder, a gallstone may ulcerate through into this portion of the large intestine.

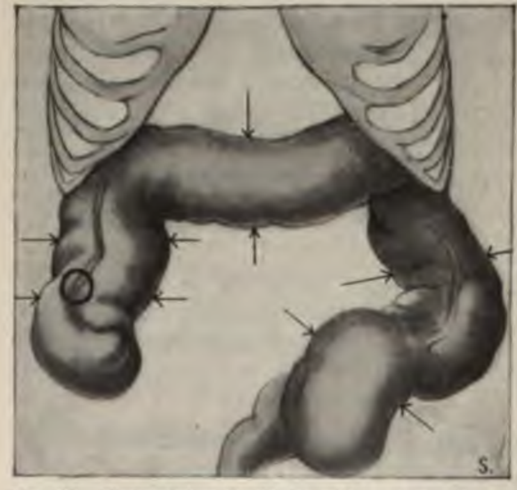


FIG. 175.—Determining the limits of the colon by auscultatory percussion. Arrows show the lines along which percussion should be conducted; the circle the point at which the stethoscope is placed.

Gurgling on pressure in the right iliac fossa occurs in typhoid fever, but is met with too often in healthy individuals to be of much diagnostic value. Volvulus affects most commonly the sigmoid flexure, because of the very considerable mobility of the latter.

(3) **Percussion of Intestines.**—Ordinary percussion is of little or no value in delimiting the large from the small intestine, the tym-

panitic quality of one resembling that of the other too closely for discrimination.

If, however, the colon is empty of solids, and distended with air or gas, it is occasionally practicable to ascertain its size and course by auscultatory percussion. If required, the colon may be emptied by irrigation and inflated by pumping air into it. It is requisite that the small intestines should not be excessively ballooned, as otherwise their percussion sound is too much like that of the colon to be clearly separated. To outline the colon by this method, place the stethoscope over the cæcum (Fig. 175). Begin percussion at a point midway between umbilicus and symphysis, and carry it to right, to left, and upward, until in each direction the greater intensity, heightened pitch, and altered quality of the sound announce that the inner limit of the colon has been encountered. Then percuss from the epigastrium downward, and from each lateral lumbar region inward toward the centre of the abdomen, to determine the outer limits of the transverse, ascending, and descending portions of the colon.

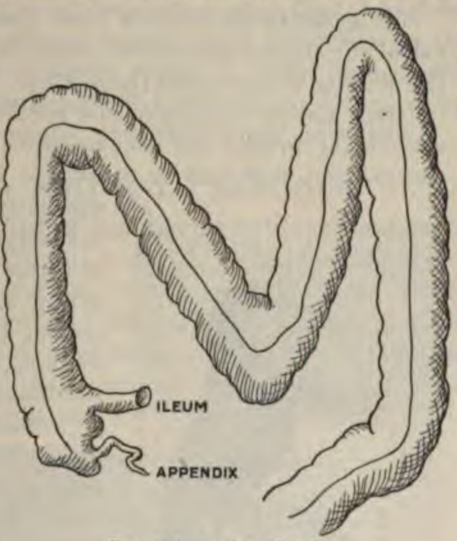


Fig. 176.—V-shaped colon.

Aside from congenital anomalies in the position of the colon discovered by operation or autopsy, displacement may occur as a part of a prolapse of one—e. g., gastroptosis—or several of the abdominal viscera (visceroptosis, splanchnoptosis). Such a displacement largely affects the transverse colon, the middle of which descends below the navel so that it assumes a V shape (Fig. 176), with consequent kinking at the hepatic and splenic flexures, and perhaps obstinate constipation, meteorism, and other symptoms of obstruction. In one such case of my own, exploratory cœliotomy was done, by the advice of a most competent surgeon, and this condition of affairs was found.

It is claimed (Stengel) that it is possible by auscultatory percussion to determine that a tumour found to lie in the previously ascertained course of the intestine originates in the wall of the colon. The stethoscope is placed over the colon near to the tumour, and

percussion made over the tumour and toward it from every
The note over the tumour (*C*, Fig. 177), if the latter is c
with the colon, will resemble much more nearly than th
(over the small intestines) the note *A* over the colon.

(4) **Auscultation.**—Aside from auscultatory percussion
use of the stethoscopic examination of the intestines is to d
the activity of peristalsis (page 479).

Here may be mentioned the condition of flatulence or fl
a tendency toward the accumulation of gas in the stoma
testines, not sufficient to constitute tympanites (*q. v.*), an
associated with increased p
borborygmi, belching, and the
of flatus *per anum*. The gas m
monly arises from fermenta
cesses; sometimes it is swallo
its rapid passage into the intes
the blood is, perhaps, possible
of sudden flatulency in cert
roses. Flatulency is a sympto
tritis and of gastric neurose
very common in hysteria, and
associated as a direct result
eating of indigestible or fer
food.

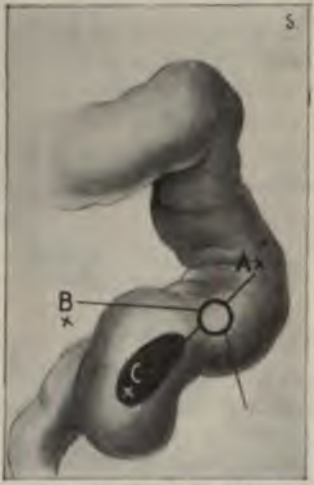


Fig. 177.—To show the method of determining, by auscultatory percussion, that a tumour belongs to the colon. If the tumour involves the wall of the colon the percussion notes over points *A*, *B*, and *C* differ, but *C* resembles *A* much more nearly than *B*, over small intestine, resembles *A*. Circle = chest piece of stethoscope.

(5) **Examination of the Rec**
spection of the *anal region* m
condylomata, ulceration, fissur
rhoids, or pruriginous eruptio
by causing pain, insomnia, o
blood, may be responsible fo
thenic or anæmic conditions.
of the rectum, if found, may
cant of pertussis, prolonged
the presence of scybalous mass
presence of other causes of rec
mus (*q. v.*).

Digital examination of the *rectum* may not only afford
diagnostic information with reference to the causation o
general conditions as just described, but in addition may
the diagnosis in various local lesions which are usually first
tered by the internalist. The latter are: Impaction of th
with dry and hard scybalous masses, or impacted gallstone
eign bodies; the tumour of appendicitis, which may be felt

appendix is unusually long and depends into the pelvis, usually to the right; the tumour of intussusception or a bleeding polypus; and malignant tumour or infiltration of the rectum causing bloody mucous diarrhœa and enlarged secondarily infected lymph glands.

For the examiner the osmic unpleasantness of a digital exploration of the rectum may be almost entirely done away with by the use of a cot for the examining finger, made of rubber tissue, sufficiently thin to allow an almost unimpeded sense of touch (DELATOUR). The cotted finger may be lubricated and washed as if uncovered.

IV. THE LIVER AND GALL BLADDER

Topographical Anatomy of the Liver.—The general shape of the liver is that of a wedge. It lies with its base or thick end in the right hypochondriac region. Its *upper surface* fits neatly into the vault of the diaphragm; its *lower surface* rests upon a bed composed of the stomach, duodenum, transverse colon, and small intestines. Its *anterior*, *lateral*, and *posterior aspects* are in relation for the most part with the abdominal wall and lower right ribs.

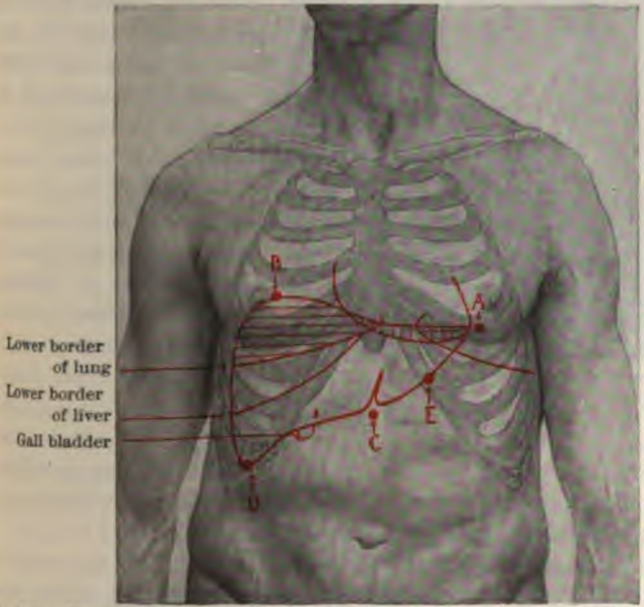


FIG. 178.—Showing the points which determine the size and position of the normal liver. Horizontal shading = portion of liver overlapped by lung; vertical shading = portion of liver overlapped by heart. Compare with Fig. 180.

(1) **Upper Limits of Liver.**—Mark a point (*A*, Fig. 178) at the lower edge of the left 5th rib, between the parasternal and mammil-

lary lines (about 2 or 2½ inches to left of left edge of sternum). Mark a second point, *B*, in the right 4th interspace in the mammillary line. From *B* draw a line to the left convex upward, but curving down to the base of the ensiform cartilage, from which it is prolonged to *A*. From *B* draw also an almost horizontal line to the right and posteriorly, cutting the midaxillary line in the 7th space and the scapular line in the 9th space, to the midspinal line. The entire line, front, side, and back, corresponds to the *upper limit* of the liver.

(2) **Lower Limits of Liver.**—Mark a point, *C*, in the median line, a handbreadth (about 3½ to 4 inches) below the base of the ensiform cartilage. This will lie somewhat above the halfway point between the ensiform appendix and the umbilicus. Mark another point, *D*, at the lower edge of the 9th right costal cartilage, and a third, *E*, at the edge of the left costal arch, on a level with the lower border of the 6th rib. Draw a line from point *D* upward and to the left, through point *C*, at which the interlobar notch should be indicated, then bending upward through point *E* to point *A*. From point *D* draw a line backward to the right, cutting the 10th interspace in the midaxillary line (Fig. 179), from which it joins the spine at the level of the 11th rib. The entire line, front, side, and back, corresponds to the *lower limit* of the liver.

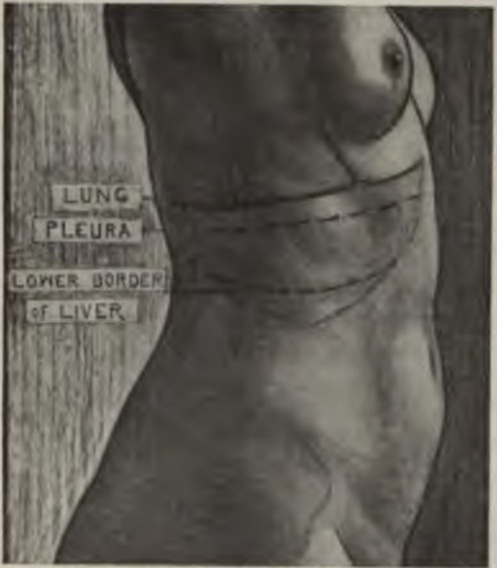


Fig. 179.—Showing the surface relations of the lung, pleura, and liver in the midaxillary line. Note the manner in which the complementary pleura overlaps the liver.

One may note that the left lobe lies to the left of the median line and extends nearly to the left nipple; the interlobar notch lies in the median line; the liver, in the right mammillary line, extends vertically downward from a point just below the nipple to the costal margin; and that the lower border of the liver on the right corresponds nearly to the costal edge. In the aged the lower edge of the liver may lie one space above the indicated limit; in infants and children it projects about an inch below the edge of the ribs; in the erect position the liver descends half an inch below the costal arch.

(3) **The Gall Bladder.**—This pear-shaped organ lies just internal to the 9th right costal cartilage and close to the outer edge of the right rectus (Fig. 178). A line drawn from the right acromion process to the umbilicus crosses the costal arch about at the point where the gall bladder lies.

Physical Examination of the Liver and Gall Bladder.—Certain cardinal symptoms (*q. v.*) of disease of the liver have been considered elsewhere—jaundice, ascites, stools, pain, and tenderness.

The direct physical examination of the liver is intended mainly to ascertain its shape, size, position, consistence, the character of its accessible surface, and the presence of tumours. Taken in order of inverse importance, the methods are:

(*a*) **Inspection of the Liver and Gall Bladder.**—Very rarely one can detect by careful inspection and a good light the lower edge of an enlarged liver showing as a distinct linear prominence and moving up and down with respiration. More frequently it is possible to see pulsation of the liver (*q. v.*). Swelling or enlargement of the viscus, if very considerable, may cause a visible fulness of the right hypochondriac region with bulging of the ribs.

(*b*) **Auscultation of the Liver and Gall Bladder.**—One may hear the friction sound of a perihepatitis over the right hypochondriac region, or between the upper and lower liver lines laterally and posteriorly. The very infrequent venous hum or murmur in enlarged veins of the liver or in cases of tricuspid regurgitation has been considered. When palpating a gall bladder containing a number of concretions, it may be possible, by the simultaneous use of a stethoscope placed close to the sac, to hear the resultant rubbing or grating contact of the stones.

(*c*) **Percussion of Liver and Gall Bladder.**—In order to percuss the liver over the *front* and *side*, the patient should be lying down; to percuss *posteriorly*, sitting or standing.

The upper portion of the right lobe of the liver is overlapped by the right lung, and a very small area of the left lobe is similarly covered by the heart and left lung (Fig. 178), the remainder being in direct contact either with the ribs or the anterior wall of the abdomen. As percussion over the covered portion of the liver affords an impaired pulmonary resonance or *modified dulness*, because of the interposition of the lung between the liver and thoracic wall, and similar percussion over the portion which is in parietal contact gives *absolute dulness*, one recognises clinically two areas. The first is the *deep*, *relative* or, preferably, the *covered* hepatic dulness; the second, the *superficial*, *absolute*, or *exposed* dulness; the two together, the *entire* hepatic dulness.

In percussing the liver (Fig. 180) it is necessary to determine (1) the upper limit of covered dulness, (2) the upper limit of exposed dulness, and (3) the lower limit of exposed dulness. Percuss downward, first in the *mammillary line*, beginning at the second interspace, then in the *midaxillary line* from the fourth interspace, finally in the *scapular line* from angle of scapula. Then percuss from below upward—in the *middle line anteriorly* from the umbilicus; and from other points, lateral and posterior, below the ribs.

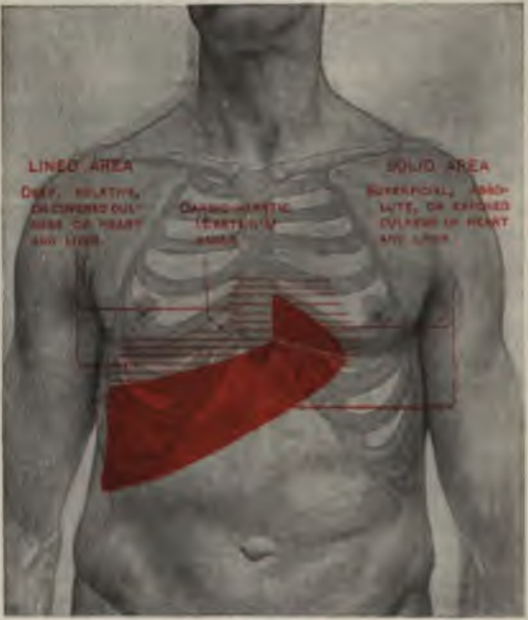

LINED AREA
DEEP, RELATIVE, OR COVERED DULNESS OF HEART AND LIVER
CARDIO-HEPATIC
SOLID AREA
SUPERFICIAL, ABSOLUTE, OR EXPOSED DULNESS OF HEART AND LIVER

FIG. 180.—Showing the results of percussion over a normal liver (and heart).

Ordinary Percussion.—(1) Upper Limit of *Covered* Hepatic Dulness. — Starting with the pure pulmonary resonance of the upper thorax and using *strong* percussion, a careful watch is kept in passing down for the first trace of impaired resonance which denotes the presence of the liver. This point is normally found in the 4th space in the mammillary line, the 7th space in the midaxillary line, and the 9th space in the scapular line. By comparing rib with rib and interspace with interspace, it may be quite accurately located.

(2) Upper Limit of *Exposed* Hepatic Dulness.—Passing down from the upper limit of covered dulness with *gentle* strokes, absolute dulness will be met with under normal circumstances—in the mammillary line at the 6th rib; midaxillary, 8th rib; scapular, 10th rib. The ascertainment of the upper limit of the exposed hepatic dulness is practically the determination of the lower border of the right lung, the respective limits coinciding. Note that, in the midsternal line, the demarcation between the exposed heart and liver dulness cannot, as a rule, be determined. By auscultatory percussion it is occa-

sionally practicable. It may be obtained, usually with sufficient correctness, by drawing a line from the cardio-hepatic angle, the junction of the dulness of the right border of the heart with the upper limit of hepatic dulness, to the apex of the heart.

(3) Lower Limit of Hepatic Dulness.—*Gentle* percussion down and up along the lines previously indicated will enable the line to be drawn between liver dulness and tympanitic resonance. The *lower limit* is: in the *median line anteriorly*, 3½ to 4 inches (a handbreadth) below the ensiform appendix; in the *mammillary line*, as a rule, the costal margin; in the *midaxillary line*, the 10th space; in the *scapular line* it fuses with the dulness of the right kidney. It may be difficult to find the lower limit in the median line in front because of the thinness of the left lobe and the dulling effect of the muscular masses of the recti.

The vertical width of hepatic dulness is normally: in the mammillary line, 4 inches; in the midaxillary, 6 inches; in the scapular, 3 inches. In elderly persons it is about 1 inch less. In percussing over the exposed portions of the liver a sense of resistance may be perceived, particularly if the viscus is enlarged or its consistence increased.

Percussion of the gall bladder is possible only when it is considerably distended or enlarged, in which case it affords an area of dulness projecting downward and inward from the lower hepatic border and continuous with the dulness of the latter. In some instances the transverse colon may become looped over the neck of a full gall bladder, lying between it and the liver, separating its dulness from that of the liver by a tympanitic interval.

Auscultatory Percussion of the Liver.—The limits of the liver can be ascertained with sufficient correctness by ordinary percussion, but if unusual accuracy is required, or if the origin of a tumour near, and perhaps belonging to, the liver is to be determined (STENGEL), auscultatory percussion may be employed.

(1) To determine the limits of the liver by this method the stethoscope should be placed over the middle of the liver area, anteriorly, laterally, and posteriorly, in turn, while percussion is made toward it from above and below, following the lines previously described for ordinary percussion, the sounds being judged by the usual rules.

(2) To determine whether or not an adjacent tumour is connected with the liver, the stethoscope is placed over a nearby portion of the organ (Figs. 181 and 182), and percussion made from several directions toward and upon the tumour. If the latter is an outgrowth from the liver, the note over it will resemble in intensity and quality that which is obtained from the liver itself, the connecting tissue

between tumour and liver conducting the sound without interruption. If, however, a tumour originating outside of the liver has contracted extensive adhesions to it, or has come to involve the liver as well as the adjacent organ in which it first began (e. g., stomach), it will be impossible to determine which viscus is the more involved.

(*d*) **Palpation of Liver and Gall Bladder.**—For palpation of the liver the patient should be in the recumbent position, with the head and shoulders somewhat raised and the knees flexed in order to relax the abdominal muscles. Sitting, preferably at the right side of the patient and facing the couch, the (warmed) right hand is laid flat upon the abdominal wall below the right costal arch, the fingers pointing upward and somewhat toward the median line, their tips resting just outside of the border of the right rectus in order not to mistake the uppermost linea transversa for the edge of the liver.

Seek first to find the *lower edge* of the organ. Depress the fingers and upper border of the hand so as to push up a fold of skin, feeling at the same time for the resistant hepatic edge. Cause the patient to take a series of deep respirations, pressing the fingers inward toward the end of expiration, when, if the edge of the liver is palpable, it may be felt to slip under the fingers, as it moves on the average half an inch up and down with the respiratory movements. As the liver may be greatly enlarged, its lower edge must be sought for at different points from below the

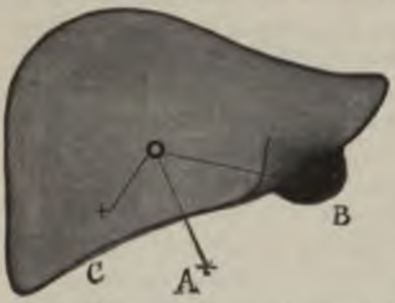


FIG. 181.—If a nearby tumour is connected with the liver, percussion note *B* (auscultatory) is more intense and clear than note *A*, resembling *C* (over liver) in character. Circle = mouth of stethoscope.

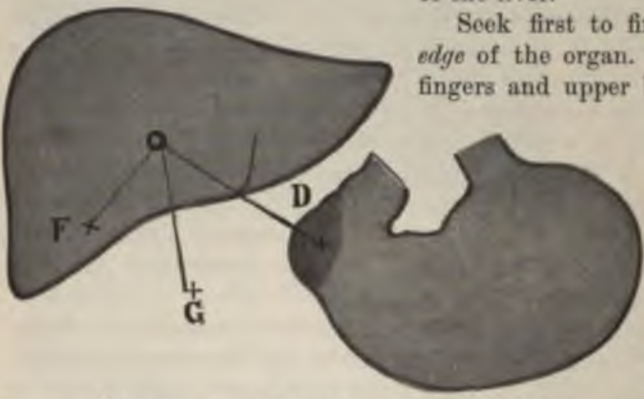


FIG. 182.—If the tumour (see Fig. 181) is not connected with the liver but with some adjacent organ (e. g., stomach), note *D* resembles *G* more than it resembles *F*.

umbilicus up to the costal margin. If a ridge is felt and one is uncertain whether or not it is the edge of the liver, the notch for the gall bladder or round ligament may be sought for. Note the character of the edge, sharp or thick, smooth or irregular.

Next palpate the *surface*, not only of the left lobe in the epigastrium, but also, if the liver is enlarged, that portion of it which projects from under the ribs. Note whether it is smooth, roughened, or nodular, or presents one or more large masses. It should be remembered that inequalities or nodelike irregularities of the muscular and fibrous tissues of the recti and abdominal walls may be mistaken for abnormal roughnesses of the liver. Note also its consistence—hard, soft, or fluctuating—and in the latter case seek for hydatid thrill (*q. v.*). It may be that a soft friction is felt during respiration, which is best perceived posteriorly (perihepatitis), or pulsation of the enlarged organ observed. If the abdomen is distended by gas or fluid, the liver may be felt by "dipping" (*q. v.*), unless the tumidity is excessive.

The gall bladder, when empty, is not palpable. If distended, it may be felt as a smooth, pear-shaped tumour. Unless adhesions have been contracted it is rather freely movable *from side to side* and rises and falls with respiration. If it is the seat of a malignant growth, it is more irregular and nodular on palpation; and if it contains gall stones, the latter may give a sensation resembling "a bag of nuts" (HUTCHISON).

Diagnostic Results of Physical Examination of the Liver and Gall Bladder.

Enlargement of the Liver.—The increase in size may be general or local and circumscribed.

A *general increase* in the size of the liver may be due to passive congestion (usually from valvular cardiac disease), amyloid disease, cancer, fatty infiltration, hypertrophic cirrhosis, leucæmia, hydatids, abscess, gummata or, in rare instances, Weil's disease. The increase due to cancer and amyloid disease may be excessive, the lower border of the enlarged organ reaching considerably below the umbilicus and almost filling the abdomen.

Before deciding that the liver is actually enlarged, certain sources of error must be eliminated.

Enlargement upward must, of course, be judged by percussion. If the upper limit of apparent hepatic dulness is found above the normal level, the extra area of dulness may be caused by consolidation of the base of the right lung, or by an effusion into the right pleural cavity. This question must be decided largely by the history, evidences of pulmonary rather than hepatic disease being present, except in subphrenic abscess; possibly by auscultatory percussion, the stethoscope being placed first over the liver area, posteriorly on the right side, and the upper limit of the liver defined by percussing upward; then placing the instrument over the lung and purcussing downward, attempting to determine the lower pulmonary border. It is also to be borne in mind that the liver may be displaced in an upward direction.

Enlargement *downward*, if judged to have been found either by percussion or by palpation, is open to the possibility that an accumulation of fæces in the transverse colon, or a flattened, hard, cancerous or tuberculous omentum may give rise to the dulness, or may resemble upon palpation the lower edge of a large liver. A fæcal mass, however, is apt to be doughy and indentable, while a tympanitic band or area usually intervenes between a thick omentum and the liver. Apparent enlargement downward may be caused by prolapse or dislocation of the liver.

Circumscribed enlargements of the liver—e. g., of the left lobe—are usually due to abscess, hydatid cyst, cancer, or gumma. As a source of embarrassment, one type of the "lacing" liver may here be mentioned, in which a thin, sometimes thick, movable tongue or lappet from the anterior portion of the right lobe projects below the costal border for a distance of 1 or 2 inches, and may extend as far down as the navel. It usually moves with respiration, by careful palpation is found to be continuous with the liver, and can be grasped in the hand.

Displacement of the Liver.—The liver may lie *above* or *below* its normal position.

Displacement upward may be due to pressure from below by large abdominal tumours, meteorism, or ascites. It may be drawn upward by collapse or retraction of the right lung, or allowed to ride at a higher point by paralysis of the diaphragm.

Displacement downward is caused by downward pressure on the diaphragm by emphysema or spasmodic asthma, large right pleural effusion or pneumothorax, large intrathoracic tumour, and perhaps (affecting mainly the left lobe) by a greatly dilated heart or a large pericardial effusion. Subphrenic peritonitis (abscess), a collection of pus or pus and gas between the liver and diaphragm, is also responsible. Finally, the liver may become prolapsed as a part of a general ptosis or falling of the abdominal viscera. As the posterior border of the liver is quite firmly moored, the descent affects mainly the anterior border, which drops down so as to throw the superior surface forward and downward (see following paragraph).

To distinguish between enlargement and prolapse of the liver—it is necessary to consider the history and associated symptoms in order to determine the existence of a thoracic lesion capable of causing *descent* of the liver; or, *per contra*, the presence of some disease causing an *enlarged* liver. In downward displacement it will be found that the upper surface, especially of the left lobe, is more than usually accessible to palpation, and its rounded shape may be felt. Moreover, a prolapsed liver, because of its separation from the diaphragm, does not move so freely with respiration as the normal or enlarged organ.

Diminution in the Size of the Liver.—There is a slow and progressive lessening in the size of the liver in cirrhosis. The latter is by far the most common cause of hepatic atrophy. In the rare cases of acute yellow atrophy the liver grows rapidly smaller day by day.

As with an increase, so with a decrease, in the size of the liver one must guard against certain possibilities of error. If a marked emphysema is present, the inflated lung intrudes into the complementary pleura between liver and ribs, pulmonary resonance thus encroaching upon the *upper limit* of hepatic dulness. A similar condition is found in right pneumothorax. The *lower limit* of liver dulness may be raised, and the vertical measurement of the liver area diminished by distention of the colon and small intestines, the coils of which interpose themselves between the liver and the abdominal wall; or, with extreme rarity, by free gas in the peritoneal cavity.

Irregularities in the Shape of the Liver.—In rare instances the percussion outline of the liver is found of a notably abnormal shape. In such cases one must think of a diaphragmatic hernia (extremely uncommon), the liver lying in the pleural sac; congenital malformation of the organ; or a disturbance of its normal relations by a rachitic chest or the deformity of Pott's disease of the spine. An entire absence of liver dulness may be due to transposition of the thoracic viscera.

Abnormal Consistence of the Liver or Roughness of its Surfaces.—The *consistence* of the liver may be found to be increased so that it feels abnormally dense, hard, and resistant. Such a condition is indicative of cirrhosis, carcinoma, amyloid, or syphilitic disease. A fluctuating or elastic swelling in or at the lower edge of the liver is an abscess, an hydatid cyst, or a gall bladder distended with bile or muco-pus.

The *surface* of the liver is smooth in amyloid disease, fatty infiltration or degeneration, and passive congestion; roughened in tuberculous peritonitis; and has a granular feel in cirrhosis. If hard nodules varying in size, perhaps with a central depression (umbilication), are found, it is significant of cancer ; smooth, slightly elevated prominences are met with in gummata of the liver ; and one or more smooth projections may be felt as evidences of abscess or cyst. I either of the latter is suspected to be present exploratory punctur (*q. v.*) may be made.

V. THE PANCREAS

Aside from the scanty signs revealed by direct physical examination of the pancreas, other diagnostic points (*q. v.*) are Pain, Fatt Diarrhœa, Glycosuria, Ascites, and Jaundice.

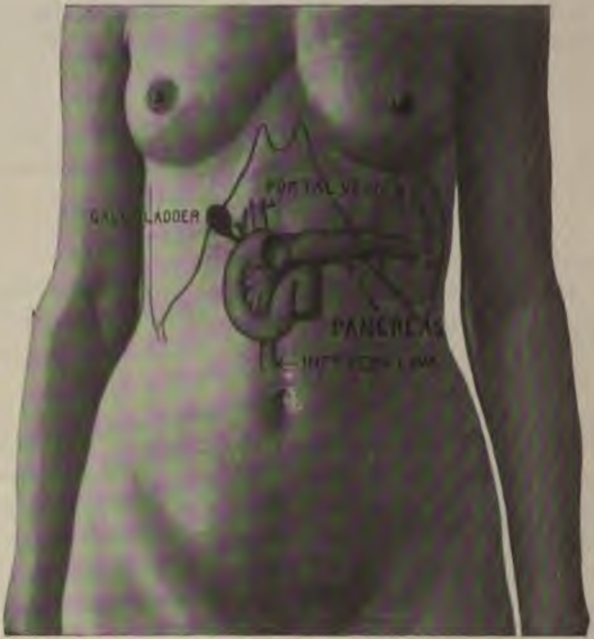


Fig. 183.—Showing the relations of the pancreas.

Topographical Anatomy of the Pancreas.—This organ lies about 3 inches above the umbilicus, midway between navel and ensiform appendix, corresponding to the level of the 2d lumbar vertebra. It is usually 6 inches long. It is deep in the epigastrium, lying transversely across the spinal column with its head resting in the curve of the duodenum and its tail extending to the spleen. The stomach hides it in front. Because of its depth and surroundings, it is rarely accessible for direct examination.

It is clinically important to remember the close relation of the head of the organ posteriorly to the inferior vena cava, the portal vein, and the common bile duct (Fig. 183). On account of its nearness, a cancer or other growth affecting the head of the pancreas may press upon the blood vessels mentioned, giving rise to œdema and ascites; or upon the bile duct, causing persistent jaundice.

Physical Examination of the Pancreas.—Under normal circumstances the pancreas can not be palpated, except in rare instances of extreme emaciation. The most important physical sign of pancreatic disease is the presence of a median tumour in the epigastrium, usually midway between navel and ensiform appendix. The tumour is necessarily deep seated, and not infrequently nothing more than a sense of resistance can be perceived by the palpating hand. The diseases of the pancreas in which either a tumour or a suggestive feeling of resistance is present are acute hemorrhagic and suppurative pancreatitis, chronic pancreatitis, and tumour, solid (usually carcinoma) or cystic.

SECTION XXXV

EXAMINATION OF THE SPLEEN

THE physical examination of the spleen is concerned mainly with the determination of its size and position by inspection, palpation, and percussion. In rare instances auscultation is of service.

Topographical Anatomy of the Spleen.—The spleen is of oval, flattened shape (rarely rhomboid) and lies in the left hypochondriac region. It measures on the average 3 by 5 inches. It reaches from a point 1½ inch to the left of the midspinal line posteriorly almost to the midaxillary line laterally, lying along the 9th, 10th, and 11th ribs. The long axis is parallel with the ribs, and therefore runs downward and forward (Fig. 184). The lower ⅔ of its outer surface are parietal, the upper ⅓ separated from the ribs by the diaphragm and the lower border of the left lung. It is bounded posteriorly by the kidney, above by the diaphragm, and its remaining portions are in contact with the stomach, colon, and small intestines. The anterior border is sharp and indented by from 2 to 4 notches.

Physical Examination of the Spleen.—(1) **Inspection.**—This is rarely of service. If the organ is greatly enlarged it may be visible as a protuberance, extending from the left hypochondriac region downward and inward and moving with respiration.

(2) **Palpation of the Spleen.**—The patient should be in the recumbent position. Then lay the (warm) hand flat upon the abdomen,

so that the finger tips, by exerting pressure and pushing up a fold of skin, lie close to and under the left costal margin at the 10th cartilage. The edge of the spleen, if sufficiently enlarged, may then be felt without further trouble. If not, desire the patient to draw several deep breaths, when the sharp splenic edge, moving with respiration, will be perceived to slip or ride over the ends of the fingers. Still further aid may be obtained from placing the unoccupied hand posteriorly between the ends of the 10th and 11th ribs and making firm pressure so as to tilt the organ forward and make it more accessible.

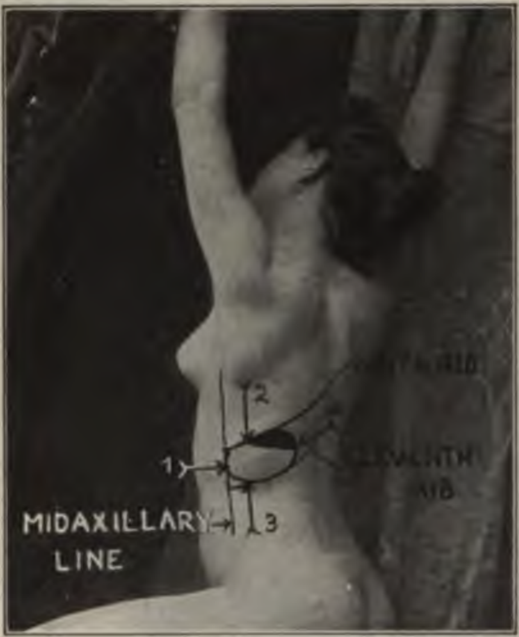


Fig. 184.—Showing the surface topography of the spleen. Shaded area = portion overlapped by lung. Numbered arrows show the lines along which percussion should be conducted.

Under normal circumstances the spleen can not be felt. If increased in size, the anterior edge, which is always directed downward and inward, is sharp and usually smooth. Notching of this border is generally but not always to be found. A point of some practical importance is that a depression or space in which the finger tips can be sunk may invariably be detected at the posterior border of the enlarged organ, between it and the erector spinæ. Infrequently the spleen, although enlarged, can not be felt owing to an unusually strong phreno-colic ligament which prevents its protrusion from under the ribs. In such a case percussion must be relied upon to declare the enlargement.

The question sometimes arises as to whether a tumour found below the left costal margin is an enlarged spleen or an enlarged kidney. The discrimination is to be made by finding, if it is the spleen, that the shape is oval, that it moves with respiration, that it is notched, that it has a sharp edge, that a gap exists between it and nbar muscles, and that it lies in front of the colon—i. e., that s no tympanitic resonance over it. On the other hand, the is rounded or reniform, never has a sharp edge or is notched, slightly, if at all, with respiration, and is overlaid by tympan- onance (Fig. 185).

onfusion arises between an enlarged left lobe of the liver and rged spleen, it is to be remembered that the edge of the spleen a lower level and that the organ can be tilted forward by e over the left lower ribs posteriorly.

Percussion of the Spleen.—The patient may be sitting or ıg, or, if recumbent, should lie partly turned to the right— nidway between the dorsal and right lateral postures—in case with the left arm e head. The percussion should be light, ex- percussing the poste- ortion of the spleen its dulness merges into the left kidney. Per- should be conducted he following lines (*1*, *2*, *4*, Fig. 184):

terior Limit.—Begin at tal margin, and percuss the 10th rib until the itic resonance of the h is replaced by dulness. lly this will be found at laxillary line.

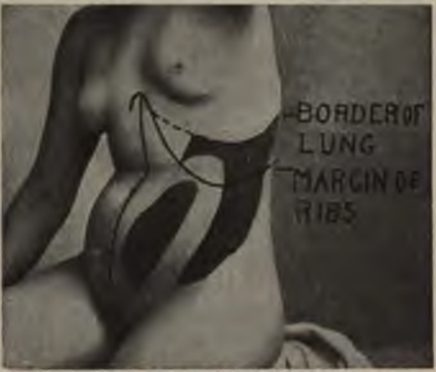


FIG. 185.—If a tumour (shaded area) found below the left costal margin is an enlarged kidney it will be overlaid by the tympanicity of the colon, as in this cut. If it is an enlarged spleen the tympanitic colon will not be found

per Limit.—Begin at the level of the angle of the scapula y between the posterior axillary and scapular lines, passing lly downward until the pulmonary resonance is impaired. lly this occurs at the 9th rib.

wer Limit.—Begin below the border of the ribs, in or a little the posterior axillary line, and percuss vertically upward tympanitic resonance is dulled. Normally dulness is met at th rib, reaching not quite down to the margin of the ribs.

sterior Limit.—Begin, using strong percussion, at the mid- line at the level of the 10th rib, and percuss along the latter. plenic dulness should be attained 1½ inch from the median line, t is always difficult and rarely, if ever, necessary to determine it. f splenic percussion is successful, the area found is oval and ures 2 to 2½ × 3 to 3½ inches. Practically, the vertical extent of the dull area is sufficient for clinical purposes. If it exceeds 3½ inches it may be surmised that the spleen is enlarged.

There are **certain sources of error** in percussion of the spleen which greatly minimize its value as a method of ascertaining the size of the organ, as follows: *Apparent increase* in the size of the splenic dulness may be caused by left pleural effusion, pulmonary basal consolidation, pleural thickening or new growth of lung or pleura, or by fæcal accumulation in the splenic flexure of the colon. *Apparent decrease* in the size of the splenic dulness may be due to an unusual arching of the diaphragm, an emphysematous lung, or a left pneumothorax, crowding the spleen away from the ribs. Dulness, if entirely absent, may be a corroborative sign of the rare floating spleen.

(4) **Auscultation of the Spleen.**—A friction sound may be heard over the spleen if its peritoneal investment is inflamed (perisplenitis), as in infarcts due to a septic embolism or thrombus, or in the splenic abscess following such infarctions. Friction sound and a systolic bruit have been noted in splenic leucæmia and other causes of great enlargement of the organ.

Results of the Examination of the Spleen.—Of the various methods of determining the size and position of the spleen, palpation is by far the most important. A positive diagnosis of splenic enlargement should never be made unless the organ can be felt. Practically, if palpable, unless dislocated, it is enlarged.

(1) **Acute Splenic Enlargement.**—The spleen enlarges more or less rapidly, usually only to a moderate extent, as a consequence of infectious and septic processes. Acute enlargement, therefore, may be due to typhoid fever, typhus fever, malarial fever, relapsing fever, scarlet fever, smallpox, diphtheria, erysipelas, acute miliary tuberculosis, tuberculous peritonitis, pneumonia, epidemic cerebro-spinal meningitis, pyæmia (including septic splenic infarction and abscess), and septicæmia.

(2) **Chronic Uniform Splenic Enlargement.**—Uniform enlargement of the spleen takes place slowly as a consequence of leucæmia (spleno-medullary form) and chronic malaria (ague cake). It is in these diseases that the organ attains its greatest size, in some instances nearly filling the abdominal cavity. Slow enlargement of varying degrees occurs also in splenic anæmia, amyloid disease, cirrhosis of the liver, rachitis, pernicious anæmia, the general venous congestion of cardiac disease, and in passive portal congestion from hepatic cirrhosis or pressure of tumours.

(3) **Irregular or Unequal Splenic Enlargement.**—This is significant of an abscess or the excessively rare carcinoma or hydatids of the spleen.

(4) **Displacement of the Spleen.**—The spleen may be pushed *downward*, so that its edge becomes palpable, by left-side pleural effusion, pneumothorax, emphysema, or thoracic tumour. That the edge can be felt because of descent and not because of enlargement, is not always easy to determine. The existence of thoracic disease, and the absence of ailments capable of causing an increase in size, will aid in the discrimination. Clinically it is not of much importance.

A *floating* spleen, due to congenital laxity of its ligaments or to overstretching from increased size and weight of the organ, may be encountered, usually in women, as a part of a general visceroptosis. It may descend into the pelvis. Ordinarily it is recognisable by its mobility, shape, sharp edge, and notches. If the organ has contracted adhesions, or its shape is altered by inflammatory deposits upon its peritoneal covering, the recognition may be very difficult.

The spleen may be pushed *upward* by tympanites or ascitic fluid, or pulled in the same direction by a contracting lung or the shrinkage of a previously inflamed pleura on the left side. Such a displacement is only of importance as a possible explanation of a percussion abormality.

(5) **Combined Enlargement of Liver and Spleen.**—Both liver and spleen are simultaneously enlarged in passive congestion, hepatic cirrhosis, leucæmia, and amyloid disease.

SECTION XXXVI

EXAMINATION OF THE KIDNEYS, URETERS, BLADDER

HERE are considered the results to be obtained from the physical examination of these organs. Other evidences (*q. v.*) of disease of the urinary organs are noted elsewhere—viz., Results of Urinalysis, Urination, Pain, and Œdema.

I. KIDNEYS

Topographical Anatomy of the Kidneys.—These organs, 2 in number, lie against the posterior abdominal wall, one on each side of the spinal column, in beds of fat and connective tissue. They have a characteristic (reniform) shape. The upper end of the left kidney is in contact with the spleen, the right with the liver. The right kidney lies about ½ an inch lower than its companion. Each kidney is about 4 inches long, 2 to 2½ inches in breadth, and 1 inch in thickness.

The normal surface relations which it is clinically important to have in mind, may be found as follows:

(1) *Anteriorly.*—Prolong the mammillary line on each side downward (or draw a vertical line upward from the middle of Poupart's ligament) until it intersects a horizontal line through the umbilicus (Fig. 186). Or measure and mark on the horizontal umbilical line 2 points, one 3 inches to the right, the other 3 inches to the left, of the median line of the abdomen. From the intersections of the horizontal umbilical and the mammillary lines, or from the points marked, measure upward 1 inch on the right, 1½ inch on the left mammillary line, and indicate by short horizontal lines. The lower ends of the kidneys lie at the level of the short horizontal lines, and the organs themselves extend upward and somewhat inward for 4 inches. One third of the kidney lies to the outer, ⅔ to the inner side of the vertical lines.

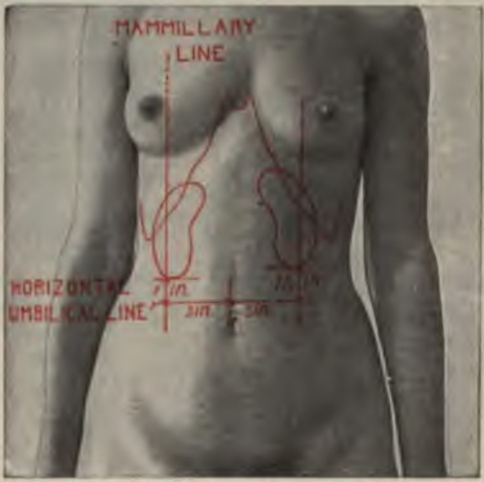


FIG. 186.—Showing the normal surface relations of the kidneys anteriorly, and the method of determining these relations.

(2) *Posteriorly.*—Draw 2 horizontal lines across the back (Fig. 187), the first at the level of the tip of the spinous process of the 11th dorsal vertebra, the second at the level of the tip of the spinous process of the 3d lumbar vertebra. On each side draw 2 vertical lines from the upper to the lower horizontal line. The first vertical line should lie 1 inch from the median line, the second vertical line 2⅜ inches farther away. The outer parallelograms thus outlined correspond to the normal location of the kidneys. The lower ends of these organs (right lower than left) lie 1 to 1½ inch above the iliac crests. About ⅓ of their upper ends are covered by the 11th and 12th ribs. The spleen overlaps the upper extremity of the left, the liver of the right, kidney.

Physical Examination of the Kidneys.—Of the ordinary methods of physical examination, inspection is seldom of value, palpation is indispensable, percussion is untrustworthy and of little use, and auscultation is never employed.

(*a*) **Inspection of Kidneys.**—A large tumour of the kidney is practically the only direct renal physical sign which may become visible. In such a case the growth may fill one or the other anterior lumbar and corresponding portion of the umbilical region. The ribs on the affected side will be bulged outward and afford an indication as the original point of origin of the neoplasm. Sarcoma of the kidney in children, or perhaps a very large hydronephrotic or cystic kidney in the adult, are the most usual causes of a visible renal enlargement. In some cases a perinephric abscess may become visible as a swelling in one or the other posterior lumbar region.

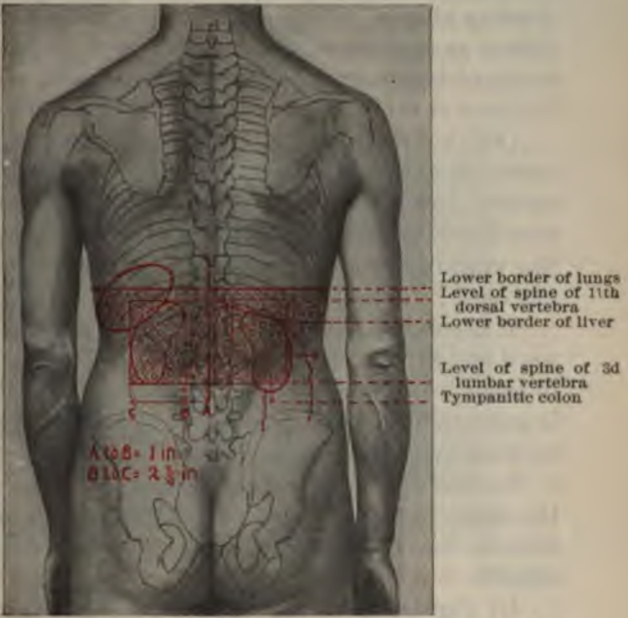


FIG. 187.—Showing surface relations of kidneys posteriorly; also the combined percussion dulness (shaded area) of liver, spleen, kidneys, and thick muscles of the back; also that if the colon is empty of fæces and distended with gas the lower and a part of the outer border of the kidney can be outlined by percussion. Lines of percussion indicated by arrows on right kidney.

(*b*) **Palpation of the Kidneys.**—To palpate the kidney let the patient lie in the dorsal position, head slightly raised, knees drawn up, and feet supported.

(1) Slip one hand under the back so that it rests upon the two lower ribs and the lumbar space immediately below them. The other hand is to be laid flat upon the abdomen in front, resting just below the costal margin, to the outer side of the rectus, in the mammillary line. Desire the patient to take deep and slow respirations. By firm pressure during expiration, with the fingers in front acting against equally firm counter pressure by the posterior hand, an attempt is made to grasp the firm, rounded kidney between the two hands. If the kidney is normal in size and position, its lower extremity may be palpated, provided that the abdominal walls are not too thick (*palpable* kidney). If it can be felt to slip down, like a

"pea in a pod," so that its entire length is accessible, especially if it can be pushed down to the horizontal umbilical line, it is a *movable* kidney. If it can be displaced into the lower half of the abdomen, pushed across the median line, or displaced in any direction, it is a *floating* kidney. It should be remembered that a failure to find the kidney at its normal site may be due to the fact that it is elsewhere in the abdomen, and search should be made accordingly. The kidney may or may not move slightly with respiration.

(2) Another method is to grasp the flank with the full grip of one hand, the thumb resting under the costal margin and the fingers posteriorly, when the kidney (the patient breathing deeply) may be seized and, if movable, felt to slip downward, where it may be held by the grasping hand, palpated by the other hand, and finally made to slip back into its former position.

It is sometimes helpful to let the patient stand, leaning well forward and supporting the weight by the hands resting upon a chair or table, while the abdominal muscles are relaxed and the breathing is quiet and easy. The methods of palpation just described may then be employed.

In addition to abnormal mobility of the kidney, enlargement of the organ may be detected by palpation anteriorly; and in rare instances the renal artery may be felt pulsating if the kidney is seizable.

(*c*) **Percussion of the Kidney.**—It is possible in some cases to determine the lower and a part of the outer border of each kidney, contrasting their dulness with the tympanicity of the ascending and descending portions of the colon which lie immediately anterior to the kidneys (Fig. 187). But the thickness of the lumbar muscles (Fig. 188), the amount of perirenal fat, and the possible packing of the colon with non-resonant fæcal matter, render the results very uncertain as compared to palpation.

To *percuss the kidneys*, the patient may be placed in one of two positions, either of which will relax the lumbar muscles and minimize their influence upon the percussion stroke. He may lie upon the face with one or more thick pillows placed under the abdomen so as to moderately arch the back; or, perhaps better, he may be placed midway between the prone and the lateral position, the examiner facing the patient's back and percussing the kidney of the uppermost side.

Beginning in the middle of the area in which the kidney is normally found (Fig. 187), percuss outward, using vigorous strokes until the renal dulness is replaced by the tympanitic resonance of the colon, thus determining the lower portion of the outer border of the organ. Starting from the same area, percuss downward until a similar change occurs, indicating the lower border of the kidney.

Diagnostic Results from the Physical Examination of the Kidneys.—(1) If the percussion dulness is *increased*, and the examiner is reasonably certain of the accuracy of his results, it may be inferred that the kidney is enlarged by tumour or other cause of increased size of the organ. If the dulness is greatly *lessened* or *absent*, a movable kidney is to be suspected. In either case abdominal palpation is decidedly more reliable.

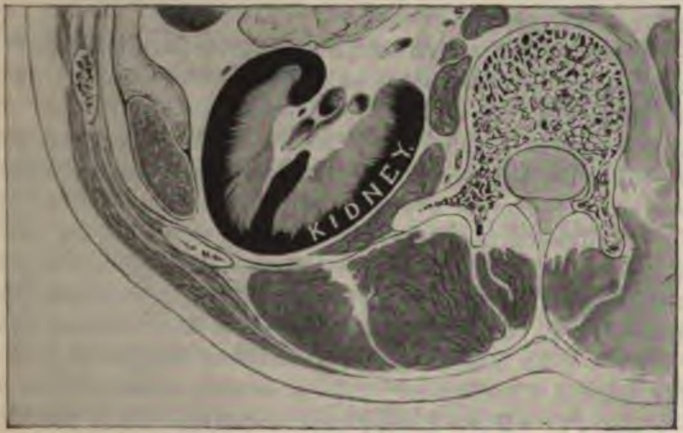


FIG. 188.—Horizontal section through kidney and lumbar muscles, showing the difficulty of kidney percussion.

(2) A movable right kidney—and in the majority of cases it is the right—may require to be discriminated from a distended gall bladder. This may be accomplished by remembering that if it is the kidney it will be freely movable in all directions; may be carried down toward the pelvis, and held there during forcible expiration; may be pushed backward and upward toward its normal position, where it tends to remain and elude further palpation; moves slightly, if at all, with respiration; and that an area of tympanitic percussion (colon) may be found between it and the costal margin. On the other hand, the gall bladder—which, when distended, may resemble the kidney very nearly in size and shape—moves with respiration; can be moved from side to side, or in various radii of a small circle having the neck of the bladder as its centre; and, if pushed backward, away from the abdominal wall, tends to spring forward to its former position when the pressure is removed. Finally, unless the colon, above which it usually lies, has become looped over the neck

of the gall bladder (an infrequent happening), there is no tympanitic band between it and the lower border of liver dulness.

(3) The kidney may be found to be enlarged, or there is an obscure swelling or sense of resistance upon palpation anteriorly; or bulging and indistinct swelling in the region of the kidney posteriorly. In such a case one must bear in mind the possible causes of its enlargement or of closely related tumidity. Such causes are pyonephrosis, hydronephrosis, cystic disease, echinococcus, carcinoma, sarcoma, and perinephric abscess.

The tumours of pyonephrosis, and hydronephrosis or renal echinococcus are alike—rounded, globular, perhaps fluctuating—and may attain a large size. Multiple large cysts of the kidneys can be felt as rounded masses. Malignant tumours of the kidney may grow to dimensions occupying one half of the abdomen, may be somewhat movable, and, if of rapid growth, a sense of fluctuation may be perceived. If in doubt as to whether or not a tumour in the left renal region involves the kidney and is not an enlarged spleen, the position of the descending colon should be ascertained by percussion, inflating it if necessary by pumping in air through a large catheter or colon tube. If the colon lies in front of the tumour (Fig. 185), it is renal and not splenic. Perinephric abscess affords, in some cases, a distinct tumour, in others a bogginess or induration in the interval between the last rib and the iliac crest. An enlarged kidney tends to develop toward the front; a perirenal abscess to bulge backward and become palpable posteriorly.

The differential diagnosis between the various causes of renal enlargement must be made by an attentive consideration of the accompanying signs and symptoms, an examination of the urine, and, in some instances, by the use of the aspirator.

II. BLADDER AND URETERS

It is not within the scope of this work to describe the methods of examining the urinary bladder or the ureters. The use of the cystoscope and the catheterization of the ureters, in men as well as in women, is occasionally of great use to the internalist in the diagnosis of suppurative disease of the kidney or vesical new growths, but the special skill required for such manipulations is as yet confined to a few.

The presence of irritation or inflammation of the urinary bladder as an indication of renal calculus, spinal-cord disease, and pyelitis should be remembered. (See also Urination, page 155).

SECTION XXXVII

XAMINATION OF THE NERVOUS SYSTEM

xamination of the nervous system and the diagnosis of its equires an intimate acquaintance with neural anatomy and y. With the exception of one or two brief references, the n not be dealt with here.

urone.—Modern investigation has framed a conception of us system which differs greatly from that in vogue not rs ago. At present it is conceived as an aggregation of an number of units—the neurones.

rone consists of (1) a *cell body*, (2) *dendrites*, and (3) an . 189).

ll body from one of its poles gives off the dendrites, which y numerous and relatively short, except in some of the sen- ones. The axone or axis-cylinder process passes out from of the cell, and becoming what was formerly termed a nerve be continued for long distances, giving off collaterals at les. The axone finally cleaves into a number of fine forming the end brush or terminal arborization. So also collaterals. The whole of this structure—cell body, den- ne, collaterals, and end brushes—is a neurone.

urone is anatomically independent of all other neurones, ctionally related by contact. The end brushes of one neu- rmingle, but do not anastomose (or but rarely), with the of another, like the branches of contiguous trees.

ndrites are *cellulipetal* in function (i. e., bring impulses *to* ody); the axones are *cellulifugal*, carrying impulses *away* cell body. The cell bodies are the containers and gen- nerve force, the dendrites conduct it to the cell body from , and the axones distribute it.

ell-being of the processes of the neurone (dendrites and pends upon their connection with the cell body and the of the latter. If the cell body is injured by accident or e processes degenerate, or if a process is severed from the the separated part undergoes degeneration—i. e., the cell phic for its processes. Conversely, a cell separated from es undergoes a slighter but still perceptible degeneration.

l and Peripheral Neurones.—Starting from the large cell the motor area of the brain cortex, efferent impulses, which his source motor, but from other centres may be secretory,

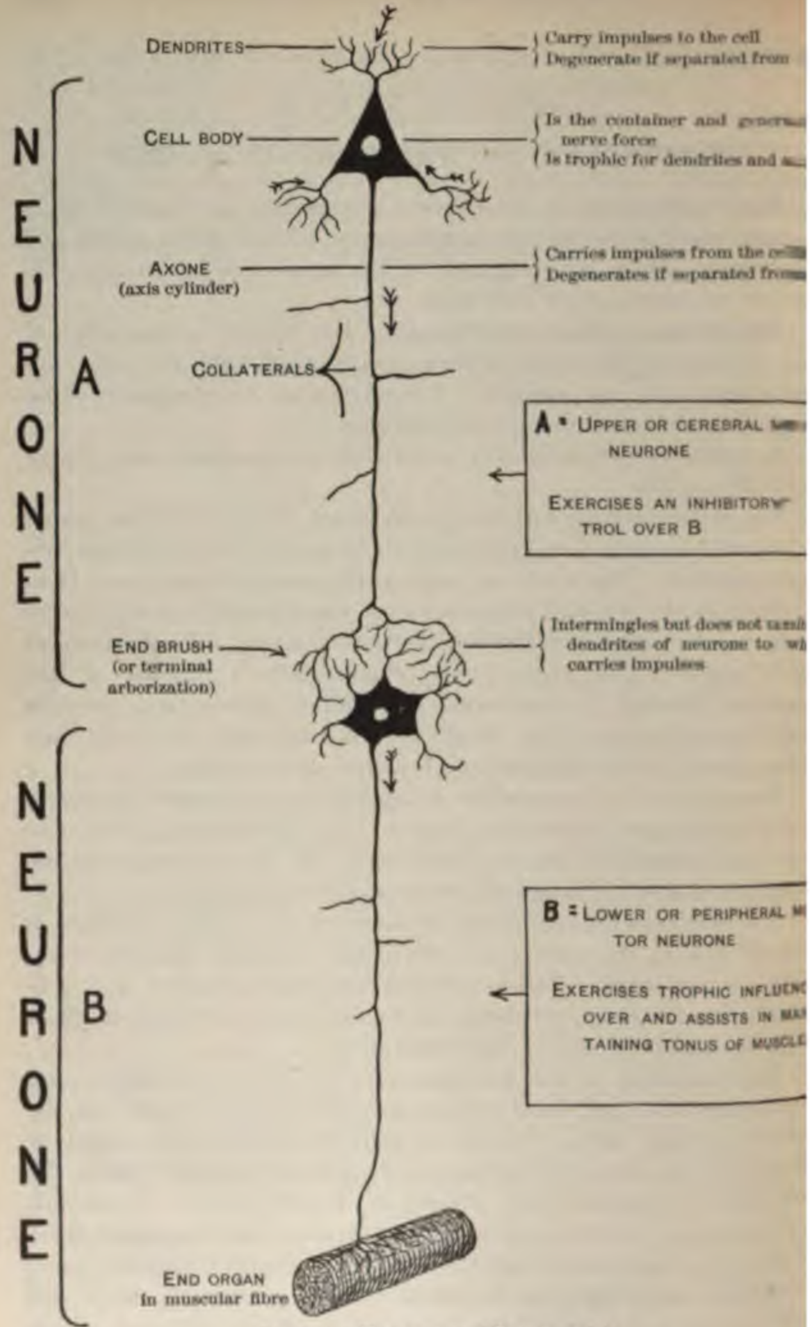

DENDRITES
Carry impulses to the cell
Degenerate if separated from
CELL BODY
Is the container and genera
nerve force
Is trophic for dendrites and a
AXONE
(axis cylinder)
Carries impulses from the ce
Degenerates if separated fro
COLLATERALS
A = UPPER OR CEREBRAL M
NEURONE
EXERCISES AN INHIBITORY
TROL OVER B
END BRUSH
(or terminal
arborization)
Intermingles but does not uni
dendrites of neurone to wh
carries impulses
B = LOWER OR PERIPHERAL M
TOR NEURONE
EXERCISES TROPHIC INFLUENC
OVER AND ASSISTS IN MAI
TAINING TONUS OF MUSCLE
END ORGAN
in muscular fibre
NEURONE
A
NEURONE
B

FIG. 189.—The neurone (diagrammatic).

trophic, or inhibitory, travel down the axones which run from the cortical cell bodies to the cell bodies in the cranial nerve nuclei and the anterior horns of the spinal cord, their end brushes surrounding and transferring impulses to the latter. The latter in turn send off axones which, leaving the nuclei and the cord in the motor cranial nerves and in the anterior roots of the spinal nerves, are finally distributed by way of the peripheral nerves to the voluntary muscles.

It is thus evident that cortical motor impulses must pass through at least two sets of neurones, the primary, central, or higher level neurones (cortex to anterior horns, or to nuclei of motor cranial nerves), and the secondary, peripheral, or lower level neurones (anterior horn or cranial nuclei to muscles). These are sometimes referred to respectively as the upper and lower segments of the motor path (Fig. 190).

The upper and lower motor neurones are not entirely independent. The upper neurones exercise a constant controlling influence over the lower. The lower neurone is just as constantly sending impulses to the muscle in which its axone terminates, and the nutrition, the reflex irritability, and the tone or tension of the muscle depend upon the steady reception of such impulses. These are primarily received from the sensory side of the nervous arc, however, and thus it is apparent why in tabes dorsalis, which is primarily a disease of the sensory neurone, with unaltered motor neurones, there is a loss of muscle tonus. Practically, the lower neurone *plus* the muscle forms a nutritive unit.

It must be clearly understood that although the cell bodies of the nuclei of the motor cranial nerves lie in the substance of the brain-stem, they are as distinctly peripheral as those of the anterior horns of the spinal cord.

Motor and Sensory Paths.—The *direct motor* path, for all voluntary impulses from the motor area (Fig. 62), and the *indirect motor* path, for the co-ordination of muscular movements and the higher reflex and automatic movements, are shown in Fig. 190.

The *direct sensory* path, through which pass the sensations of touch, pain, and temperature, and the *indirect sensory* path, which is concerned with the sensations from the muscles, joints, and viscera, are shown in Fig. 191.

Spinal Cord.—The surface relations of the spinal cord are shown in Fig. 192; and certain facts, with reference to the names, functions, and diseases of the parts of the cord, in Figs. 193, 194, 195 *a* and *b*. The segmental localization of the motor, sensory, reflex, and other functions of the cord are as follows:

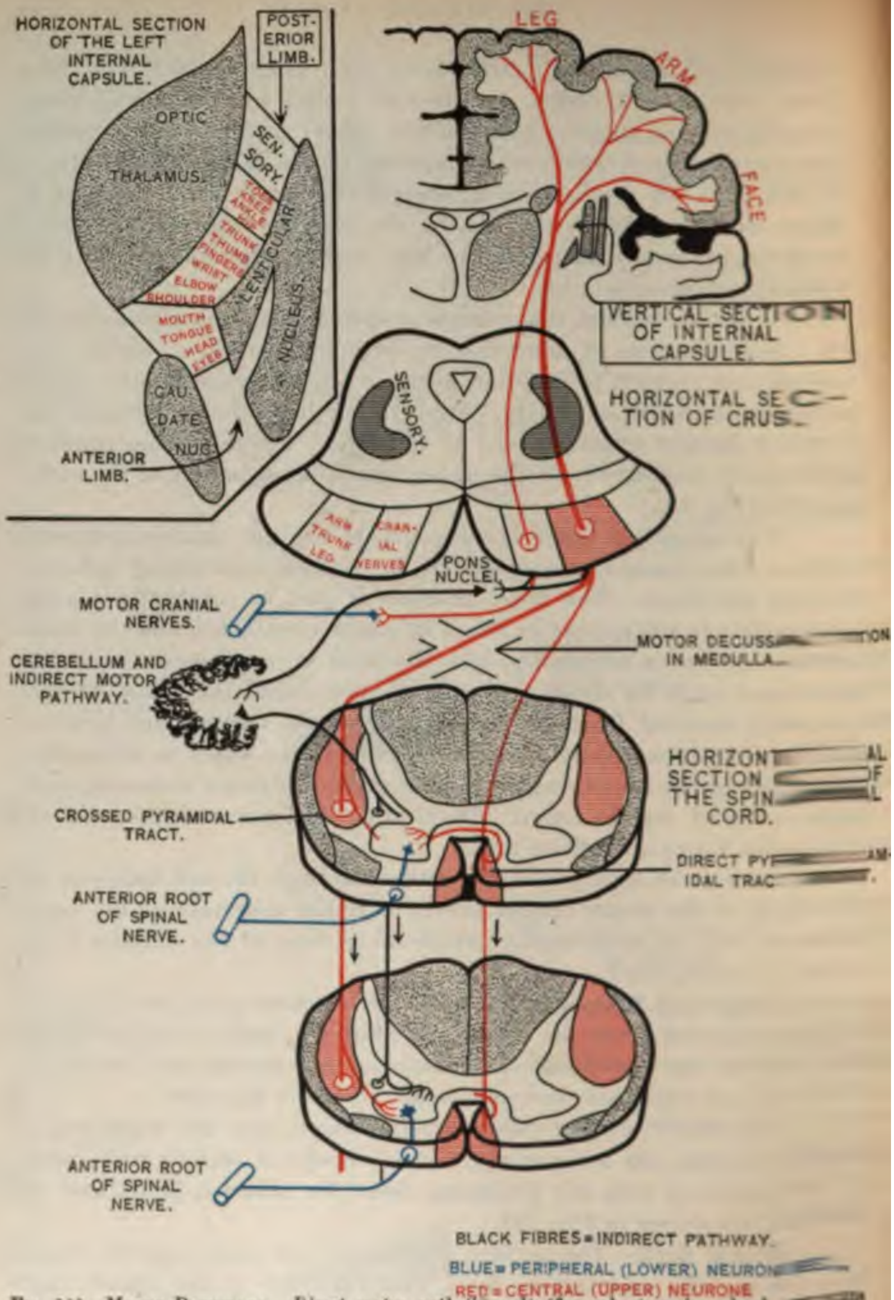
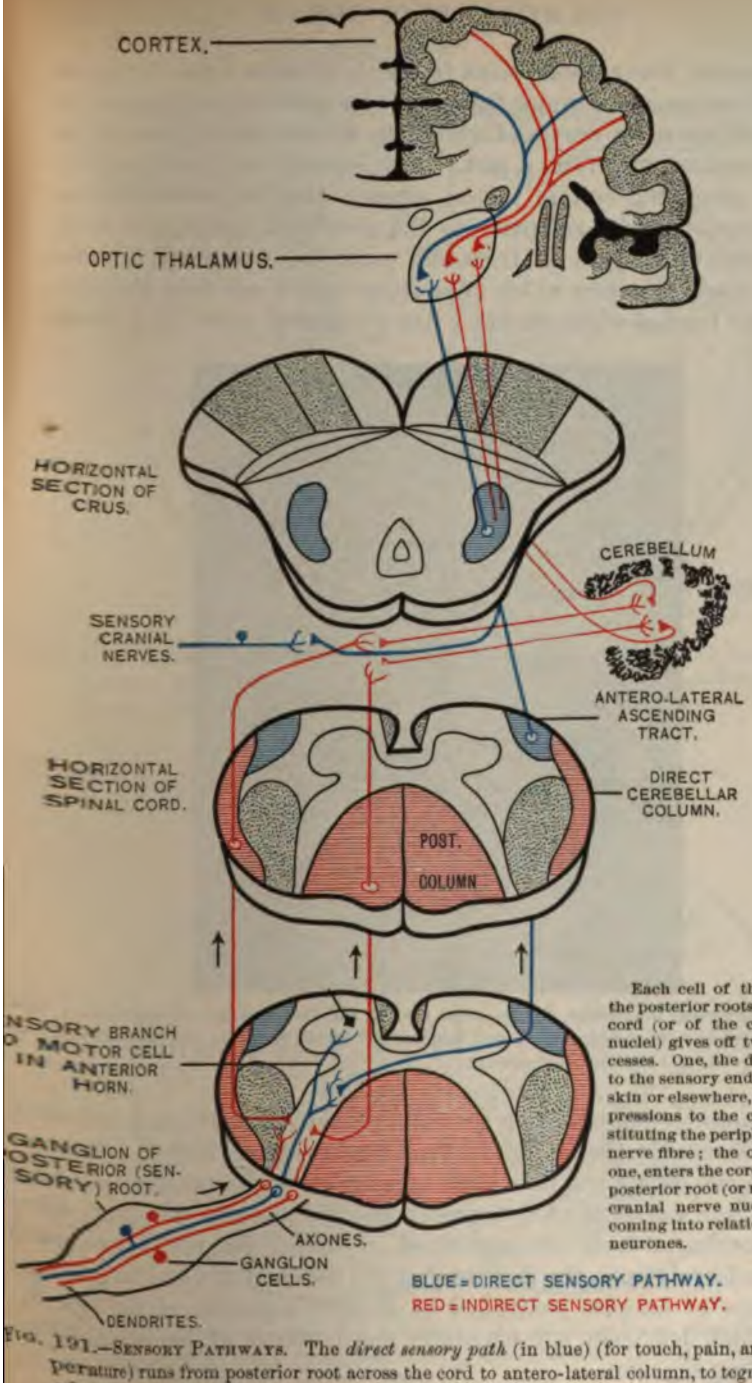

HORIZONTAL SECTION OF THE LEFT INTERNAL CAPSULE.
POSTERIOR LIMB.
OPTIC
THALAMUS.
SENSORY
TOES
KNEE
ANKLE
HIP
TRUNK
THUMB
FINGERS
WRIST
ELBOW
SHOULDER
MOUTH
TONGUE
HEAD
EYES
LENTICULAR
NUCLEUS.
CAUDATE NUC.
ANTERIOR LIMB.
LEG
ARM
FACE
VERTICAL SECTION OF INTERNAL CAPSULE.
SENSORY.
HORIZONTAL SECTION OF CRUS.
ARM
TRUNK
LEG.
CRANIAL NERVES
PONS NUCLEI
MOTOR CRANIAL NERVES.
CEREBELLUM AND INDIRECT MOTOR PATHWAY.
MOTOR DECUSS[illegible]ON IN MEDULLA.
HORIZONT[illegible]AL SECTION [illegible]F THE SPIN[illegible]L CORD.
CROSSED PYRAMIDAL TRACT.
DIRECT PY[illegible]AMIDAL TRAC[illegible].
ANTERIOR ROOT OF SPINAL NERVE.
ANTERIOR ROOT OF SPINAL NERVE.
BLACK FIBRES = INDIRECT PATHWAY.
BLUE = PERIPHERAL (LOWER) NEURON[illegible]
RED = CENTRAL (UPPER) NEURONE

FIG. 190.—MOTOR PATHWAYS. *Direct motor path* (in red) (for voluntary impulses) [illegible] from cortex, via corona radiata, internal capsule, crus, pons, medulla, crossed and d[illegible] pyramidal columns, to motor cells of anterior horn; the cranial nerve motor fibre[illegible] (in (red) cross at various levels in crus, pons, and medulla. *Indirect motor path* (in b[illegible]k) (for muscular co-ordination and higher reflex and automatic movements) runs from [illegible] cortex to pons nuclei, to cerebellum, to lateral fundamental column, via the peduncle[illegible] the fibres terminating at various levels in the anterior horn.


CORTEX.
OPTIC THALAMUS.
HORIZONTAL SECTION OF CRUS.
CEREBELLUM
SENSORY CRANIAL NERVES.
ANTERO-LATERAL ASCENDING TRACT.
HORIZONTAL SECTION OF SPINAL CORD.
DIRECT CEREBELLAR COLUMN.
POST. COLUMN
NSORY BRANCH
O MOTOR CELL
IN ANTERIOR HORN.
GANGLION OF
OSTERIOR (SENSORY) ROOT.
AXONES.
GANGLION CELLS.
DENDRITES.
BLUE = DIRECT SENSORY PATHWAY.
RED = INDIRECT SENSORY PATHWAY.

Each cell of the ganglia of the posterior roots of the spinal cord (or of the cranial nerve nuclei) gives off two main processes. One, the dendrite, goes to the sensory end organ in the skin or elsewhere, bringing impressions to the cell, and constituting the peripheral sensory nerve fibre; the other, the axone, enters the cord through the posterior root (or runs from the cranial nerve nuclei inward), coming into relation with other neurones.

FIG. 191.—SENSORY PATHWAYS. The *direct sensory path* (in blue) (for touch, pain, and temperature) runs from posterior root across the cord to antero-lateral column, to tegmentum of crus, to optic thalamus, to cortex. The *indirect sensory paths* (in red) (for co-ordinative sensations from muscles, joints, and viscera) run upward on same side, via the direct cerebellar tract and the posterior column, decussating at upper part of cord, to cerebellum, to optic thalamus, to cortex.

Segmental Motor Localization in the Cord.—One must distingu carefully between the motor functions of a segment and those of peripheral nerve or nerves of which the axones coming from a ticular segment may form a part. Each segment may be regarde a unit possessing certain motor, sensory, trophic, vasomotor, reflex functions, with reference to the peripheral distribution of nerve roots which pass off from and enter it. A segment is na after the pair of nerves which arise from it, and not from the ve bra at the level of which it lies. Any peripheral nerve may con

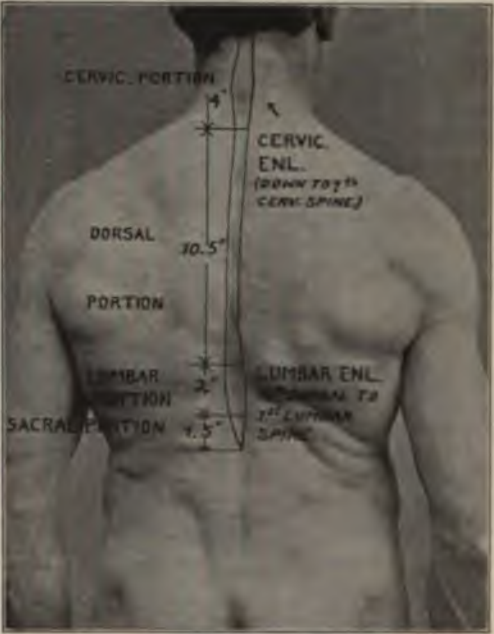
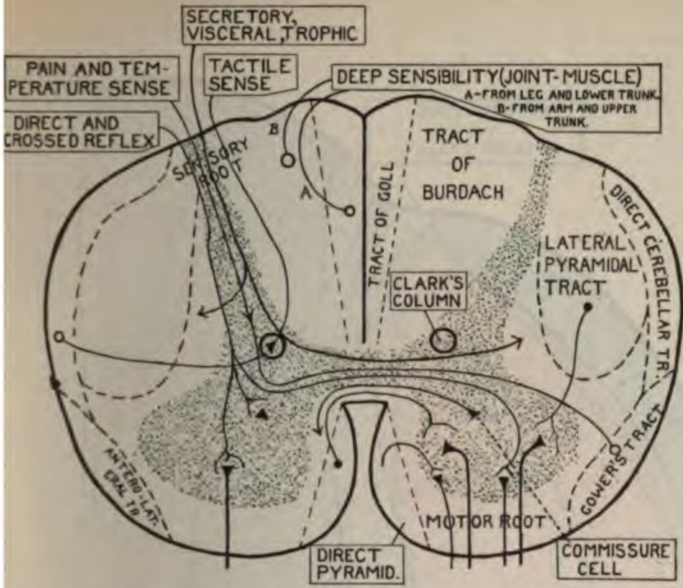
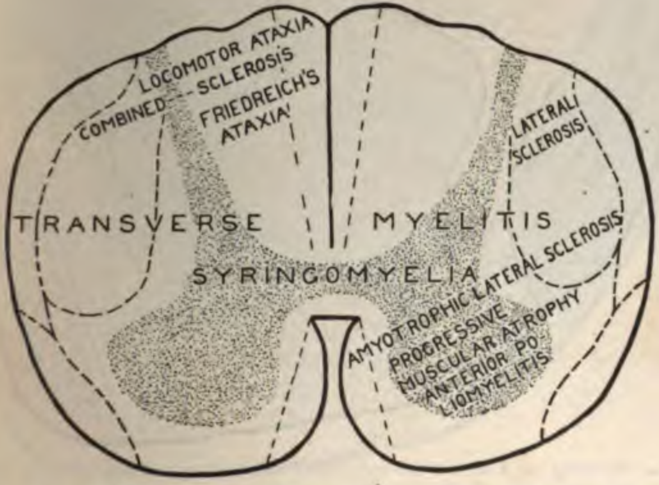


FIG. 192.—Showing the relation of the spinal cord to the dorsal surface of the trunk relative length of the cervical, dorsal, lumbar, and sacral portions; and the positi the cervical and lumbar enlargements.

fibres, motor or sensory, from several different segments, and, ind this is the case in the majority of the cerebro-spinal nerves. Co quently, *movements*, not individual muscles or nerves, are represen in the gray matter of each segment, and the determination of exact position of focal (circumscribed or limited) lesions of the c depends largely upon the discovery of the deficient action of a gr or groups of muscles controlled by definite segments of the c from which deficiency one may infer the existence of disease in controlling segment.



193—Showing the functions of the fibres of the anterior and posterior roots, and their relations to the horns and columns of the spinal cord. Redrawn and modified from Dana. Compare Figs. 195 *a* and 195 *b*.



194.—Showing the parts and columns of the spinal cord and some of the diseases which affect them. Compare with Fig. 193; also with Figs. 195 *a* and 195 *b*.

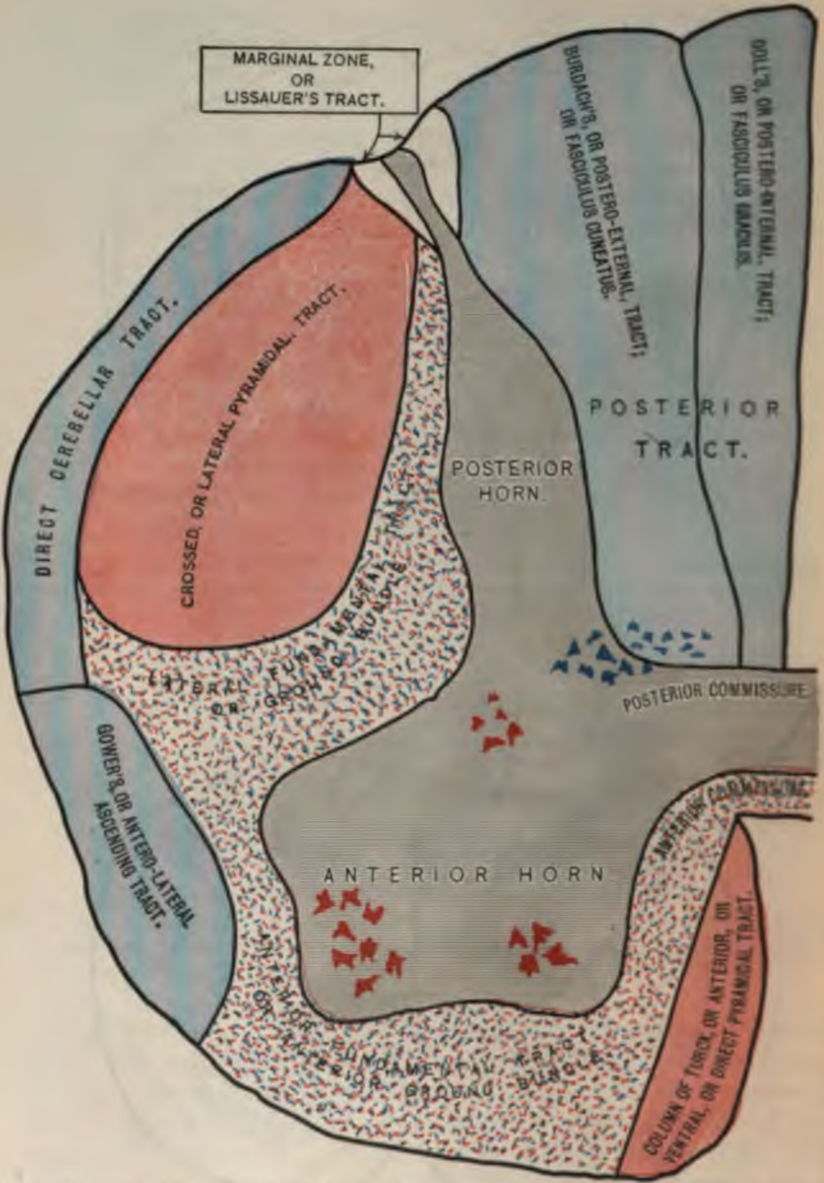

MARGINAL ZONE, OR LISSAUER'S TRACT.
GOLL'S, OR POSTERO-INTERNAL, TRACT; OR FASCICULUS GRACILIS.
BURDACH'S, OR POSTERO-EXTERNAL, TRACT; OR FASCICULUS CUNEATUS.
DIRECT CEREBELLAR TRACT.
CROSSED, OR LATERAL PYRAMIDAL, TRACT.
POSTERIOR TRACT.
POSTERIOR HORN.
LATERAL FUNDAMENTAL TRACT OR GROUND BUNDLE
POSTERIOR COMMISSURE
GOWER'S, OR ANTERO-LATERAL ASCENDING TRACT.
ANTERIOR COMMISSURE
ANTERIOR HORN
ANTERIOR FUNDAMENTAL TRACT OR ANTERIOR GROUND BUNDLE
COLUMN OF TURCK, OR ANTERIOR, OR VENTRAL, OR DIRECT PYRAMIDAL TRACT.

Fig. 195 a.—Showing the tracts of the spinal cord and their varied nomenclature. Red = motor (or efferent). Blue = sensory (or afferent).

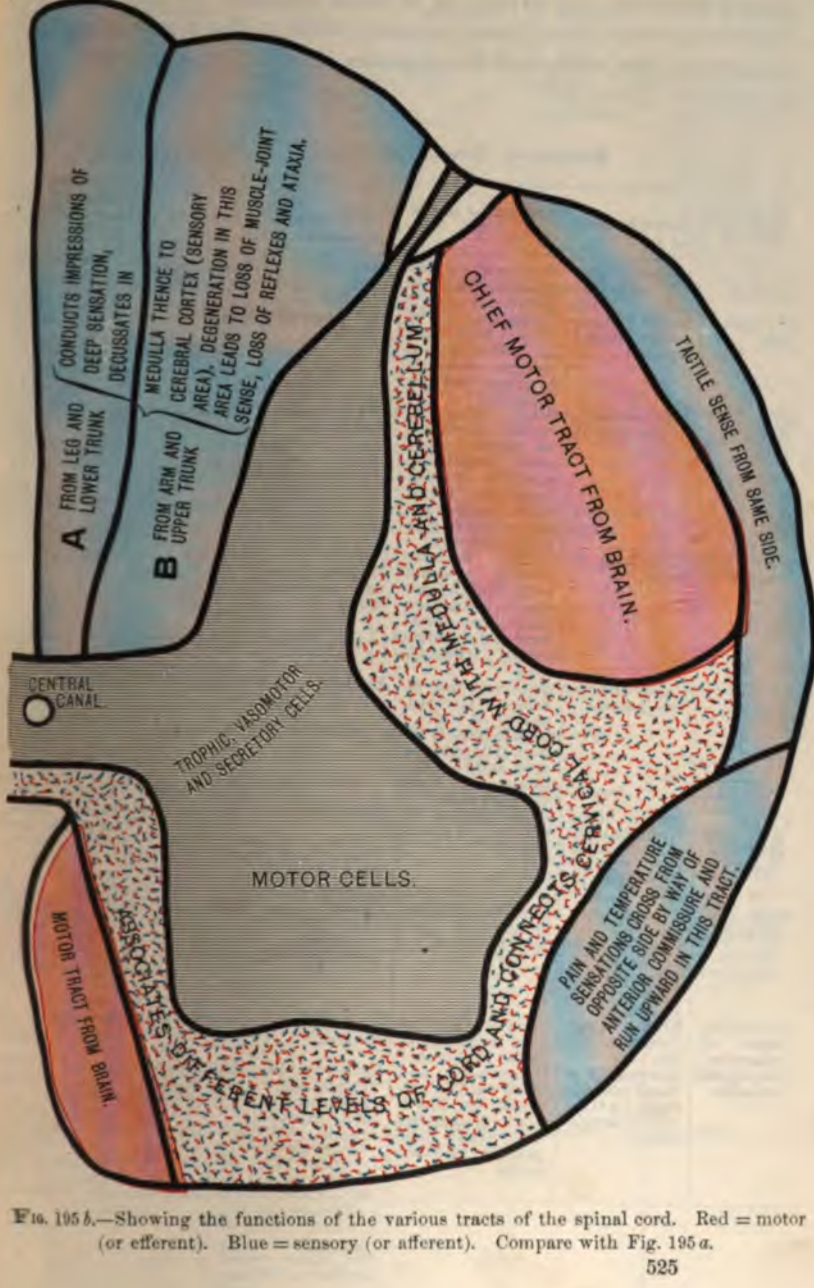

A FROM LEG AND LOWER TRUNK
B FROM ARM AND UPPER TRUNK
CONDUCTS IMPRESSIONS OF DEEP SENSATION, DECUSSATES IN MEDULLA THENCE TO CEREBRAL CORTEX (SENSORY AREA). DEGENERATION IN THIS AREA LEADS TO LOSS OF MUSCLE-JOINT SENSE, LOSS OF REFLEXES AND ATAXIA.
CENTRAL CANAL.
TROPHIC, VASOMOTOR AND SECRETORY CELLS.
MOTOR CELLS.
CHIEF MOTOR TRACT FROM BRAIN.
TACTILE SENSE FROM SAME SIDE.
ASSOCIATES DIFFERENT LEVELS OF CORD AND CONNECTS CERVICAL CORD WITH MEDULLA AND CEREBELLUM.
PAIN AND TEMPERATURE SENSATIONS CROSS FROM OPPOSITE SIDE BY WAY OF ANTERIOR COMMISSURE AND RUN UPWARD IN THIS TRACT.
MOTOR TRACT FROM BRAIN.

FIG. 195 *b*.—Showing the functions of the various tracts of the spinal cord. Red = motor (or efferent). Blue = sensory (or afferent). Compare with Fig. 195 *a*.

The following table, from various sources, shows the muscles, their normal function, the evidences of their deficient action, the nerves which innervate them, their representation in the medulla and the segments of the cord, and the diseases in which they are commonly involved.

Muscles of Tongue, Palate, and Pharynx

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	Diseases in which muscle is commonly involved.
Genioglossus.	Pushes tongue to opposite side.	Tongue when protruded deviates to paralyzed side.	The twelfth nerve (hypoglossal).	Medulla.......	Bulbar palsies (acute and chronic); in specific traumatic and tuberculous diseases of base; dystrophies (rare). General paresis. Syringobulbia. Tabes, rare. Multiple sclerosis (rare).
Styloglossus.	Raises tongue backward and upward.	Tongue can not be moved backward or hollowed out (action deficient in many healthy subjects).	The twelfth nerve.	Medulla.......	
Lingual muscle proper.	All movements of the tongue itself.	When lying in mouth deviation to healthy side; when protruded, deviates to paralyzed side; if one or both halves are atrophied tongue looks shrivelled.	The twelfth nerve.	Medulla.......	
Azygos uvulæ.	Shortening of uvula.	Uvula deviates toward sound side; if both sides are paralyzed there are nasal tone and regurgitation through nose.	Probably pharyngeal plexus; seventh nerve (?).	Medulla.......	As above.
Levator palati.	Raises the velum palati.	Arch can not be raised in the intonation of "ah"; if paralysis is bilateral flapping of arch and regurgitation of food through nose.	As above......	Medulla.......	As above.
Palato-pharyngeal muscles.	Prevent food from passing toward upper part of pharynx and posterior nares.	Regurgitation of food; nasal speech.	The fifth nerve.	Pons, medulla, cervical cord.	Basilar affections; tabes; multiple sclerosis.
Stylo-pharyngeus.	Helps to draw larynx upward so as to be closed by epiglottis and overtopped by tongue.	Imperfect deglutition; food gets into windpipe.	Glosso-pharyngeal.	Medulla.......	Bulbar affections and diseases of the base; tabes; multiple sclerosis; general paresis.
Constrictors of pharynx.	Help to push food into gullet.	Food is swallowed very imperfectly; sticks in throat.	Pharyngeal plexus.	Medulla.......	Diseases of the base (bulbar).
Laryngeal muscles.	Movements of vocal cords in respiration and in articulation.	Hoarseness and difficulty in breathing; laryngoscopic examination reveals false position of vocal cords.	Recurrent laryngeal nerve excepting the crico-thyroid muscle.	Medulla.......	Bulbar troubles (similar symptoms may be caused by tumours and foreign bodies in larynx).

Muscles of Head and Neck

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	Diseases in which muscle is commonly involved.
Sterno-cleido-mastoid.	Raises and turns face to opposite side; head inclines to same side; if both muscles act conjointly head is brought forward.	Inability to raise head from bed, or other horizontal position, if both musclesare affected; if one muscle is affected, no marked change of position, unless opposite muscle is contractured; spasm of muscle frequent; head inclined to one side.	Spinal accessory.	Medulla and second and third cervical segments.	In bulbar and cervical-cord affections; in later stages of progressive muscular atrophies; occasionally in neuritis, syringomyelia, multiple sclerosis, and tic.
Rectus capitis anticus major.	To flex head ..	Can not flex head so as to bring chin on chest.	Upper cervical.	Uppercervical segments.	Diseases of the cervical region (myelitis, meningitis, tumour; progressive wasting of muscles; syringomyelia, neuritis).
Rectus capitis anticus minor.	To flex head ..				
Rectus capitis lateralis.	Slight rotation.	Deficient rotation scarcely noticeable, unless sterno-cleido-mastoids are diseased.			
Scaleni anterior medius, et posterior.	Elevate ribs when vertebral column is fixed; aid in inspiration; slight lateral flexion.	Deficient inspiratory movements.	Lower cervical nerves.	Lowercervical segments.	
Longus colli.	Flexion of vertebral column.	Imperfect flexion of upper spine.	Lower cervical nerves.		

Muscles of Shoulders and Upper Extremity

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	Diseases in which muscle is commonly involved.
Trapezius. 1. Clavicular portion (respiratory; outer third of clavicle to occipital bone).	Pulls head backward; rotates slightly toward side of muscle, so that chin is turned to opposite side; contraction of both clavicular portions bends head backward; slight elevation of shoulders; aids in deep inspiration.	Deficient backward movement of head; not marked as a rule because deep muscles perform this function; shoulder does not move during inspiration.	Spinal accessory.	Medulla and second and third cervical segments.	Progressive muscular wasting; diseases of medulla and upper cervical cord; clavicular portion least frequently involved.
2. Middle portion (from acromion and outer spine of scapulato ligament. nuchæ and upper dorsal spines).	Raises shoulder blade: elevation of acromion (clavicle goes along).	Acromion depressed by weight of upper extremity; inner upper angle may be pulled upward by levator anguli scapulæ: internal lower angle is nearer to median line.	Spinal accessory nerve.	As above......	As above.

Muscles of Shoulders and Upper Extremity (continued)

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	[illegible]
3. Lower portion and adductor.	Adduction of scapula toward median line.	Margin of scapula is about 10 centimetres distant, instead of being 5 or 6 centimetres distant from median line; loss of abductor may be covered up by action of rhomboids; rounding of back.	Spinal accessory nerve.	Medulla and second and third cervical segments.	
Rhomboids	Oblique movement of scapula from below, upward and inward, so that inferior angle is brought nearer the median line; hold spinal margin of scapula down to thorax.	Deep groove between inner margin of scapula and thorax; if serratus is normal, this groove disappears if arm is extended forward; shoulder blade can not be approximated to median line. (According to Duchenne, this can be effected by upper portion of latissimus dorsi.)	Fifth cervical.	Fourth and fifth cervical segments.	As above.
Levator anguli scapulæ.	Draws superior inner angle of scapula upward; aids in shrugging of shoulders.	Isolated paralysis rare.	Third and fifth cervical nerves.	Second and fourth (?) cervical segments.	Dystroph and cervi diseases.
Serratus magnus.	Rotation of shoulder blade outward, and slight elevation of acromion; holds inner margin of scapula to thorax; brings arm from horizontal to vertical position.	Scapula pulled upward; lower inner angle nearer the median line; arm can not be raised above horizontal position; if arm is stretched forward scapula is removed from thorax ("winged scapula"); during abduction of arm, scapula is moved nearer to median line, and crowds trapezius and rhomboids forward.	Posterior thoracic nerve.	Fifth and sixth cervical segments.	Progress muscular a phies (d trophies); ne ritis of part the brach plexus; a traumatic juries to sho der; in cer cal-cord aff tions. Am trophic late sclerosis, s ingomyelia.
Deltoid (three divisions).	To raise arm to horizontal position, and forward, outward, or backward; movements possible only if scapula is fixed by action of serratus and trapezius.	Can raise shoulder but not arm; shoulder flattened (atrophy); groove between acromion and head of humerus; each division of deltoid may be paralyzed singly.	Circumflex....	Fourth, fifth, and sixth cervical segments.	As above; in Erb's f of obstet paralysis.
Infraspinatus. Teres minor.	Rotator humeri posticus (Duchenne); rotate arm outward.	Arm can not be moved outward. Difficulty in writing (Duchenne).	Suprascapular. Circumflex.	Fourth, fifth, and sixth cervical segments.	As in ca deltoid.
Subscapularis.	Rotator humeri anticus (Duchenne); rotates arm inward.	Arm can not be moved inward; scapula is rubbed against ribs.	Subscapular nerve.		

Muscles of Shoulders and Upper Extremity (continued)

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	Diseases in which muscle is commonly involved.
Supraspinatus.	Helps to steady shoulder joint and to elevate arm forward and outward; outer angle of scapula is depressed.	According to Duchenne, humerus is separated still farther from acromion, if supraspinatus is affected in addition to deltoid.	Suprascapular.	Fourth cervical.	As above.
Latissimus dorsi.	Pulls the arm when raised, downward and backward; if arm is at rest upper portion brings scapula nearer the median line; united action of upper third of both muscles causes extension of dorsal trunk; single action causes lateral movement of trunk.	Arm can not be moved backward; insufficient extension of dorsal spine; trunk can not be moved laterally.	Subscapular, also branches of dorsal and lumbar nerves passing through muscle.	Sixth and seventh cervical.	As in progressive atrophies and dystrophies; in cervico-dorsal lesions; in neuritis.
Teres major.	Rotates raised humerus inward; adduction of arm to thorax; slight elevation of shoulder.	Very few symptoms; action supplied by other muscles.	Subscapular...	Seventh cervical.	As above.
Pectoralis major.	Clavicular portion depresses humerus from raised position to horizontal; adduction of arm, as in giving a blessing; sternal portion depresses arm completely, and if arm is at rest draws acromion forward and backward.	Imperfect adduction of arm; paralysis can be discovered best by extending arms and trying to press volar surfaces against each other.	Anterior thoracic.	Fifth, sixth, and seventh cervical.	Amyotrophies and dystrophies, chiefly; also in lesions of brachial plexus.

Muscles of Arm, Forearm, and Hand

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	Diseases in which muscle is commonly involved.
Triceps....	Extends forearm; long head of triceps, and coraco-brachialis help to keep head of humerus in position.	Arm can not be extended except by its own weight; if long head of triceps is affected subluxation of head of humerus occurs easily.	Musculo-spiral.	Sixth, seventh, eighth cervical segments.	Poliomyelitis and other affections of cervical cord; traumatic injuries; amyotrophies and dystrophies (triceps escapes in many peripheral palsies); syringomyelia; cervical rib; neuritis (alcoholic or syphilitic).
Biceps.....	Flexion and supination of forearm.	Flexion deficient, but can be carried out in part by other muscles.	Musculo-cutaneous.	Fourth, fifth, sixth cervical.	

Muscles of Arm, Forearm, and Hand (*continued*)

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	Diseases in which muscle is commonly involved.
Supinator longus.	Flexes forearm and aids in pronation.	Flexion and pronation deficient; muscle does not stand out prominently if arm is flexed and attempt is made by another to extend it forcibly; if muscle is atrophied arm is spindle-shaped.	Musculo-spiral.	Fourth, fifth cervical.	As above; involved in peripheral neuritis (traumatic), not in lead palsy.
Supinator brevis.	Supinates hand when forearm is extended.	Deficient supination of hand.	Musculo-spiral.	Fifth cervical.	Diseases as above; also in peripheral palsies.
Extensor carpi radialis longus et brevis.	Extension and abduction of wrist; the shorter muscle has pure extension action only.	Wrist can not be flexed dorsally (extended) or abducted; flattening of forearm.	Musculo-spiral.	Seventh cervical.	As before; especially in neuritis.
Extensor carpi ulnaris.	Extension and abduction of wrist.	Wrist can not be flexed dorsally or adducted; "drop-wrist" is characteristic of paralysis of extensors.	As above......	Seventh cervical.	As above.
Extensor digitorum communis. Extensor indicis. Extensor minimi digiti.	Extension of first phalanges of all fingers and abduction.	First phalanges can not be extended nor fingers abducted; grasp is weak because flexor muscles are shortened and can not contract forcibly.	Musculo-spiral.	Seventh cervical.	As above.
Flexor carpi radialis	Flexion of wrist and pronation.	Deficient flexion ..	Median........	Eighth cervical.	As above.
Flexor carpi ulnaris.	Flexion of wrist and supination.	Flexion and supination impaired.	Ulnar	Eighth cervical.	As above.
Palmaris longus.	Flexion of wrist only.	Flexion impaired; no anomalous position of hand from paralysis of wrist as hand falls by its own weight; the flexors of fingers may act as substitutes.	Median........	Eighth cervical.	As above.
Flexor digitorum sublimis.	Flexes second phalanx toward first.	Second phalanx can not be flexed.	Median........	Eighth cervical.	As above.
Flexor digitorum profundus.	Flexes last two phalanges toward first.	Last two phalanges can not be flexed.	Ulnar and median.	Eighth cervical.	As above; muscle should be tested with special care in cases of traumatic injuries.
Interossei and lumbricales.	Abduction and adduction of fingers if first phalanges are extended; flexion of first phalanges and simultaneous extension of second and third phalanges.	Fingers can not be abducted or adducted; interosseous spaces are very marked; "Main en griffe" due to extension of first phalanges and flexion of second and third phalanges.	Ulnar, which also supplies third and fourth lumbricales; median supplies first two and sometimes third lumbricales.	Eighth cervical, first dorsal.	As above; often the first muscles to be affected in progressive spinal atrophies.

Muscles of Arm, Forearm, and Hand (continued)

NAME OF MUSCLE.	Normal function.	Symptoms of deficient action.	Innervated by	Represented in	Diseases in which muscle is commonly involved.
Thenar muscles: Extensor pollicis brevis.	Extends first phalanx and abducts metacarpal bone; acts with adductor pollicis longus.	Impairment of extension and adduction; flattening of ball of thumb.	Musculo-spiral.	First dorsal ...	As before; more especially in amyotrophies and neuritis.
Extensor pollicis longus.	Extends both phalanges of thumb; also adduction of metacarpal bone and backward movement of thumb.	Deficient extension and adduction; second phalanx is flexed toward first.	Musculo-spiral.	First dorsal ...	As above.
Abductor pollicis longus.	Abduction of metacarpal bone; aids in flexion of hand.	Deficient abduction of metacarpal bone; if this muscle and extensor pollicis brevis are paralyzed adduction results.	Musculo-spiral.	First dorsal ...	As above.
Abductor pollicis brevis.	Oppposition of thumb.		Musculo-spiral.		
Opponens pollicis and outer portion of the flexor brevis.		No opposition movement.	Median........	First dorsal ...	As above.
Abductor pollicis brevis; flexor brevis and adductor.	Flex first phalanx and extend second phalanx (like interossei), also have an abduction and adduction action.	No flexion; if muscles are paralyzed and atrophied, ape hand is formed.	Median and ulnar.	....	As above.
Flexor Pollicis longus.	Flexes end phalanx.	No flexion of end phalanx.	Median........	....	As above.

Muscles of Back and Lower Extremities

NAME OF MUSCLE.	Innervated by	Symptoms of deficient action.
Erector spinæ; sacrolumbalis; longissimus dorsi.	Dorsal nerves. Second to twelfth dorsal segments.	Lordosis of lower spine; perpendicular line from shoulder falls behind os sacrum; unilateral palsy causes deflection of spine toward sound side.
Abdominal muscles ...	Dorsal nerves. Second to twelfth dorsal.	Lordosis with protrusion of nates and abdomen; other actions deficient; can not straighten up from recumbent position without assistance of hands.
Quadratus lumborum	Lumbar nerves	Lateral movements of lower vertebræ imperfect.
Adductor muscles.....	Obturator nerve, great sciatic and crural.	No adduction; thigh rolls outward.
Sartorius	Crural. Third lumbar segment.	Flexion impaired; acts imperfectly.
Quadriceps femoris ...	Crural. Third lumbar....	Leg can not be extended; to test it ask patient, who is lying down with hip bent, to stretch out the leg; when patient is sitting down to extend leg.
Ilio-psoas..............	Crural (lumbar plexus). Fourth lumbar.	Flexion difficult; in bed thigh can not be flexed; difficulty rising from horizontal position.
Tensor fasciæ latæ....	Superior gluteal. Fourth lumbar.	

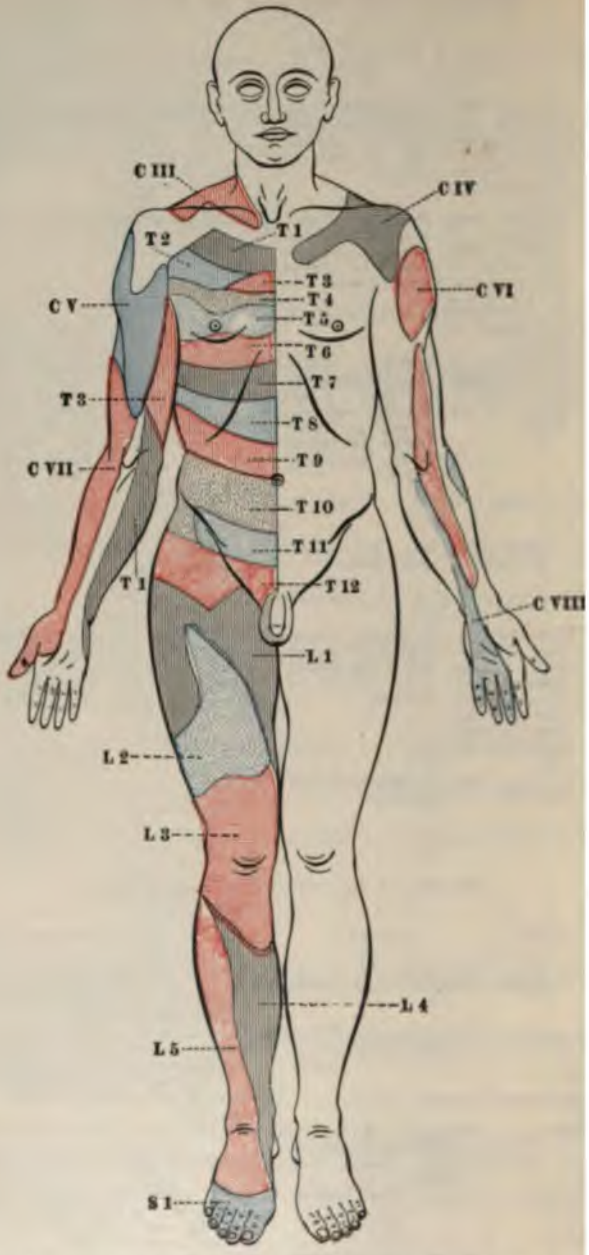


Fig. 196.—Diagram of skin areas corresponding to the different spinal seg
Combined from Head's diagrams by Osler.

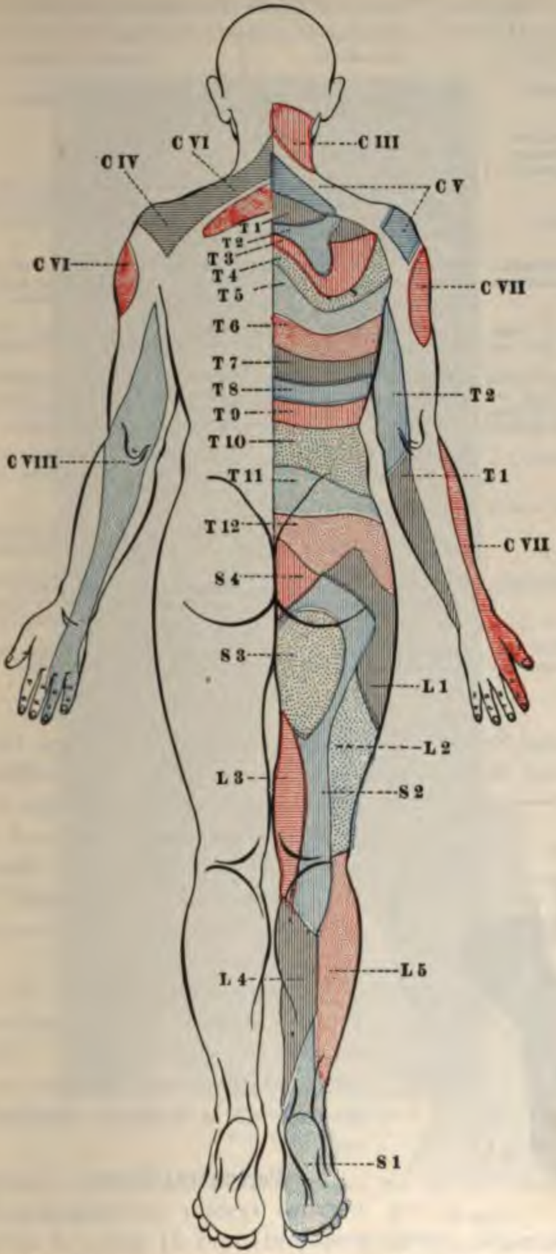


FIG. 197.—Diagram of skin areas corresponding to the different spinal segments Combined from Head's diagrams by Osler.

Muscles of Back and Lower Extremities (*continued*)

NAME OF MUSCLE.	Innervated by	Symptoms of deficient action.
External rotators: Pyriformis. Gemelli. Quadratus femoris.	Sacral plexus (muscular branches). Fifth lumbar.	Deficient outward rotation; leg turned inward.
Internal obturator. External obturator.	Obturator nerve (lumbar plexus).	
Gluteal muscles	Inferior gluteal (sacral plexus). First and second sacral. Gluteal superior. First and second sacral.	No extension of thigh; great difficulty in climbing; no abduction of thigh; waddling gait, exaggerated movement of pelvis.
Biceps; semitendinosus and semimembranosus.	Sciatic. Fifth lumbar segment.	Deficient flexion; action of quadriceps may cause excessive extension; in standing thigh is flexed to excess; trunk moved backward.
Gastrocnemius (also plantarius and soleus).	Internal popliteal. Fifth lumbar.	Deficient flexion of foot; heel can not be raised; can not stand on tiptoes.
Anterior tibial muscles (tibialis anticus, extensor digitorum, and extensor pollicis longus).	Anterior tibial. Fifth lumbar and first sacral.	Deficient extension; "dropfoot," toes scrape floor; to clear this, excessive flexion at knee and hip; contracture of flexors and pes equinus or equinovarus.
Peroneus longus.......	Peroneal. First and second sacral segments.	Deficient abduction; plantar arch lessened; increased by contracture. Flat foot; walking tiresome.
Posterior tibial muscle	Posterior tibial nerve. First and second segments.	Deficient abduction or adduction; deformities result from deficiencies.
Peroneus brevis	Peroneal. First and second segments.	
Interossei pedis et lumbricales.	Posterior tibial. First and second segments.	Abduction and adduction of toes deficient; paralysis of interossei; hyperextension of first phalanges; second and third flexed (clawed foot).
Adductor; flexor brevis and abductor hallucis.	Posterior tibial. First and second segments.	Deficient flexion of toes; foot can not be pushed off ground easily.

Segmental Sensory Localization in the Cord.—Figs. 196, 197, and 198 represent the skin areas which correspond to the different spinal segments (compare with Figs. 199 to 207). It is obvious that if areas of anæsthesia are found to exist, which, when mapped out, correspond to the areas in the diagram, one may infer the situation of the causative lesion in the cord.

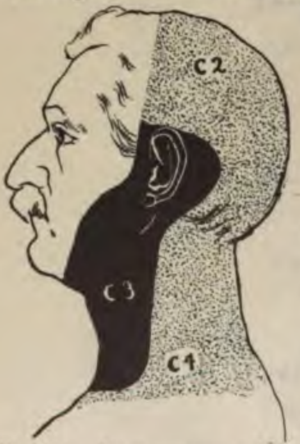


FIG. 198.—Cutaneous areas of the head and neck supplied by the 2d, 3d, and 4th cervical segments of the spinal cord.

The *automatic centres of the cord* (Fig. 223) are found in the gray matter on either side of the central canal. They are so arranged that each responds in a special manner to a definite stimulus (excito-reflex).

The Peripheral Nerves.—The peripheral nervous system includes the 12 cranial (cerebral) and 31 pairs of spinal nerves, with their root ganglia and end organs, and also the so-called sympathetic nervous system. It should be borne in mind that the latter, embracing the cranial, vertebral, and peripheral ganglia, is simply a part of the peripheral apparatus.

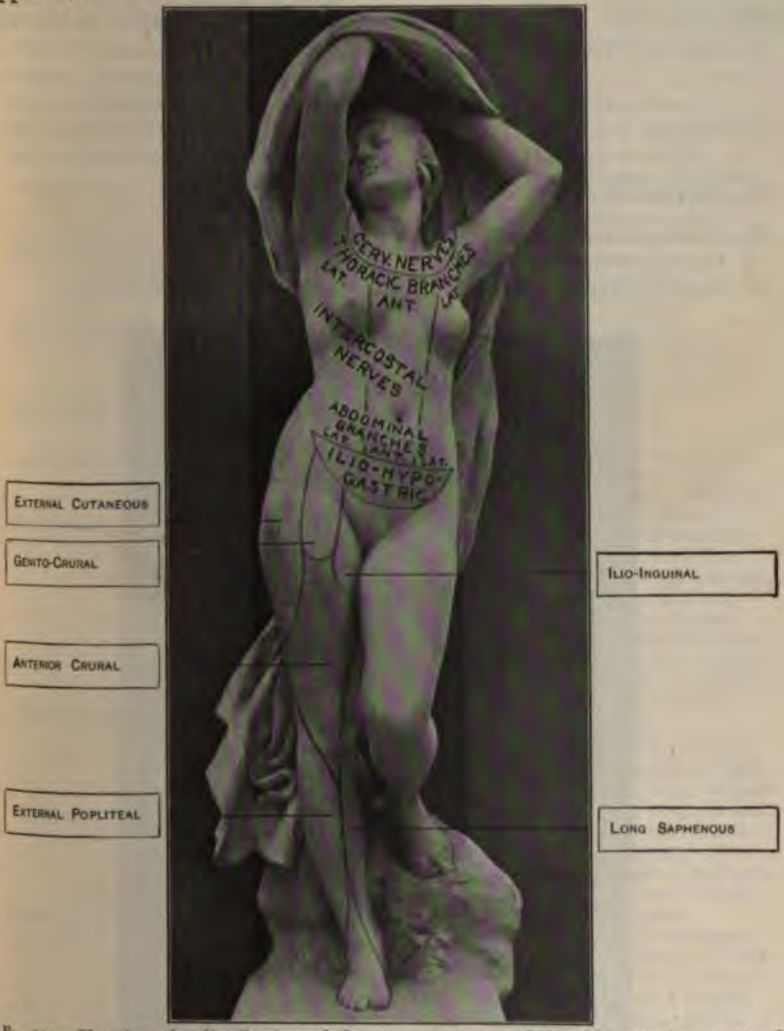


FIG. 199.—Showing the distribution of the sensory nerves of the skin, anterior aspect of trunk and leg.

(1) The *motor functions* of the *cranial nerves* are summarized in the following table:

	MUSCLES.
III. CRANIAL (MOTOR OCULI)	Sphincter iridis. Ciliary muscles. Levator palpebræ superioris. Rectus internus in convergence. Rectus superior. Rectus inferior. Inferior oblique.

	Muscles.
IV. Cranial (Patheticus)	Obliquus superior.
VI. Cranial (Abducens)	Rectus externus. Rectus intern opposite side in lateral movemen
V. Cranial (Trigeminus)	Associated movement of levator pebræ. Tensor tympani. Masseter, temporal, external ptery
VII. Cranial (Facial)	Facial muscles. Orbicularis palpe rum; muscles of external ear. St dius, platysma myoides.
XII. Cranial (Hypoglossal)	Lower facial group. Muscles of tongue.
IX. Cranial (Glosso-pharyngeal) X. Cranial (Pneumogastric) XI. Cranial (Spinal accessory)	Muscles of pharynx. Muscles of esophagus. Muscles of larynx. Trapezius.

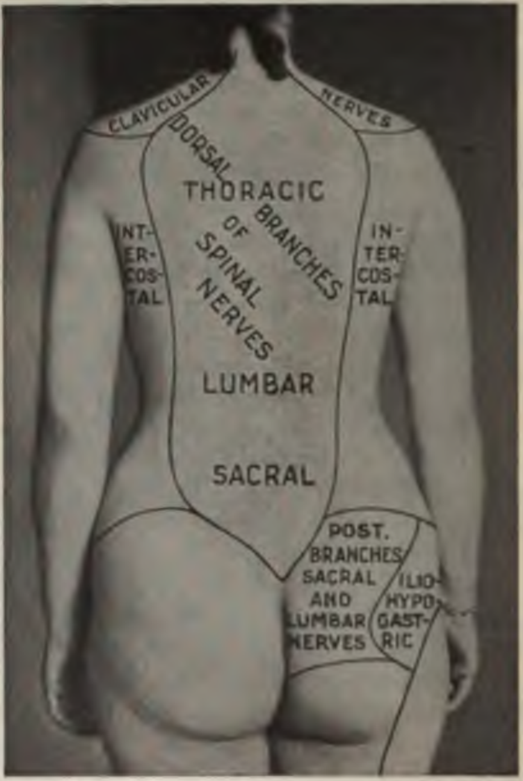


Fig. 200.—Showing the distribution of the sensory nerves of the skin, posterior asp trunk.

With reference to the *motor function* of the *mixed spinal ne* it may be desirable to know the spinal nerve by which a given mu receives its motor supply. This may be ascertained by consulting the first and fourth columns of the table on pages 526–534. On the other hand, the muscles innervated by a given nerve may be determined (for the trunk and limbs) by the following table (HUTCHISON and RAINY):

UPPER LIMB.	
POST-THORACIC	Serratus magnus.
SUPRASCAPULAR	Supra-spinatus. Infra-spinatus.
EX. ANT. THORACIC. INT. ANT. THORACIC.	Pectoralis major (upp. part, low. part). Pectoralis minor.
MUSCULO-CUTANEOUS	Coraco-brachialis. Biceps. Brachialis anticus.
SUBSCAPULAR	Subscapularis. Teres major. Latiss. dorsi.
CIRCUMFLEX	Deltoid. Teres minor.
MUSCULO-SPIRAL	Triceps. Ext. carp. rad. long. Supinator long.
POST-INTEROSSEUS	Supinator brevis. Ext. carp. rad. brev. Ext. carp. uln. Ext. comm. digit. Ext. ossis metac. poll. Ext. primi. intern. poll. Ext. secund. intern. poll. Ext. indicis. Ext. minimi digiti.
MEDIAN	Pronator radii teres. Palmaris longus. Opponens pollicis. Abductor pollicis.
MEDIAN AND ULNAR (jointly)	Flexor longus pollicis. Flexor carpi radialis. Flexor sublim. digit. Flexor brevis pollicis.
ULNAR	Flexor carpi ulnaris. Adductor pollicis. Muscles of little finger. Interossei.

TRUNK AND LOWER LIMB.	
INTERCOSTALS	Intercostals. Rectus abdominis. External oblique.
BRANCHES OF LUMBAR NERVES.	Erector spinæ. Quadratus lumborum.
GENITO-CRURAL	Cremaster.
ANTERIOR CRURAL	Sartorius. Pectineus. Rectus femoris. Vastus externus. Vastus internus. Crureus.
OBTURATOR	Gracilis. Adductor longus. Adductor brevis. Adductor magnus (with sciatic).
SMALL SCIATIC	Gluteus maximus.
SUP. GLUTEAL	Gluteus medius. Tens. vag. femoris.
GREAT SCIATIC	Biceps femoris. Semitendinosus. Semimembranosus. Adductor magnus (with obturator).
INT. POPLITEAL	Gastrocnemius. Soleus. Tibialis posticus. Flex. comm. digit. Flex. long. hallucis.
PLANTARS	Flex. brev. hallucis. Flex. brev. digit. Abductor hallucis. Adductor hallucis. Ext. brevis. digit. Interossei.
EXT. POPLITEAL.	Tibialis anticus. Ext. prop. hallucis. Ext. digit. longus. Peroneus longus. Peroneus brevis.

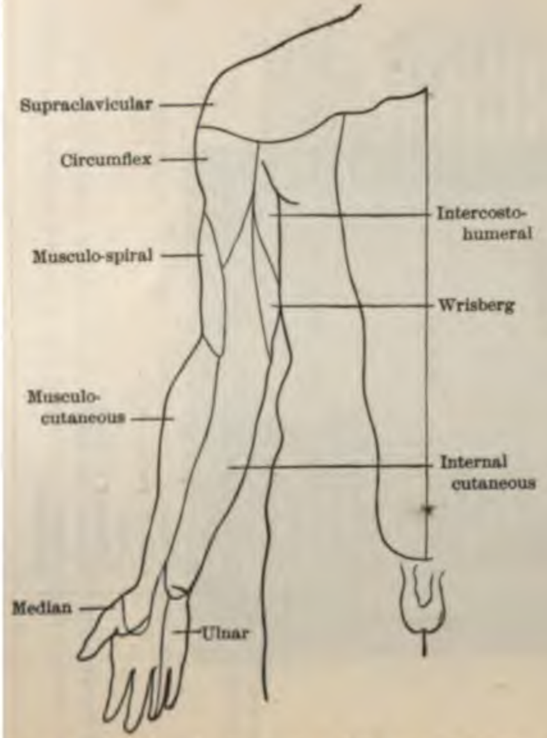


FIG. 201.—Showing the distribution of the sensory nerves of the skin of the arm, anterior aspect.

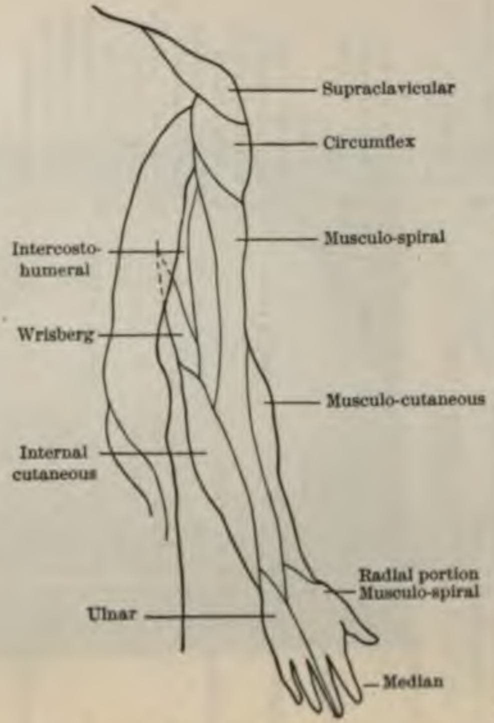


FIG. 202.—Showing the distribution of the sensory nerves of the skin of the arm, posterior aspect.

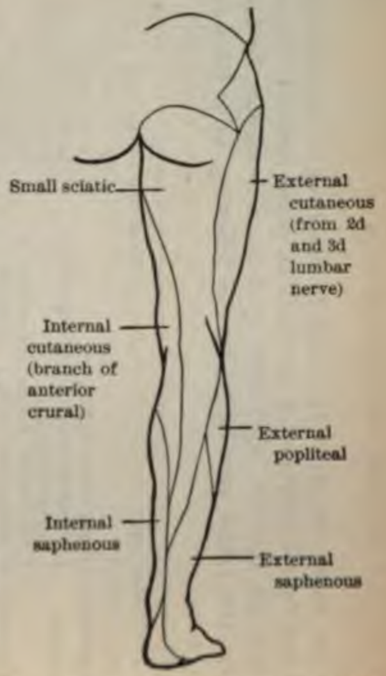
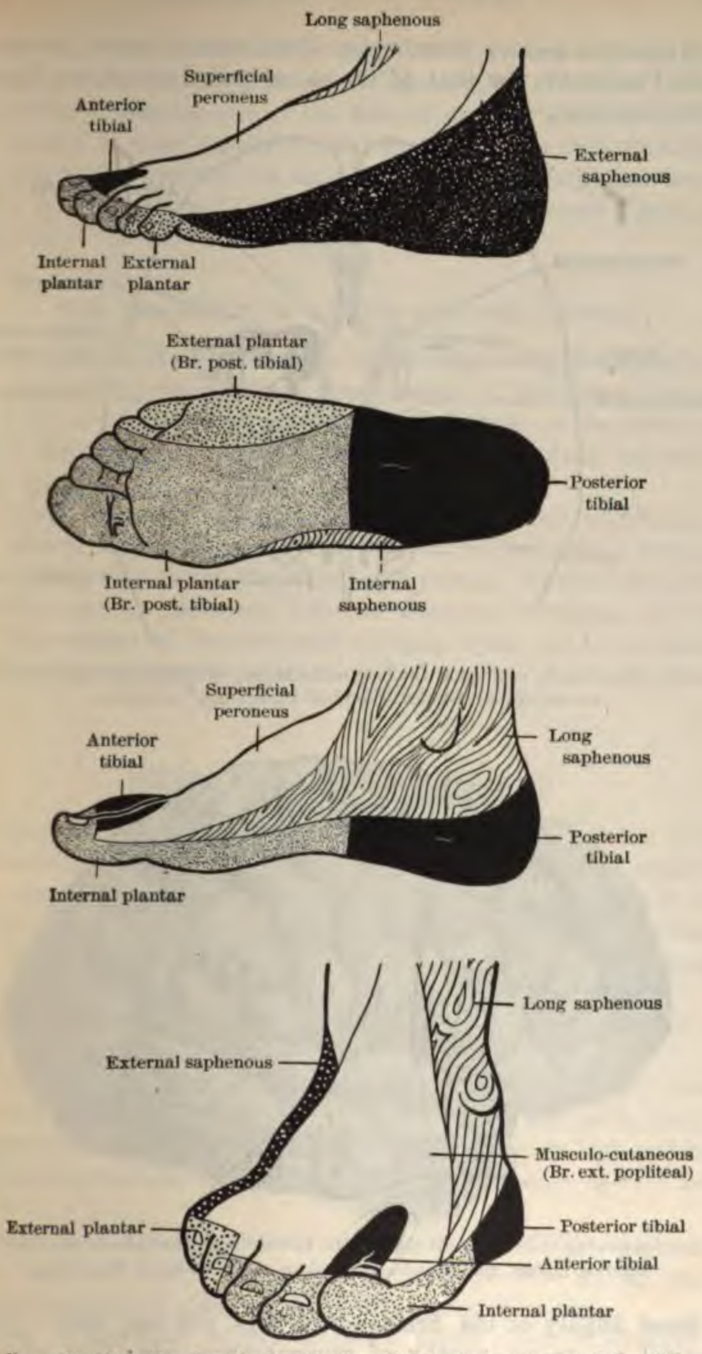

Small sciatic
Internal cutaneous (branch of anterior crural)
Internal saphenous
External cutaneous (from 2d and 3d lumbar nerve)
External popliteal
External saphenous

FIG. 203.—Showing the distribution of the sensory nerves of the skin of the leg, posterior aspect.



Figs. 204–207.—Showing the distribution of the sensory nerves of the skin of the foot. (Hasse.)

(2) For the *sensory* distribution of the *cranial* nerves, see Cranial Nerve Functions; for that of the *mixed spinal* nerves, see Figs. [illegible]98 to 207, inclusive.

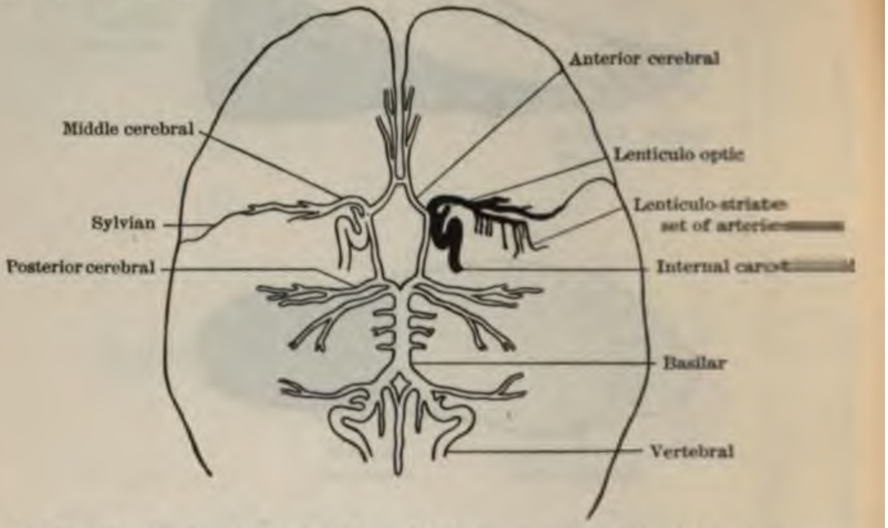


FIG. 208.—Showing the arteries at the base of the brain. Several of the lenticulo-striate set are called the "arteries of cerebral hemorrhage" (Charcot).

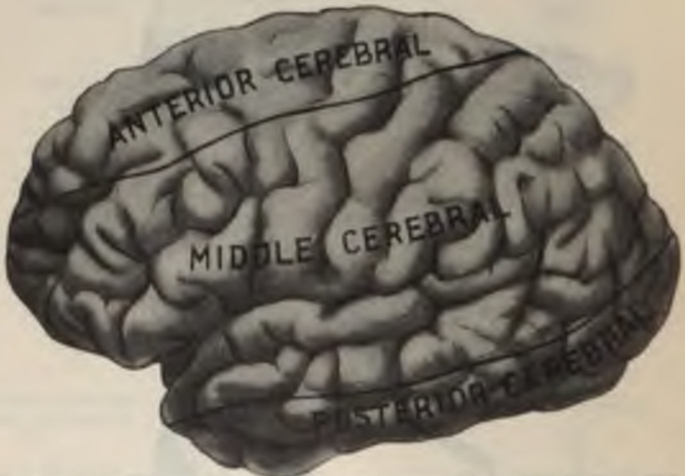


FIG. 209.—Showing the superficial portions of the cerebral hemispheres supplied by the anterior, middle, and posterior cerebral arteries. Redrawn from Dana.

Blood Supply of the Brain.—(See Figs. 208 and 209.) Certain practical points are worthy of remembrance—viz., that when the arteries are diseased some of the vessels of the lenticulo-striate group rupture more frequently than others—the arteries of cerebral hemorrhage; that in consequence of the manner of its origin the left common carotid is more readily entered by an embolus than its fellow; and that, having entered, the embolus is usually arrested in a branch of the middle cerebral, because the latter is an almost direct continuation of the internal carotid.

THE EXAMINATION OF THE NERVOUS SYSTEM

An examination of the nervous system requires an investigation of the temperament, the diathesis, the presence of deviations, the condition of the intellect and of speech, and the state of the motor and sensory functions, including the reflexes and the electrical irritability of the muscles.

Certain of these elements (*q. v.*) in the diagnosis of diseases of the nervous system have already been considered—viz., Temperament; Diathesis; Facial Expression; Emotional State; Intellection, including mental confusion, defective memory, delusions, delirium, and disturbances of consciousness (stupor, coma, etc.); Insomnia; and Disorders of Speech (aphasia, etc.).

DEVIATIONS (DEGENERACY)

There are certain anatomical, physiological, and psychic peculiarities (stigmata or marks) which may be encountered as evidences of a congenital and usually hereditary neuropathic diathesis or constitution. This condition—termed degeneration, better, deviation—is a departure from what is commonly recognised as the average type.

Anatomical Stigmata or Variations.—The physical or somatic variations of development are:

Cranium and Face.—Asymmetrical cranium, extremely small head (microcephalus), unusual configuration of the skull, lack of ordinary symmetry of the two sides of the face, a very high and narrow forehead, and excessively projecting (prognathous) or very large jaws.

Palate and Uvula.—Defects or deformities of the palate and uvula, especially a high and narrow arch of the hard palate, and a marked longitudinal ridge on the latter (torus palatinus).

Eyes.—Abnormally narrow palpebral fissure; lack of strength in the ocular muscles (muscular insufficiency or asthenopia, squint);

high grades of astigmatism, and nystagmus (rapid twitching of the eyeballs from side to side or vertically).

Ears.—Placed in unusual positions, poorly shaped or unsymmetrical; absence of lobe, the lower part of the ear directly adherent to the head; and noticeably conchoidal or shell-shaped.

Miscellaneous.—Imperfect or badly set teeth; defects in shape or size of lips and tongue; unusual shortness of height; excessively long or short fingers, arms, or legs; small, atrophic, or ill-formed genitals; absence of hair, or great hairiness; or any congenital atrophy of muscles or other anatomical deformities.

Physiological Variations.—These are: Some forms of tic (muscular twitching) or tremor, hypersensitiveness or lack of sensitiveness of the skin and special senses, defective speech—stammering, etc., perversion of the sexual instinct, and inability to endure emotional and nervous strain.

Psychic Variations.—These are: Exaggerated egotism, excessive self-consciousness, absence of will power and emotional control, disturbances of the sense of personality, and feeble, erratic, or ill-balanced mental activity.

According to the number, degree, and kind of the characteristics of deviation presented by the individual, two classes are recognised.

(1) *Superior Deviates.*—Moderate deviation is often associated with unusual mental (usually artistic) endowments, and the geniuses of the world have almost always exhibited some of the stigmata mentioned. A combination of brilliant mentality and deviate characteristics constitutes a superior deviate. Such persons may be absolutely sound in mind and enjoy average good health.

(2) *Inferior Deviates.*—This class comprises those who present not only the evidences of deviation, but are also erratic, morbid, eccentric, criminal, or insane.

The most significant of the anatomical stigmata are asymmetrical or oddly shaped crania; abnormal palates, found in 10 per cent of normal individuals, and 46 to 80 per cent of degenerates; and defective or badly set ears (found in 20 to 64 per cent of degenerates) or teeth.

With reference to the diagnostic and prognostic value of the deviate characteristics, it must be borne in mind that otherwise normal individuals may possess 2 or 3 of the anatomical stigmata, in which case their presence is of no significance. If, however, a patient exhibits a striking combination or an unusual number of the various stigmata, one is more likely to find anomalies in the neurological or psychical makeup. Such diseases occurring in degenerates have a decidedly less promising prognosis. If a child is born of two deviates it may inherit the anomalies of each.

EXAMINATION OF THE MUSCLES WITH REFERENCE TO THEIR NUTRITION, TONE, AND MOTOR POWER

Nutrition of the Muscles.—By grasping the muscles it may be determined whether they are normally large and firm or whether they are flabby and small. A diminution in their size is termed *atrophy;* an increase, *hypertrophy.* If either change is present it may involve all the muscles of a limb (diffuse atrophy or hypertrophy), or 1 or 2 muscles (circumscribed atrophy or hypertrophy). In judging the size or volume of the muscles, corresponding sides should be compared, and, when possible, tape-line measurements of the circumference of the limb are to be made. The upper arm and calf may be measured at the point of greatest girth; the forearm about 1 inch below the inner condyle; the thigh about 6 inches above the patella.

Atrophy (Fig. 210) may occur, to a slight extent, simply from disuse of the muscles, and without change in the electrical irritability; if marked (especially if the diminution is rapid) and presenting the reaction of degeneration it is indicative of a lesion of the lower motor neurones; if marked and without change in its electrical reactions it is usually indicative of primary disease of the muscles (dystrophies), or chronic disease of the joints.

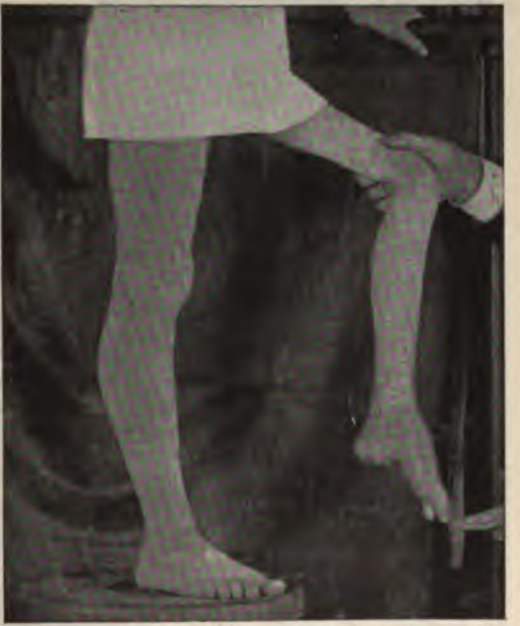


Fig. 210.—An old case of infantile spinal paralysis of the entire left lower extremity, showing extreme atrophy of the thigh and leg, and a very characteristic deformity of the foot (Holt).

Hypertrophy may be true or false. True hypertrophy is distinguished not only by increased size, but also by increased strength,

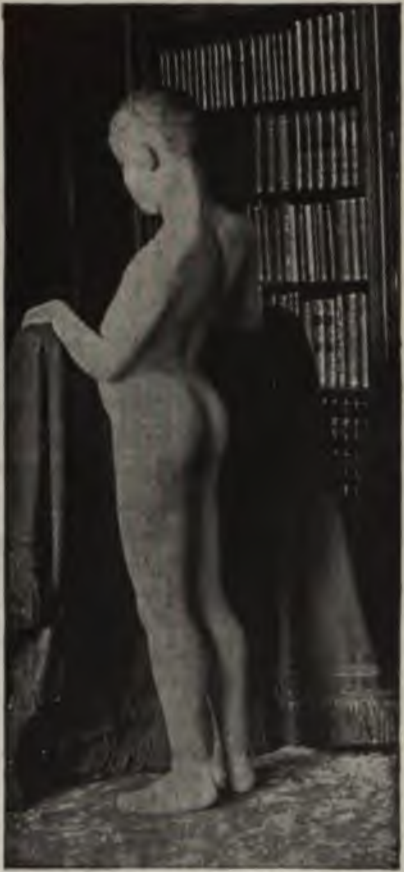


FIG. 211.—Pseudo-muscular hypertrophy, showing to a moderate degree the large calves and gluteal regions with a marked lordosis (Holt). From a photograph by Dr. M. A. Starr.

and is found in muscles which have been much, but not over
It occurs also in Thomsen's disease. Pseudo-hypertrophy p
increased size but lessened power—e. g., pseudo-hypertrophic
sis in which the calf muscles in particular are apparently bulk
211), owing mainly to a
growth of the fatty a
terstitial elements, the
cular fibres having und
atrophy.

Tone of the Musc
The normal tension of a
at rest is slight but perce
The muscular tone is
mined partly by palpatio
mainly by the degree of
ance offered to passive
of the limb. An inc
tonus or tension, usually
out atrophy, of muscles
are paralyzed so far as
tary effort is concerned (
paralysis) is an importan
of disease of the upper
rones, and if long con
constitutes one form o
tracture (*q. v.*); the op
condition — an abnorma
ness or flaccidity, usuall
atrophy—of paralyzed m
is significant of lesions i
ing certain sensory neuro

Testing the Motor
er.—The patient should
sired to sit up, to move
limb in turn, to walk, th
abling the examiner to
existing gross defects of
ity. If the patient is co
the limbs should be lifted separately and allowed to fall up
bed. If the coma is complete, it may be a matter of some dif
to determine the existence of paralysis—e. g., a hemiplegia —
may be found that one or more extremities, if paralyzed, w
in a more distinctly limp and helpless manner than others.

patient be conscious, any loss of power is readily discovered by causing him to resist passive motions designed to bring the suspected muscles into play. The loss of power may vary from a slight impairment (*paresis*) to an absolute loss of voluntary action (*paralysis*, nearly or quite complete). The degree of impairment may be accurately measured by a dynamometer, but for ordinary purposes it can be sufficiently well estimated by the hand. Always compare corresponding muscles on opposite sides of the body.

One also observes any abnormal muscular movement (page 548).

The power of individual muscles may be ascertained as follows:

(1) *Shoulder, Arm, Hand, and Fingers.—Deltoid.*—Request the patient to raise the arms laterally to a horizontal position. Inability so to do indicates deltoid paralysis.

Pectoral Muscles.—Stretch out the arms straight in front, and then approximate the hands against resistance by the examiner, meanwhile watching both heads of the pectoral muscle.

Latissimus Dorsi.—Raise the arms laterally to a level; then, while keeping them fully extended, bring the arms downward and backward, as if to make the hands meet behind the sacrum. The examiner standing behind the patient resists the movement.

Serratus Magnus.—Desire the patient to push with his hands against those of the examiner. If the serratus has lost its power the scapula will project, and the digitations of the muscle, which ordinarily should be visible, will not be seen.

Trapezius.—Ask the patient to raise the shoulders as close to his ears as possible against the pressure of the examiner's hands. This will demonstrate the strength of the upper part of the trapezius. The middle and lower portions are tested by desiring him to bring the scapulæ as close together as possible.

It is hardly possible to detect paralysis of the levator anguli scapulæ and rhomboids unless the trapezius is also involved.

Biceps.—Let the patient flex his extended arm, his elbow resting on the observer's left hand, while the latter's right hand, grasping the wrist of the patient, offers the necessary resistance. Also supinate the hand against resistance.

Triceps.—The triceps may be tested as is the biceps, excepting that the previously flexed arm is to be extended against resistance.

Supinator Longus.—Test as for the biceps, except that the hand should be midway between supination and pronation. If the muscle is paralyzed, it will fail to become conspicuous on the radial side of the upper part of the forearm.

Flexors of the Wrist.—Grasping the patient's hand, the palm being upward, desire him to bend the hand up toward his forearm against resistance.

Extensors of the Wrist.—The patient's hand being held palm downward, he is required to bend it backward against resistance. Moderate weakness of the extensors of the wrist may be manifested by asking him to squeeze the examiner's hand, in which case the wrist will become involuntarily flexed, the weakened extensors being unable to counteract the flexors. Marked or complet paralysis of the extensors is wrist-drop (Fig. 212).

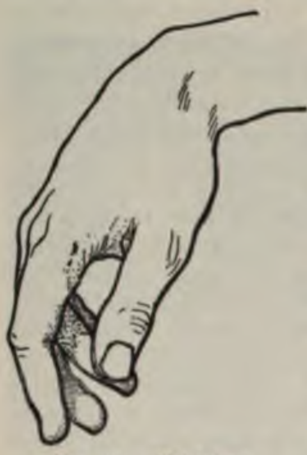


Fig. 212.—Wrist-drop.

Flexors of the Fingers.—Because of the usual difference in the strength o the two hands, the examiner should cross his forearms and place his right han in the right hand of the patient, and *vice vers* Then let the patient squeeze the hands. If th observer keeps his own fingers extended an bunched loosely together, he will be able t withstand a very hearty grasp without discomfort.

Adductor Pollicis.—Ask the patient to pinch with his thumb and forefinger, one of the examiner's fingers.

Opponens Pollicis.—Desire the patient to approximate the ends of the little finger and th thumb.

The *interosseous* and *lumbrical* muscles o the hand flex the proximal phalanges, and extend the middle and terminal phalanges. Th dorsal interossei abduct, the palmar adduct, th fingers from and toward a longitudinal lin drawn through the centre of the middle finge Test by making the patient separate and approximate the fingers, and flex the proximal phalanges, keeping the middl and terminal phalanges extended. Paralysis of these muscles causes the "cla hand" previously considered (*q. v.*).

(2) *Trunk Muscles.*—Paralysis of the diaphragm (*q. v.*) has been describe The erector muscles of the spine are examined by causing the patient to li face downward, and asking him to raise the head and shoulders without assis ance from the hands. Unless paralyzed, the erectors become clearly visib during the attempt. The abdominal muscles are tested in a similar manne except that the patient lies in the dorsal position while making an effort raise the head.

(3) *Neck Muscles.*—Practically one examines the sterno-cleido-mastoid mu cle alone. If the patient is lying down request him to raise the head; or sitting up to turn the head as far as possible to the right and left, with without resistance offered by applying the hands to the sides of the head; or bend it forward against pressure upon the forehead. If the muscle is not par lyzed it stands out prominently.

(4) *Muscles of the Eye.*—See page 214.

(5) *Muscles of the Face.*—The occipito-frontal raises the eyebrows and d velops horizontal wrinkles in the forehead. The corrugator supercilii produc vertical wrinkles over the root of the nose. The orbicularis palpebrarum shu the eyes, lightly or tightly. The orbicularis oris closes the lips, or if contrac ing strongly, closes and protrudes them. The levatores and zygomatic maj lift the upper lip and the angles of the mouth. The buccinator preserves t tension of the cheeks when blowing. Facial paralysis (*q. v.*) is described el where.

(6) *Muscles of Tongue*, (7) *Palate*, (8) *Pharynx*, (9) *Larynx*, and (10) *Mastication* (see Index).

(11) *Muscles of the Lower Extremity.—Flexors of Thigh.*—The patient lying upon his back, ask him to raise the leg up from the bed, against resistance, the knee being kept straight. This determines the strength mainly of the ileo-psoas, partly of the quadriceps.

Extensors of Thigh.—The leg being kept straight and the patient lying upon his back, raise the foot and ask him to bring it down upon the bed against resistance. This determines the strength of the gluteus maximus and partly of the hamstring muscles.

Abductors of Thigh.—Fetch the leg across the middle line and desire the patient to carry it toward the outer side against resistance, thus testing mainly the gluteus medius.

Adductors of the Thigh.—Carry the leg outward and cause the patient to bring it back to the middle line against resistance, thus testing the adductors longus, brevis, and magnus.

Inrotators of the Thigh.—With the patient prone (face downward), flex the knee to a right angle, grasp the foot and oppose resistance while he inrotates the thigh, testing mainly the gluteus minimus.

Outrotators of the Thigh.—Similarly test the power of outrotation, thus determining the condition of the obturators, pyriformis, gemelli, and quadratus femoris.

Flexors of Knee.—The patient lying upon his face, desire him to bend the knee while the examiner resists the movement by pressure upon the heel, thus ascertaining the power of the biceps, semimembranosus, and semitendinosus.

Extensors of Knee.—With the patient in the dorsal position, flex the knee and by pressure on the sole of the foot resist his endeavour to extend the knee. The quadriceps femoris is the principal muscle concerned.

Plantar Flexors (Extensors) of the Foot.—With the leg straight, resist, by pressure upon the sole of the foot, the patient's endeavour to bring the tarsus in a line with the leg, thus testing the gastrocnemius, soleus, peroneus longus and brevis. Have patient stand on toes.

Dorsiflectors of the Foot.—With the leg straight, resist the patient's attempt to bend up the foot, thus testing the tibialis anticus and the peroneus tertius. Marked paralysis of these muscles causes "foot-drop" (Fig. 213). Have patient stand on heels.

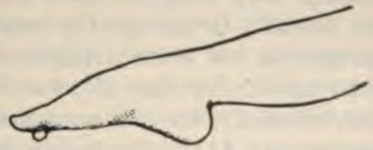


FIG. 213.—Foot-drop.

Muscles of the foot.—The flexors, extensors, interossei, and lumbricals of the toes are examined in a similar manner to those of the fingers. There is a form of claw foot analogous to the claw hand.

Having investigated the condition of the individual muscles and muscle groups as just described, and comparing the results of the examination with the table on page 537, it may be found that the paralyzed muscles correspond to the supply of a single nerve—e. g., musculo-spiral or great sciatic—or to the segmentary distribution, according to the tables on pages 526 to 534; or constitute a hemiplegia, paraplegia, or monoplegia.

MOTOR DISTURBANCES

An examination of the motor functions (mainly of the muscles) may reveal an increase or exaggeration of normal motility (*spasm*), a lessening (*paresis*), or perhaps an entire absence of motor power (*paralysis*), or an increase (*hypertonus*), or diminution (*hypotonus*), of muscle tone on passive motion.

I. Increased Motility (Spasm)

Spasm or an abnormal degree of muscular contraction may be *tonic*—continuous, and lasting from minutes to months; or *clonic*—contraction and relaxation rapidly alternating. The term *convulsion* (p. 79) is commonly applied to spasm involving the majority of the skeletal muscles; while if the contractions concern a single muscle or group of muscles it is called a local convulsion, or simply a *spasm*. If a paralyzed voluntary muscle is continuously in a state of abnormal tension it is said to be *spastic*.

The abnormal muscular movements of importance are:

(*a*) **Tremor.**—This is a more or less continuous quivering or trembling, especially of the extremities, due to a species of clonic spasm affecting a single group or many groups of muscles. Tremor of the eyeballs is nystagmus (*q. v.*). A muscle is physiologically maintained in a condition of moderate tension by rhythmic impulses passing down from the motor cell bodies at the rate of about 12 to the second. Ordinarily the resulting muscular contractions are imperceptible, but if the strength of the impulses is increased the muscular movements may be seen or felt as a tremor. If the rhythm and, especially, the rate remain the same it is a *fine* tremor, 8 to 12 per second. If the disturbance is greater, every second impulse may be lacking and *coarse* tremor (4 to 6) results.

(1) Tremor should be tested with the limb at *rest*, e. g., hand hanging down and supported at wrist, with extended spread fingers (*static* tremor); or in *motion*, finger to nose or finger to finger test, with open and closed eyes (*locomotor* tremor). Certain fine tremors may be felt better than seen. Tremor of the facial muscles may be made manifest by causing the patient to shut the eyes tightly or to show the upper teeth; of the tongue, by its protrusion. *Passive tremor* is present while the affected muscles are at rest, but during volitional motion it diminishes or may stop entirely. *Intention tremor* begins or, if present, increases, upon voluntary movement; it may be tested

by handing the patient a glass of water, desiring him to hold it a moment and then carry it slowly to his mouth, observing the effect of the performance. *Segmental tremor* is that which involves a limited portion of an extremity—e. g., fingers, or one hand and its fingers. Whether a tremor is fine or coarse can usually be estimated after a little experience, but for accurate results special laboratory instruments are required. Coarse and irregular tremor may be confused with moderate choreiform movements.

(2) The *diagnostic associations* of tremor are *in general* as follows: Intention tremor is often, but by no means always, due to organic disease; conversely, passive tremor is commonly functional. Coarse tremor is generally an evidence of organic disease or paralysis agitans, but is also seen in serious alcoholism and hysteria. Tremor of the facial muscles, lips, and tongue is indicative of marked neurasthenia, paresis, or alcoholism.

Following are some of the *special conditions* or diseases in which tremor is a noticeable symptom:

It is normal in persons of a nervous temperament under excitement or alarm, and is sometimes marked, even in robust individuals, after violent or long-continued exercise, but in both cases is temporary. It exists apparently as a constitutional peculiarity in some persons who are otherwise in good condition, becoming more distinct if the health is impaired. Senile tremor (fine) beginning in the hands and finally spreading to the neck muscles so that the head becomes involved, is not infrequent, but rarely occurs under the age of seventy.

The tremor of paralysis agitans (coarse or slow, 6 per second) affects the four extremities, sometimes the head, most commonly the hands. The thumb and forefinger usually present the movements characterized as "bread crumbling" or "pill-making." The tremor ceases during sleep and during passive and voluntary movement. Exophthalmic goitre is attended by a fine, rapid (12 per second) tremor, often to be felt but not seen. It is an important early diagnostic sign. Volitional or intentional tremor (coarse or slow) is especially characteristic of disseminated sclerosis, most marked in the hands and arms, but occurring also in the legs and head. When the patient is absolutely quiet the tremor may disappear. The tremor of hysteria (rapid or fine, 8 to 12 per second) usually affects the hands and arms, less frequently the head and hands. The tremor may be volitional, resembling that of disseminated sclerosis.

Tremor is often significant of the overuse of tea, coffee, alcohol, and tobacco, as well as poisoning by lead, mercury and, less frequently, arsenic and opium. Alcoholic tremor affects especially the tongue and extremities. It may be fine or coarse, and often manifests itself only upon movement. Tremor is an important symptom in plumbism and mercurialism. It is seen also in opium or morphine eaters, if the customary supply of the drug be stopped.

(*b*) **Fibrillary Tremor.**—This is a more or less rhythmic twitching or tremor confined to certain fibres, bundles or parts of a muscle. Most commonly it is seen in the tongue, the facial muscles, and the muscles of the extremities. It indicates great exhaustion of a muscle or wasting of the muscle from lack of neurotrophic influence.

(*c*) **Athetosis or Athetoid Movements.**—These are slow, continuous, unconscious, somewhat forcible twisting movements, usually of the fingers and hands (Fig. 214), less frequently of other muscles, and are sometimes painful. The fingers and toes flex and extend, the hands are pronated and supinated in an irregular bizarre manner. They are usually associated with increased tonus of the limbs. The movements stop during sleep and often remit during the day.

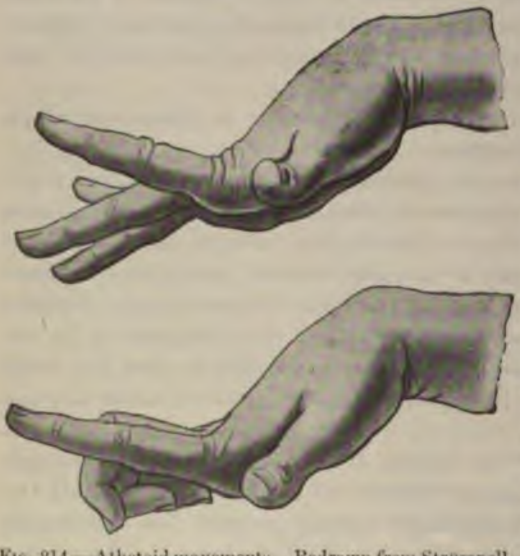


FIG. 214.—Athetoid movements. Redrawn from Strümpell.

Athetosis is a motor irritation symptom of organic disease of the brain, usually hemorrhage in the neighborhood of the optic thalamus, the lenticular nucleus, or the posterior part of the internal capsule. It may be present on both sides as a result of infantile cerebral diplegia, or it may be on one side (hemiathetosis) from cerebral hemorrhage or local softening (hemiplegia, paresis).

(*d*) **Localized Convulsive Seizures.**—A sign which may be of great importance is the so-called Jacksonian or localized epilepsy, a clonic convulsion beginning in a single muscle or group of muscles—e. g., the face, fingers, or toes—preceded by pain or tingling in the part, extending so as to involve the entire extremity and sometimes more than one. The convulsive movements are usually confined to one side. The patient retains consciousness except in the comparatively infrequent cases in which general convulsions follow. A typical attack of this kind is very significant of a localized source of irritation in the sensori-motor zone of the cerebrum. The causative lesion may be brain tumour, softening, localized meningitis, hemorrhage, abscess, and injury. It may follow hemiplegia in children. That localized epilepsy does not necessarily imply a limited lesion is shown by the fact that it may occur typically in general paresis and uræmia.

Here also may be mentioned the irregular and usually moderate twitchings of various muscles, most commonly those of the face, arms, and hands, less frequently of the feet, which may be witnessed in high fever, especially in the gastro-intestinal diseases of children; the typhoid status (*subsultus tendinum*), uræmia, meningitis, jaundice, delirium tremens; and as a result of full medicinal doses of strychnine, particularly in neurotic patients. Picking at the bedclothes or attempting to seize invisible objects in the air (*carphologia*) is seen in the typhoid status and is a sign of serious weakness, perhaps of impending death.

(*e*) **Choreic Movements.**—These are abrupt twitchings or jerking movements of different muscle groups, involuntary and without an object. Affecting one lateral half of the body it is called hemichorea. A child suffering from chorea appears restless, unsettled, and fidgety. If the choreic movements are slight, it may be necessary, in order for their detection, to have the patient lay his hands, palms down, upon the hands of the examiner, when, after a short wait, small twitching movements of the fingers may be perceived. The handwriting is apt to be impaired. Ordinarily choreic movements cease during sleep, are often diminished by voluntary movement, and always increased by mental excitement or bodily fatigue. *Tics* are choreic movements of certain groups of muscles physiologically associated for the performance of a definite function, affecting most commonly the muscles of expression, less frequently those of speech, respiration, and locomotion.

The most ordinary cause of choreic movements is the acute chorea of children (Sydenham's C., St. Vitus's Dance). Other affections which present choreic movements as a symptom are habit spasm; tic convulsif (Gilles de la Tourette's disease); chronic (Huntington's) chorea; saltatory spasm (Latah, Jumpers); post-hemiplegic chorea (hemichorea), occurring under the same conditions as hemiathetosis (*q. v.*); rhythmic chorea (really hysteria), more or less regular spasms of certain muscle groups, e. g., nodding spasm when the neck muscles are affected, salaam convulsions when the abdominal muscles are involved; and the seldom seen electric chorea

(Dubini). Here also may be mentioned a peculiar affection, paramyoclonus multiplex, characterized by either constant or paroxysmal clonic contractions, sometimes very violent, mainly of the muscles of the extremities.

(*f*) **Cramp.**—If a localized spasm is painful, it is called cramp. It most commonly affects the calf muscles, and may be due to muscular exertion, alcoholism, nephritis, gout, diabetes or hysteria.

(*g*) **Forced Positions or Movements.**—A person may be more or less suddenly thrown forward, backward, or sideways, or forced to move in a circle, by involuntary muscular action; or by a tonic contraction, which draws the head and trunk to one side, is compelled to lie in a lateral position. Such imperative movements or positions are significant of a lesion of the vermis (or middle lobe) of the cerebellum. With imperative or forced movements may also be classed the screaming, laughing, or jumping spasms of hysteria and epilepsy.

(*h*) **Associated Movements.**—In certain cases when an extremity is paralyzed—e. g., hemiplegia—movement of the corresponding unaffected limb of the opposite side may produce similar but less extensive movements of the paralyzed extremity.

(*i*) **Myoidema.**—A smart blow, delivered by finger or hammer upon a muscle near its tendinous attachment, may under certain circumstances cause a sudden localized contraction of the muscular fibres (myoidema); or, if the belly of the muscle is struck, a similar belt of contraction may appear (idiopathic muscular spasm), in either case lasting only a few seconds. These phenomena are evidences of the exaggerated irritability of muscles which are undergoing rapid wasting, especially in phthisis.

(*j*) **Tetany.**—The disease known by this name (*q. v.*) is a paroxysmal tonic spasm usually confined to the flexors of the hands and feet, but sometimes involving the muscles of mastication (trismus). Carpopedal spasms in rickety children are by many writers classed as a mild tetany. When affecting the upper extremities the fingers assume a characteristic position, "the accoucheur's hand" (Fig. 215). In the foot the toes are strongly flexed.

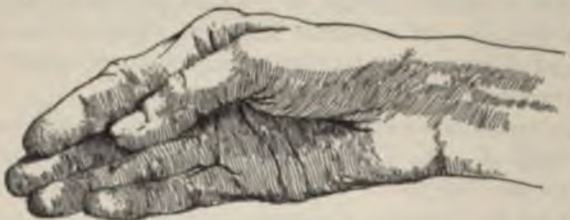


Fig. 215.—Hand of tetany.

A form of tonic cramp of the muscles, which is not tetany, characterizes Thomsen's disease (myotonia). The cramp occurs only upon voluntary movement, the muscles stiffening and responding very slowly to the behest of the will. A similar localized condition may be seen in writer's cramp, the muscles becoming rigid when called upon to act.

(*k*) **Catalepsy.** — This is a peculiar form of muscular rigidity or increased muscular tonus affecting the voluntary muscles. An affected limb may be moved with but slight resistance, and will maintain, in opposition to gravity, for an hour or even longer the position in which it has been placed, the so-called "waxy flexibility" (*cerea flexibilitas*). It is a condition which is most frequently seen as a symptom of hysteria, or of several psychoses, notably catatonia and paresis. Rarely it may occur in connection with brain tumour and meningitis.

(*l*) **Contractures.** — A contracture (Fig. 216) is a tonic muscular spasm of long standing. The spasm may be so slight that the resistance of the affected part to passive motion is hardly perceptible, or so strong that movement becomes impossible. If the contractured extremity—finger, arm, foot, or leg—be suddenly extended by the examiner, a curious "check" may be felt. In contractures of considerable duration there is in all likelihood an actual or anatomical shortening of the affected muscles.

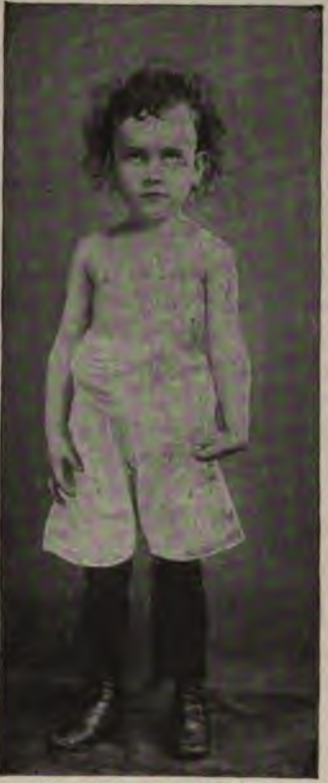


FIG. 216.—Deformity of left hand, the result of contractures following an attack of hemiplegia four years before; child seven years old (Holt).

Contractures are most commonly seen either as a symptom of hysteria or, affecting the palsied muscles, as a result of cerebral paralysis, hemiplegia (*q. v.*) in particular. If functional, the contracture disappears during sleep or anæsthesia; if of organic origin, it will persist. Contractures affecting the arms may be seen in chronic hydrocephalus. The contracture may affect the paralyzed muscles or healthy muscles whose antagonists have lost their contractility, or may be present in the strongest muscles of a paralyzed group. Contractures and spastic rigidity of the muscles are further considered in connection with the various forms of paralysis (*q. v.*).

II. Decreased Motility (Paralysis)

A partial or total loss of voluntary motor power is called *paralysis*. A partial loss is often termed *paresis* (not to be confused with general paresis of the insane). *Amyosthenia* is a sudden, usually temporary, weakness of an arm or of both legs, and is a symptom of hysteria. *Hemiplegia* is a paralysis of one lateral half of the body. Alternating or *crossed hemiplegia* signifies paralysis of the extremities of one side and the facial, ocular, or other muscles of the opposite side. *Diplegia* is a double hemiplegia. *Quadruplegia* is a paralysis of all four extremities. *Paraplegia* used without qualification means paralysis of both lower extremities. Some writers speak of paralysis of both legs as paraplegia inferior; of both arms as paraplegia superior. *Monoplegia* is a paralysis of one extremity—e. g., brachial = of the arm, crural = of the leg, facial = of the face. It may be single or double—e. g., of both arms = double brachial monoplegia = also paraplegia superior.

In the examination of a case of paralysis the following points must be determined: (1) The limb or limbs, the muscle or muscle groups involved—the extent of the paralysis. (2) Is the paralyzed limb or muscle group *spastic* or is it *flaccid?*—an extremely important point. If the limb resists attempts at passive motion because of existing contraction of the muscles, or if the muscles contract in opposition to the attempted movement, the paralysis is spastic. If, on the other hand, the member is limp, the muscles are lax, and there is no resistance to passive motion, the paralysis is flaccid. (3) Are the muscles atrophied? (4) What is the condition of the reflexes (*q. v.*)? (5) How do the paralyzed muscles react to electricity (*q. v.*)? (6) Are there sensory disturbances (*q. v.*)?

(*a*) **Paralysis according to its Type.**—Having obtained the required data according to the foregoing scheme in a satisfactory manner, it is usually possible to assign all cases of paralysis to one of two *types*, viz., *cerebral* or *spinal*. Such an assignment does not in all cases imply of necessity a cerebral or spinal lesion, for the paralysis may be due to inflammation of the peripheral nerves (neuritis), or to primary disease of the muscles and the terminal motor filaments embedded in them (intramuscular paralysis or muscular dystrophy), or it may be psychic (hysteria). Nevertheless, a determination of the type of the paralysis is a clinical prerequisite to a more particular diagnosis of the exact condition upon which it depends—a diagnosis which is to be made by a consideration of the associated symptoms.

(1) *Upper Neurone* (*Central, Cerebral, or Spastic*) *Paralysis.*—The clinical features of cerebral paralysis are: *Hemiplegia*, partial or total,

the most characteristic distribution of this type of paralysis. The paralyzed muscles are *spastic*, and *contractures* may develop. The *reflexes* are *exaggerated*. The *reaction* of the muscles to electric stimulation is *normal*. *Atrophy* of the muscles, if it occurs, is *slight*, and in the large majority of cases is due simply to their disuse.

The cerebral type of paralysis is due to lesions affecting the central (upper) motor neurones. The destructive lesion may involve the cell bodies proper, or the axones in any part of their length, including the terminal end brushes, and is followed by a secondary degeneration of the axis cylinders below the diseased point (descending degeneration). It should be clearly understood that disease affecting any part of the upper motor path gives rise to the cerebral or spastic type of paralysis (Figs. 219 and 225). Thus in sclerosis affecting the lateral pyramidal columns (Figs. 193 and 194), although the disease is in the cord the type of the paralysis is cerebral, because the pyramidal column is composed of the axis cylinders of the central (cerebral) neurones. If the disease extends outside of these columns, the symptoms of the peripheral type may be superadded, one usually predominating according to the course of the malady.

Cerebral paralysis is spastic and attended with contractures, because the peripheral or lower neurones are deprived of the controlling and inhibiting influence of the central neurones (Fig. 189), and for the same reason the reflexes are exaggerated. As the muscles still receive the trophic and other influences of the peripheral neurones, they do not undergo atrophy and degeneration; consequently the electrical reaction is normal.

Hemiplegias are usually of cerebral origin; diplegias or paraplegias, usually of spinal origin. Unilateral cord lesions, pressure from tumour, etc., cause a hemiplegic syndrome. In cerebral hemiplegias the lesion is on the opposite side.

(2) *Lower Neurone* (*Peripheral*, *Spinal*, *Flaccid*, *or Atrophic*) *Paralysis*.—The clinical symptoms of peripheral paralysis are: *Paraplegia*, the most characteristic variety of this form of paralysis. The paralyzed muscles are *flaccid*, having lost their tone. The *reflexes* are *diminished* or *lost*, and there is an entire or partial *loss of response* to the *faradic* current, with a partial or complete *reaction of degeneration* to the *galvanic* current. *Marked atrophy* and wasting of the paralyzed muscles. Peripheral palsies may be one-sided.

Paralysis of the peripheral (spinal) type (Figs. 219 and 225) may be due to a lesion of any part, cell bodies or axones, of the peripheral (lower) motor neurones, including those of the cranial motor nuclei in the pons and medulla (Figs. 217 and 218) as well as the motor cells of the anterior horns of the spinal cord. Consequently this type is seen if the cell bodies are involved, as in anterior poliomyelitis (Fig. 194) or polioencephalitis; or if the peripheral nerves are diseased as in neuritis. If the disease begins primarily in the muscles, involving also to some extent the nerve endings which they contain, as in the muscular dystrophies, the paralysis is of peripheral type, except that the reaction of degeneration is rarely observed, and an apparent enlargement (pseudo-hypertrophy) may be present in the early stages of the malady; the muscles in reality being atrophied.

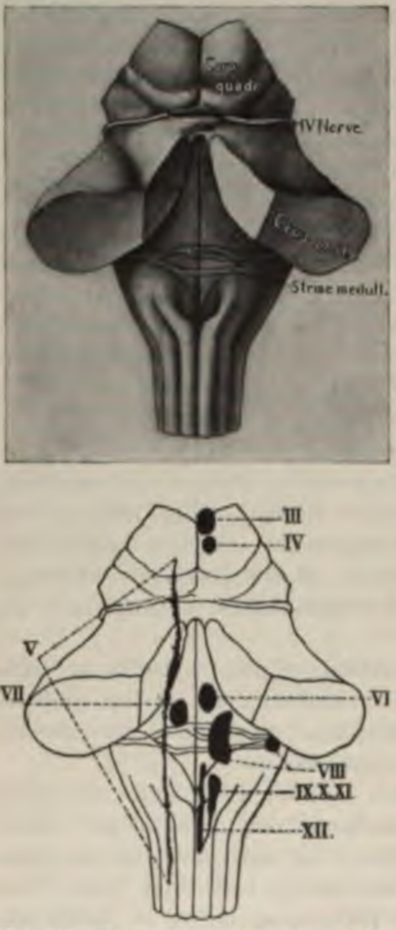


FIG. 217.—Diagrams showing the relative positions of the cranial nerve nuclei, posterior aspect. Redrawn from Merkel.

The explanation of the clinical symptoms of the peripheral type of paralysis lies in the fact that the peripheral motor neurones and the muscles which they innervate constitute neuromuscular trophic units. The cell body of the lower motor neurone not only maintains the tone or tension of the muscle, but also exercises a trophic or nutritive action upon the latter. If, therefore, the integrity of the cell body or its axis cylinder is impaired by injury or disease the tonic impulses received by the muscle cease and it loses its tone—i. e., becomes flaccid—and the reflexes are abolished. Furthermore, as it is deprived of the necessary trophic influence, atrophy takes place and it presents the reaction of degeneration.

(3) *Summary of the Diagnostic Significance of the Type of Paralysis.*—The **cerebral type** indicates: A lesion of the motor cortex and pyramidal tracts, usually unilateral (hemiplegia), sometimes bilateral or symmetrical (diplegia, especially in children); or an affection of the crossed pyramidal column of the cord (Figs. 193 *a* and *b*), the

latter being to all intents and purposes a continuation of the cerebral motor tract.

The **peripheral type** indicates: Lesions of the motor cell bodies of the medulla and pons nuclei (e.g., bulbar paralysis and ophthalmoplegias, classed as polioencephalitis). Lesions of the motor cells in the anterior horns (e.g., poliomyelitis, acute and chronic). Neuritis of the peripheral nerves, with, in the acute forms at least, disturbances of sensation which are lacking in poliomyelitis (an important differential symptom). Muscular dystrophies, which, as the paralysis originates in the muscle itself, do not present the reaction of degeneration, and there is apparent hypertrophy but real atrophy, wherein they differ from poliomyelitis and neuritis.

A *mixture* of these types may be found in amyotrophic lateral sclerosis where there is rapid muscular atrophy with spasticity and exaggerated reflexes, the disease involving simultaneously the lower ends of the upper and the cell bodies of the lower neurones; and transverse myelitis, in which the muscles innervated from the site of the lesion exhibit the spinal type (Fig. 219), while below the level of the lesion the spastic variety of paralysis is found.

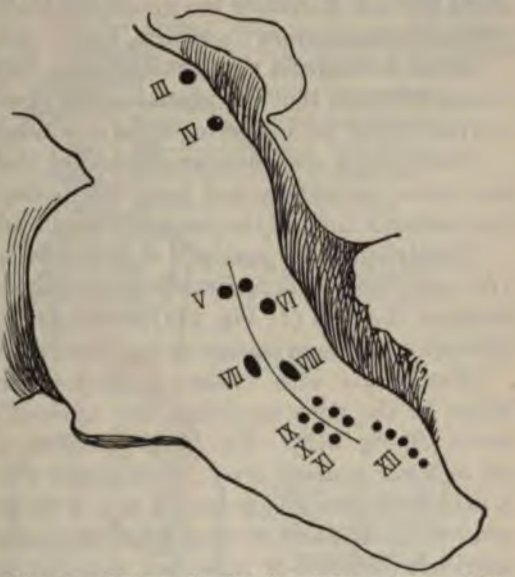


FIG. 218.—Diagram showing the relative positions of the cranial nerve nuclei, lateral aspect.

The functional paralyses (hysteria, strong emotion) are usually flaccid, but do not exhibit atrophy or the reaction of degeneration, and the deep reflexes are variable, usually preserved or exaggerated. The presence of contractures and anæsthesias and the peculiar psychic condition of the patient are serviceable in making the diagnosis. Hysteria is capable of resembling very closely nearly every type of organic disease.

(*b*) **The Distribution of the Paralysis with reference to Topical Diagnosis and Particular Forms of Disease.**—As a rule, if the paralysis is unilateral it is due to a cerebral lesion; if bilateral, to disease of the spinal cord, except in the diplegias (double hemiplegias) of children.

(1) *Hemiplegia.*—Complete paralysis of one lateral half of the body (face, arm, and leg), unless functional, is always due to a cerebral lesion. There are certain varieties of this form of paralysis which serve to localize the causative lesion.

Complete hemiplegia (see *B*, Fig. 219) is caused by a lesion of the knee and anterior two thirds of the posterior limb of the internal capsule, the motor fibres from the sensori-motor area being at this point gathered into a narrow band. Consequently a comparatively small lesion will involve the pyramidal tract fibres which have a very extensive distribution (face, arm, leg).

Right hemiplegia plus aphasia (page 263) in a right-handed individual indicates that the lesion further involves the left third frontal convolution or its connecting fibres (see also Figs. 61 and 62, page 266).

Hemiplegia *plus* hemianæsthesia of the same side indicates that the lesion has encroached upon the posterior one third (sensory) of the posterior limb of the internal capsule (*G*, Fig. 219).

Hemiplegia with paralysis of the third nerve on the opposite side (*H*, with crossed or alternating oculo-motor paralysis) indicates a lesion of the crus (*C*, Fig. 219) involving the third nerve as it passes through the crus to emerge at the inner border of the latter (Fig. 45).

Hemiplegia with crossed (opposite) facial paralysis signifies a lesion in the pons involving the nucleus of the seventh or facial nerve (*D*, Fig. 219). The facial paralysis in this case is of the flaccid or lower neurone type. Hemiplegia with double facial paralysis is extremely rare, and is caused by a pons lesion involving both right and left facial fibres at their point of decussation. Hemiplegia with anarthria (difficult or imperfect articulation, not aphasia) and difficulty in swallowing is due to a lesion of the bulb (medulla). Double hemiplegia (diplegia) is due to bilateral or symmetrical cerebral lesions, and is of notable occurrence in children.

In the majority of cases hemiplegia is due to cerebral hemorrhage, embolism, or thrombosis, associated with arteriosclerosis, and followed by softening. Syphilis may be a factor in the vascular changes. It may be due to brain tumour, multiple sclerosis or sclerosis of one hemisphere, meningeal hemorrhage or suppuration, inflammation, Raynaud's disease, and general paresis of the insane. It may be functional from hysteria, and transient from uræmia. It is a very infrequent event in the last stages of carcinoma and pulmonary tuberculosis, and 2 cases have been reported (JANEWAY) as a result of the injection of hydrogen peroxide into the pleural cavity (oxygen embolism). An acute and sudden hemiplegia is usually the

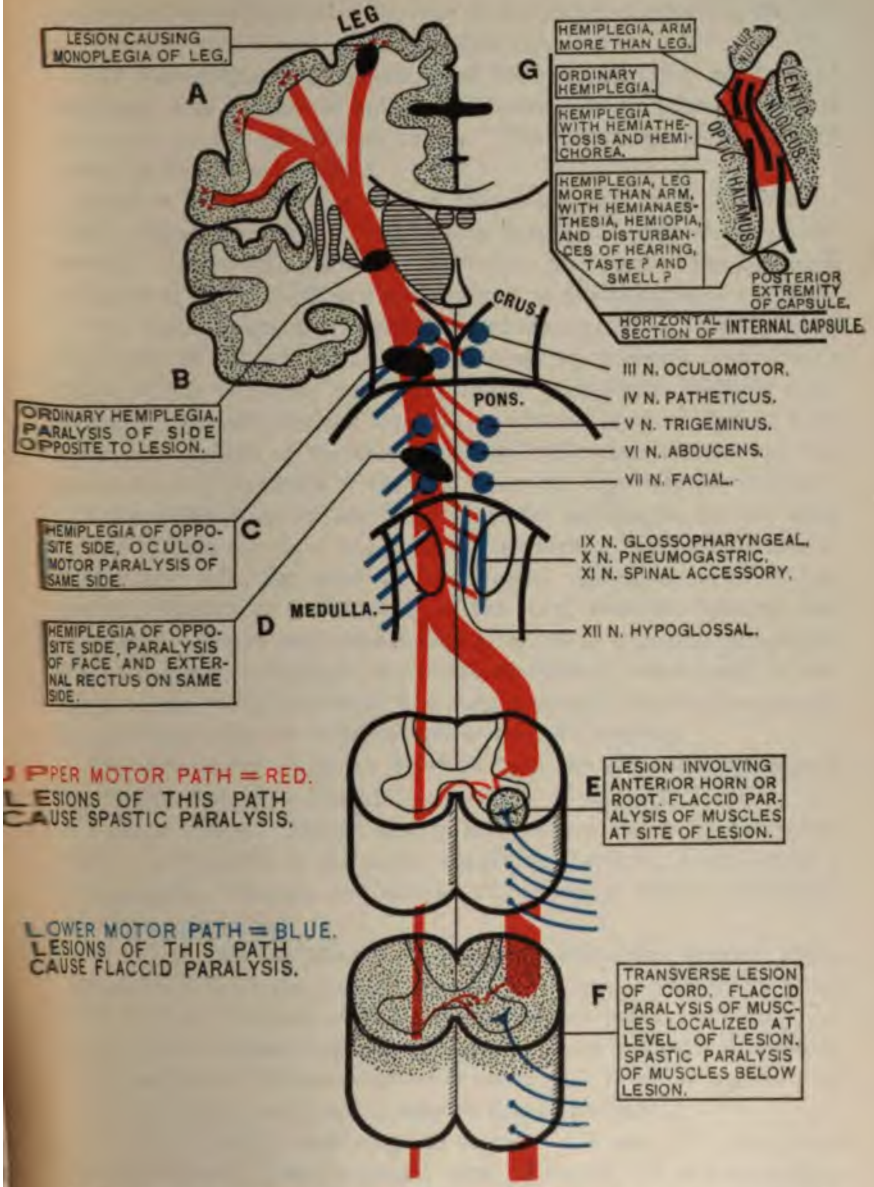

LESION CAUSING MONOPLEGIA OF LEG.
LEG
A
G
HEMIPLEGIA, ARM MORE THAN LEG.
ORDINARY HEMIPLEGIA.
HEMIPLEGIA WITH HEMIATHETOSIS AND HEMICHOREA.
HEMIPLEGIA, LEG MORE THAN ARM, WITH HEMIANAESTHESIA, HEMIOPIA, AND DISTURBANCES OF HEARING, TASTE ? AND SMELL ?
CAUD. NUC.
LENTIC. NUCLEUS.
OPTIC THALAMUS.
POSTERIOR EXTREMITY OF CAPSULE.
HORIZONTAL SECTION OF INTERNAL CAPSULE.
CRUS.
B
ORDINARY HEMIPLEGIA. PARALYSIS OF SIDE OPPOSITE TO LESION.
PONS.
III N. OCULOMOTOR.
IV N. PATHETICUS.
V N. TRIGEMINUS.
VI N. ABDUCENS.
VII N. FACIAL.
C
HEMIPLEGIA OF OPPOSITE SIDE, OCULOMOTOR PARALYSIS OF SAME SIDE.
IX N. GLOSSOPHARYNGEAL.
X N. PNEUMOGASTRIC.
XI N. SPINAL ACCESSORY.
D
MEDULLA.
HEMIPLEGIA OF OPPOSITE SIDE, PARALYSIS OF FACE AND EXTERNAL RECTUS ON SAME SIDE.
XII N. HYPOGLOSSAL.
UPPER MOTOR PATH = RED.
LESIONS OF THIS PATH CAUSE SPASTIC PARALYSIS.
E
LESION INVOLVING ANTERIOR HORN OR ROOT. FLACCID PARALYSIS OF MUSCLES AT SITE OF LESION.
LOWER MOTOR PATH = BLUE.
LESIONS OF THIS PATH CAUSE FLACCID PARALYSIS.
F
TRANSVERSE LESION OF CORD. FLACCID PARALYSIS OF MUSCLES LOCALIZED AT LEVEL OF LESION. SPASTIC PARALYSIS OF MUSCLES BELOW LESION.

FIG. 219.—Showing the effects of various lesions of the motor path in the brain and spinal cord. The upper right-hand sketch shows the variations in symptoms caused by a difference in the antero-posterior position of the lesions in the internal capsule.

result of hemorrhage, embolism, or thrombosis; if gradual and progressive, of unilateral brain tumour or an island of sclerosis, the latter, perhaps, only a part of the multiple form.

(2) *Paraplegia.*—Paraplegia, used here as indicating paralysis of both legs, may be due to spinal-cord lesions or to bilateral disease (neuritis) of the peripheral nerves. The following points are of service in the discrimination.

Spastic paraplegias are almost invariably due to spinal-cord lesions. Flaccid paraplegias may be due to spinal disease or peripheral neuritis. If the sphincters of the bladder and rectum are involved, the lesion is in the cord, and not confined to the peripheral nerves.

The diseases which are especially characterized by spastic paraplegia are lateral sclerosis, spinal meningitis, pressure from Pott's disease of the vertebræ, hereditary ataxic paraplegia, multiple cerebro-spinal sclerosis, chronic myelitis, transverse myelitis (below level of lesion), syphilis of the cord, combined scleroses, tumour, and the spastic cerebral paralysis of children when the legs only are involved.

Other diseases which are responsible for paraplegia, for the most part of the flaccid type, are Landry's paralysis, Friedreich's ataxia, hemorrhage into the cord or the spinal meninges, acute poliomyelitis, tumour or tuberculosis of the cord, neuritis, hysteria and diphtheria. Scurvy and rickets may give rise to a pseudo-paraplegia. There is a senile paraplegia due to arteriosclerosis, thrombosis, or embolism of the spinal arteries. The most common causes of paraplegia are, perhaps, alcoholic neuritis and acute poliomyelitis.

The differential diagnosis depends upon the associated symptoms.

(3) *Monoplegia.*—Brachial.—Paralysis of one arm is rarely due to a spinal lesion. Almost always it is caused either by a cerebral lesion or by neuritis of the nerves supplying the arm. Occasionally a syringomyelia, tumour, or cervical rib causes a slowly developing brachial monoplegia.

If due to cerebral disease (hemorrhage, embolism, tumour, etc.), the paralysis is of the spastic, non-atrophic type. The lesion is almost inevitably cortical or lies a short distance below the cortex. A lesion in the internal capsule would need to be narrowly limited to cause paralysis of the arm without involving the face and leg fibres as well. Like other paralyses, it may be due to hysteria.

If due to neuritis involving the nerves of the arm, the paralysis is of the peripheral (lower neurone) type. Neuritis (of various origin) may and usually does affect special nerves (e. g., musculo-spiral, median). Consequently certain groups of muscles which are innervated by the inflamed nerve will be paralyzed, other groups supplied by the uninvolved nerves escaping—partial brachial monoplegia.

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Bilateral brachial monoplegia is usually due to the neuritis of lead poisoning; pressure *palsies* (crutches), tumour, and *syringomyelia* are occasional factors.

Crural Monoplegia.—Paralysis of one leg alone is rarely caused by a cerebral lesion. If its cerebral origin can be established (by concomitant symptoms), the lesion will be found in or beneath the cortex in the paracentral lobule and at the upper end of the fissure of Rolando (Fig. 61, and *A*, Fig. 219).

More commonly it is due to disease of the cord, or neuritis involving the nerves of the leg. The spinal causes are myelitis, tumour, syringomyelia, or poliomyelitis. The latter in particular is responsible for loss of power in special muscle groups or individual muscles. Neuritis from disease or traumatism (blows, pressure) may also be the source of a limited paralysis of the leg muscles. A pelvic tumour by pressure on the crural nerve may give rise to paralysis; and in locomotor ataxia the function of certain nerves may be abolished. A false or apparent monoplegia, usually not a source of error, may arise from painful affections of the limb—e. g., rheumatism or phlebitis—by interfering with its normal mobility.

Facial Monoplegia.—Upper neurone (cerebral) paralysis of one side of the face (and tongue) is due to a lesion of the lower part of the anterior central convolution (Fig. 61). The causes of facial paralysis have been previously discussed in detail (page 185).

SENSORY DISTURBANCES

Disturbances of certain special senses (*q. v.*) have been described—viz., Sight, Hearing, Smell, and Taste—leaving for consideration the cutaneous, muscular, articular, and tendinous sensibilities.

The central Body, or Somæsthetic Sense, is made up of certain component sense qualities as follows:

1. **Æsthesic or Tactile Sense** .... Tested with light brush or bristle.
2. **Algesic or Pain Sense** .... Tested with sharp instrument or pin.
3. **Thermæsthesic or Temperature Sense**. Tested with hot and cold bottles.
4. **Pallæsthesic or Vibratory Sense**..... Tested with tuning fork over subcutaneous bony areas.
5. **Bathæsthesic or Deep Sense**......... Tested by different postures of limb.
6. **Myæsthesic or Muscle-Sense** .... Tested with differing degrees of pressure.

general or *common* sensation appears in consciousness to have at in the body—e. g., pain; a *special* sensation is referred mento some external agency—e. g., the pressure of a weight.

ethods of Examining for Sensory Disturbances.—ensory functions are examined in order to determine whether are abnormally active, absent or perverted. In testing the eous and pain senses, the patient's eyes should be kept strictly l or blindfolded, and he should be instructed to respond ptly at the instant he perceives the sensation required, using s the same word, "Yes" or "Now," but otherwise to keep . Note the promptness of the response, as there may be a in conduction. The time elapsing between stimulus and nse is, under normal circumstances, $\frac{1}{10}$ of a second; in disease y require 10 seconds. In testing tactile, temperature, and pain ility, the areas in which disturbances are found should be careoutlined. It is often necessary to go over the whole cutaneous e inch by inch before the examination can be considered to been thorough. In some cases it is necessary to examine not he cutaneous surface, but the mucous membrane of the nasal ral cavities and the external genitalia.

) **Tactile Sense.**—This sense, which embraces the elements of re and contact, may be tested *as a whole* by the æsthesiometer ctically a pair of dividers with blunted points. In default of a tion instrument, ordinary compasses with their tips guarded by f adhesive plaster, a hairpin, or the heads of two ordinary pins, e used. The patient's eyes being closed, the points are to be l upon the skin sufficiently wide apart to be recognised as a e contact and gradually brought together until it appears to the ct that he is being touched with a single point only. Note also er one point is felt as two or more: and whether a touch upon de of a limb, or of the body, is felt respectively upon the other f the same limb, or the opposite side of the body. The sensivaries within wide limits. If the distances are twice as great se given in the following table it may be considered abnormal:

tongue.........	1 mm.	= $\frac{1}{25}$ in.	Back of hands...	30 mm.	=	$1\frac{1}{5}$ in.
fingers.........	2 "	= $\frac{1}{12}$ "	Neck	35 "	=	$1\frac{3}{8}$ "
....	3 "	= $\frac{1}{8}$ "	Forearm, leg, dorsum of foot....	40 "	=	$1\frac{3}{5}$ "
surface of fingers	6 "	= $\frac{1}{4}$ "	Back	60–80 "	=	$2\frac{2}{5}$–$3\frac{1}{5}$ "
nose and forearm	8 "	= $\frac{1}{3}$ "	Arm and thigh ..	80 "	=	$3\frac{1}{5}$ "
toes, cheeks, eyetemple.........	12 "	= $\frac{1}{2}$ "				

he element of *contact* or *touch* is tested by a camel's-hair brush, wisp of cotton touched lightly to the skin, the patient answering immediately upon feeling the touch. To avoid unthinking or mechanical responses by the patient, it is desirable from time to time ostensibly, but not actually, to touch the surface. Symmetrical parts should be compared. The power of *localization*, dependent upon the tactile sense *plus* muscular or weight sense, is determined by touching the skin with the finger tip and asking the patient, with his eyes closed, to put his own finger on the same point. He should not err to a greater extent than 2 inches. Localization on opposite side (allochiria) should be tested.

(*b*) **Temperature Sense.**—For testing this sense one may use test tubes containing hot and cold water. Touch the surface first with one, then with the other, desiring the patient in each case to state whether it feels hot or cold.

(*c*) **Pain and Pressure Sense.**—The point of a needle should be employed to determine the acuteness of the pain sense. It may be absent or excessive. Localization should be tested and opposite sides compared as to intensity.

In testing the cutaneous *pressure sense*, the extremity or part to be examined must lie upon a table, bed, or other support, so as to eliminate muscular action. Weighted rubber balls, weights held by a wire, shot cartridges filled to different levels with shot, 2 pill boxes (1 filled, 1 empty), or, more simply, coins differing in weight but preferably of nearly the same size, may be laid upon the part and the patient requested to indicate which is heavier. This sense is most acute on the brow, temples, forearm, dorsal surface of hand, and abdomen.

(*d*) **Muscular Sense.**—This special sense is to be tested by using weights, as in the examination of the pressure sense, except that the limb or part must not be supported. It is desirable to use objects which resemble each other in size and shape but differ in weight. First a light, then a heavy, object may be placed in the unsupported hand, or upon the dorsum of the foot, and the patient be required to say which is the heavier.

(*e*) **Articular and Tendinous Sense.**—To test this, have the patient's eyes strictly closed or blindfolded. Then taking hold of one of the extremities, flex, extend and move it in a variety of directions, desiring the patient to imitate these motions with the corresponding limb of the opposite side; or place one limb in a certain posture and have him describe the position in which it remains.

(*f*) **Ataxia.**—Upon the *muscular sense*, by which is ascertained the amount of strength to be employed, and the *articular and tendinous sense*, which informs the sensorium of the position of the various limbs and parts of the body, depend the power of *co-ordina-*

tion—i. e., the regular and smooth co-operation of individual muscles or muscle groups which is requisite to accomplish a definite action or movement. If these senses are dulled or abolished the condition of *ataxia* exists. Other factors are also concerned—sight, touch, etc. —but those just mentioned are the most important.

I think it well, furthermore, to add the following comment by Dr. Smith Ely Jelliffe: Ataxia "is a disturbance of co-ordination of muscular movements, and is usually of three types—cortical, spinal, and cerebellar. In *cortical* ataxia the conduction fibres between different centres are usually involved, as in the speech defects of general paresis. In *spinal* ataxia the conducting fibres of deep sensibility are involved. In *cerebellar* ataxia the sensory conduction fibres are involved, especially those of the vestibular apparatus. In ataxias, eye control, disturbance of the sense of equilibrium, and of vestibular control are the most important accompanying factors."

Ataxia manifests itself in certain disturbances of station and gait or other voluntary movements. These disorders of co-ordination may be searched for as follows:

(1) Ataxia of the *upper extremities* is tested by asking the patient, his eyes being closed, to touch first with one index finger, then with the other, the tip of the nose, the lobe of the ear, the centre of the closed eye, or the end of an indicated finger of the opposite hand; or, with the eyes open, to thread a needle, button his coat, or write. If these attempts are successful the co-ordination is good.

(2) Ataxia of the *lower extremities,* if present, may be exploited by various methods. Require the patient to stand with closed eyes, the heels and toes together. In a perfectly healthy person the swaying of the head will be about 1 inch, even less than with the eyes open. If ataxia is present the swaying will be very noticeable, and the patient, if not prevented, may even fall down. This symptom is called *static* ataxia (or the "Romberg symptom"). Ask him further to walk along a straight line (seam of carpet, line of junction of floor boards). If he is unable to follow the line he has *motor* ataxia. If he is unable to walk, desire him, with his eyes open, to imitate with his foot, movements made by the examiner's hand (e. g., writing in the air, circles), or with closed eyes to touch with the heel of one foot the knee of the opposite side (knee-heel test).

In all tests for ataxia made with closed eyes, the patient should first rehearse once or twice the required movement.

In cerebellar and some cortical (frontal) ataxias the patient walks in a reeling, drunken manner, with short steps and feet wide apart—the titubating gait. With closed eyes in walking they may reel first to one side or the other, or fall forward or backward. In the frontal, cortical ataxias, which closely resemble cerebellar ataxias, the symptom known as adiadokokinesis (inability to make certain rapid fine movements of the hands, such as five-finger exercises or rapid wiggling of the toes) is usually absent. In cerebellar ataxias it is more often present.

The Diagnostic Significance of Sensory Disturbances.—The disturbances to be considered are:

(*a*) **Anæsthesia.**—This term, according to a strict definition, indicates a loss of light touch-sensibility, but is often employed to denote a loss of any form of sensation. The distribution of tactile anæsthesia is somewhat varied. It may be either psychical or due to some organic lesion. In general, the most important diseases or conditions which present anæsthesia as a symptom are: cerebral lesions causing hemiplegia; hysteria and traumatic neuroses; disease of the posterior column, roots, or horns of the spinal cord, especially locomotor ataxia; pressure on the cord (tumour, fracture); neuritis (a frequent cause); leprosy and Morvan's disease.

(1) *Hemianæsthesia,* as its name indicates, is a loss of sensibility confined to one lateral half of the body. When found it is in the majority of cases a symptom of hysteria. It usually affects the left side, and terminates very exactly at the middle line of the body. Not infrequently there is anæsthesia of the senses of hearing, taste, smell, and sight on the same side. The next most common cause is a lesion (hemorrhage, tumour) of the posterior third (retrolenticular portion) of the posterior limb of the internal capsule, at which point the sensory fibres pass upward (*G*, Fig. 219). Under such circumstances it is usually incomplete and is conjoined with hemiplegia of the same side. Hemianæsthesia and hemiplegia on the same side *plus* oculo-motor paralysis of the opposite side (crossed ocular paralysis) is indicative of a lesion in the crus on the same side as the ocular paralysis (*C*, Fig. 219). Anæsthesia of one side of the body with anæsthesia of the opposite side of the face (crossed facial anæsthesia) may be due to a lesion of the upper portion of the pons. Very rarely hemianæsthesia is indicative of a lesion in the optic thalamus, or a large cortical or subcortical lesion of the parietal, temporal, or occipital lobes, or is associated with multiple sclerosis or hemichorea. An incomplete hemianæsthesia of one side, with partial hemiplegia of the opposite side, may occur as a result of a unilateral lesion of the spinal cord. Spinal-cord lesions cause irregularly distributed disturbances of light-touch sensibility, especially important in the early diagnosis of spinal-cord tumours.

(2) *Bilateral anæsthesia* sometimes amounting to a double hemi-

anæsthesia, but generally confined to the two lower extremities (Fig. 220) and the lower part of the trunk, is rarely, if ever, due to a lesion of the brain. It is an infrequent symptom in hysteria. Anæsthesia of the lower extremities, if in conjunction with varying

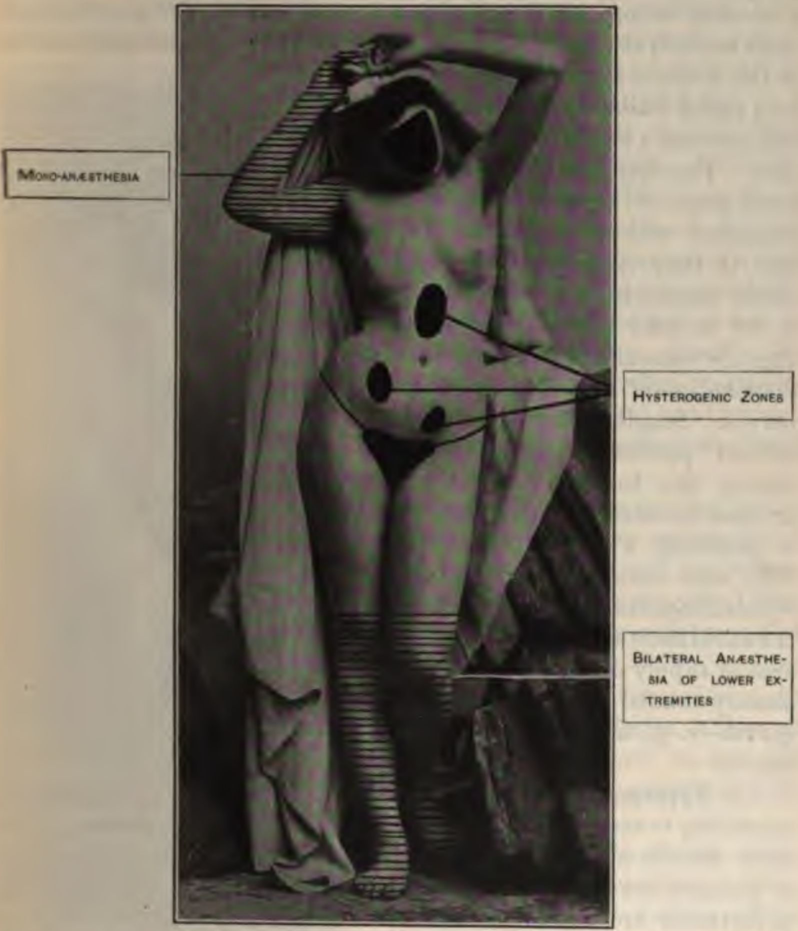


Fig. 220.—Showing (shaded areas) mono-anæsthesia, bilateral anæsthesia, and the usual situation of hysterogenic zones.

degrees of paralysis, is usually due to some lesion affecting the spinal cord, viz., injury or compression of the cord by dislocation, fracture, or caries of the vertebræ; spinal meningitis, hemorrhage into the cord (rare) or meninges (anæsthesia sudden), tumour, myelitis, and locomotor ataxia.

(3) *Anæsthesia of irregular distribution* is due, as a rule, either to hysteria or neuritis. The loss of sensibility may involve one arm (Fig. 220) or one leg, or one side of the face alone (mono-anæsthesia), or occur in multiple and scattered patches in any portion of the body (Fig. 221). Anæsthesia of a single extremity rarely originates from a cerebral lesion, but when arising from this cause the anæsthesia is most marked at the terminal portion of the limb and gradually lessens as the trunk is approached; if caused by a spinal lesion its proximal border will present a sharp line of demarcation. The shape and location of isolated areas of anæsthesia should be compared with Figs. 196 to 198, to find if they correspond to definite spinal segments; and with Figs. 199 to 207 in order to determine whether they are coterminous with the area of distribution of any of the peripheral nerves. Single or multiple circumscribed patches of anæsthesia are usually due to hysteria or neuritis or tabes dorsalis. If to the first, there is generally a lack of correspondence with either segmentary or peripheral localization; if to the second, the anæsthetic area or areas will fit the area of supply of one or more of the sensory cranial or the mixed spinal nerves—e. g., trigeminal, radial, plantar, etc.

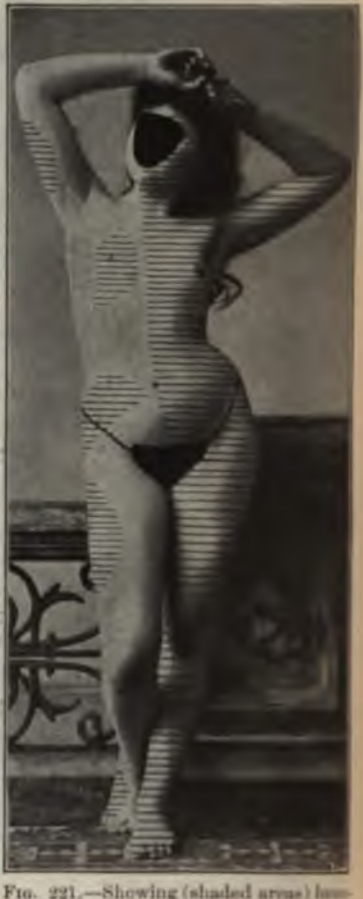


Fig. 221.—Showing (shaded areas) hemianæsthesia, usually left side; and disseminated anæsthesia.

(*b*) **Hyperæsthesia.**—An excessive sensibility to tactile and other impressions attends a considerable number of diseased conditions. Areas of hyperæsthesia are of frequent occurrence in hysteria constituting the so-called hysterogenic zones—i. e., tender points on the chest, the lower abdomen and the back (Fig. 220), pressure upon which will excite and, if continued, may stop a hysterical paroxysm. An excessively tender and painful mammary gland, without evidence of tumour or inflammation, is the "hysterical breast." In neurasthenia there is a frequent hyperæsthesia of localized points along the spine and on the scalp and chest. The scalp is often tender following headaches (especially of migranous type), and facial or occipital neuralgias. The area supplied by any neuralgic nerve may be temporarily hyperæsthetic. Tenderness of the scalp may also occur at the menopause or in gouty persons. A general, sometimes localized, hyperæsthesia may be present in epidemic influenza, typhoid fever, anæmia, and some of the chronic toxic states, especially from alcohol and opium. A suggestive and early symptom of rickets is a diffuse hyperæsthesia. The paralyzed side in a hemiplegia may be slightly oversensitive, and in some cases of brain tumour there is a marked and extensive hyperæsthesia. In unilateral lesions of the spinal cord there is a narrow zone of hyperæsthesia above the level of anæsthesia on the side of the lesion. A decided hyperæsthetic condition of the head and extremities may occur at the onset of cerebro-spinal meningitis, and the finding of hyperæsthetic areas in locomotor ataxia is not uncommon. Inflamed nerves may at some stage of the disease be noticeably hyperæsthetic. Finally, the referred tenderness of visceral disease has been described (page 42).

Anæsthesia dolorosa, areas which are acutely painful but in which there is tactile anæsthesia and analgesia (loss of pain sense), are found in compression of the spinal cord.

(*c*) Disturbances of the **temperature sense** consists in a loss of the cold sense or the heat sense, or both, or a condition of reversal of sensation, cold being called "hot" and *vice versa*. Such alterations from the normal are especially characteristic of syringomyelia (Figs. 193 to 195), and to a less extent of locomotor ataxia and lesions of the medulla.

(*d*) Disturbances of the **pain sense** comprise *hyperalgesia*, an excessive sensibility to painful stimuli; and *analgesia*, a loss of sensibility to pain. Analgesia is found particularly in syringomyelia, Morvan's disease, and hysteria; also in syphilitic cord disease.

(*e*) Loss of the **muscular, articular, and tendinous senses** constitutes ordinary, *versus* cerebellar, ataxia (*q. v.*). It indicates an interruption of the sensory conducting tracts for such impressions (Figs. 191, 193 to 195). It is seen especially in lesions involving the incoming sensory fibres, as in locomotor ataxia and Friedreich's disease. Disease in the ascending tracts, myelitic disease, compression, and occasionally lesions at the higher levels of the medulla and pons may cause it. Diffuse lesions as in multiple sclerosis and paresis may also be responsible for it.

Of these separate senses, loss of weight sense (muscular anæsthesia) may be found alone in hysteria and cortical lesions; impairment of posture sense (articular and tendinous anæsthesia) may be present in tabes and neuritis before ataxia is found; static ataxia involves muscular and articular sensation; and motor ataxia concerns articular and tendinous sensations.

Astasia-abasia—inability to stand and to walk—is generally a symptom of hysteria rather than a separate disease, and most commonly succeeds an emotional storm or an injury. It has been observed also in tabes, multiple sclerosis, spastic paraplegia, multiple neuritis, and exophthalmic goitre. It has been known to follow infectious diseases, carbon monoxide poisoning, overexertion in walking, and painful affections of the leg. There is no paralysis, and while in bed the movements of the feet and legs show no incoordination; but upon attempting to stand or walk the legs give way, as if plastic. Such cases occur mainly in women and young adults.

(*f*) **Other Irregularities of Sensation.**—*Allochiria*, transference of sensation so that a touch on one side of the body is felt on the opposite side, may be a symptom of hysteria, locomotor ataxia, disseminated sclerosis, and myelitis. *Polyæsthesia* (a touch with one point felt as two), is of the same significance. *Delayed conduction* (tactile or pain), so that the response may require 10 seconds instead of $\frac{1}{10}$ second, may be found, especially of pain, in locomotor ataxia and various peripheral paralyses. Of similar moderate suggestiveness are *after-sensation*, an increasing pain lasting for some minutes after a pin prick; and *double sensibility* to touch and pain, in which the tactile impression is first perceived, the painful impression coming to consciousness after a varying time. *Astereognosis*—an inability to recognise the shape or density of solid bodies—usually signifies a gross lesion (tumour, hemorrhage), often of the parietal lobe.

THE REFLEXES

The reflexes to be tested are the *cutaneous* or superficial; the tendinous or *deep* reflexes; and certain organic or *excito-reflex* actions.

All of these reflexes presuppose the travelling of a stimulus from the periphery along the afferent nerve to the motor cells in the cord or medulla. The motor cells transform the received stimulus into an impulse which is reflected to the periphery along an efferent (motor) nerve to certain muscles, which in consequence contract involuntarily. This reflex action ordinarily occupies from one twelfth to one tenth of a second. The superficial or cutaneous reflexes are elicited by irritating the skin or mucous membrane, thus causing contraction of the muscles near the irritated part; the deep or tendon reflexes are produced usually by striking the tendon, but also by sharp percussion of the muscle or the periosteum near the tendon.

The organic or visceral reflexes involve a definite and co-ordinate response to special stimuli—e. g., defecation.

The Superficial (Cutaneous) Reflexes.—(*a*) **Method of Examination.**—The cutaneous reflexes are usually tested by sharply stroking the skin with the finger or a pencil, or by scratching, tickling, pinching, and pricking. The following table gives a list of the superficial reflexes which are of the most clinical importance:

Reflex.	Method of Eliciting.	Response.	Segmental Level.
Conjunctival.	Touch cornea.	Eyelid closes.	Pons.
Pharyngeal.	Touch wall of pharynx.	Pharynx constricted.	Medulla.
Palatal.	Touch soft palate.	Palate elevates.	Medulla.
Scapular.	Stroke interscapular region.	Scapular muscles contract.	CV to DI.
Epigastric.	Stroke downward from nipple.	Epigastrium dimples.	DVII to DIX.
Abdominal.	Stroke down from costal margin.	Abdominal muscles contract.	DXI to LII.
Cremasteric.	Stroke inner side of thigh.	Testicle is pulled up.	LI to LII.
Gluteal.	Stroke skin over buttocks.	Gluteal muscles contract.	LIV to LV.
Plantar.	Stroke sole of foot.	Great toe plantar-flexed.	LIII to SII.
Bulbocavernosus.	Pinch dorsum of glans penis.	Compressor urethræ contracts.	SIII to SIV.
Superficial Anal.	Prick skin of perineum.	External sphincter contracts.	SV and Conus.
Gordon's Reflexus Paradoxus.	Deep pressure over calf.	Dorsiflexion great toe.	LIII to SII.
Oppenheim's Reflex.	Stroke downward along inner side of calf.	Dorsiflexion great toe.	LIII to SII.

(*b*) **Diagnostic Significance of the Superficial Reflexes.**—The cutaneous reflexes are of extreme importance when accurately tested. Some are less constant than others; thus the cremasteric and abdominal reflexes are sometimes normally weak or absent. Both sides should always be tested, and any evidence of lack of uniformity is always suspicious. Thus certain cases of multiple sclerosis may show in the beginning phases loss of the abdominal reflex on one or both sides with few other signs. In tabes careful tests may show a lost abdominal reflex as one of the earlier symptoms. The abdominal reflex is generally absent in all acute abdominal conditions, especially appendicitis and typhoid fever.

Of all the superficial reflexes the plantar reflex is of the greatest diagnostic significance. To determine it properly the patient must be recumbent and the foot comfortably warm, while the limb should be partially flexed at hip and knee and at the same time rotated outward. The sole should then be gently stroked from the heel toward the toe.

In the normal individual in all periods of life after locomotion in the biped position has been acquired *the great toe will be drawn into plantar flexion.* This reflex depends upon continuity between the pyramidal tracts and the spinal reflex arc proper to the plantar muscles. Should the pyramidal tract be interrupted by any cause whatever, the reflex phenomenon is immediately changed, so that instead of plantar flexion the great toe now goes into dorsiflexion. This *dorsiflexion of the great toe is known as Babinski's sign.* In infants who have not yet acquired the skilled act of walking, Babinski's reflex is normally present. This is probably due to the fact that the pyramidal tracts have not at this period become myelinized, and are therefore functionally incompetent. Its occurrence at all other periods of life should be regarded as indicative of some pathological condition. As a rule, Babinski's sign indicates an organic lesion somewhere in the pyramidal tract, either in the encephalon or in the cord. It has also been observed in some functional disorders, especially hysterical hemiplegia.

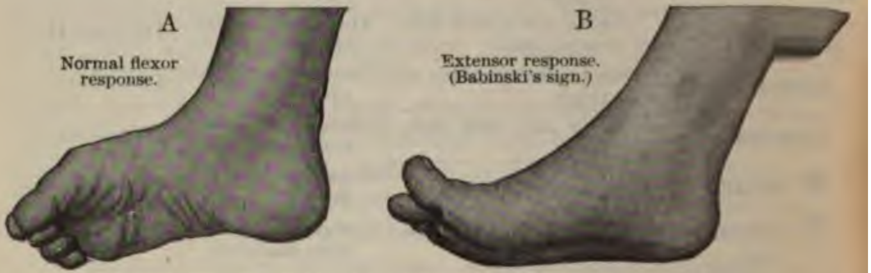


FIG. 222.—The Babinski toe-reflex (Hutchison and Rainy).

Somewhat similar in its general character is the *reflexus paradoxus* (GORDON) which is elicited by pressing deeply upon the calf muscles, when the great toe will be observed to go into dorsiflexion. This reaction is never present in health; it may serve to confirm the Babinski reflex, although it may often be brought out when the latter entirely fails. *Oppenheim's reflex* also pertains to the abnormal dorsiflexion of the great toe. It may be elicited by stroking deeply with some hard object along the postero-internal border of the tibia. Dorsiflexion of the great toe produced either by Babinski's, Gordon's, or Oppenheim's method is generally significant of a lesion in the pyramidal tract somewhere between its origin in the motor cortex and its distribution to the various levels of the spinal cord.

The palate reflex may be lost in hysteria and bulbar paralysis. Diminished plantar reflex with exaggerated patellar tendon reflex is somewhat characteristic of functional paraplegia, and the superficial reflexes in general may be absent on the affected side in the early stages of cerebral hemiplegia.

The presence of a cutaneous reflex is of value because it demonstrates that the reflex arc (sensory nerve, spinal segment, motor nerve) upon which it depends is normal. The location of the segments for the more important of these reflexes is shown in Fig. 223.

The Deep (or Tendon) Reflexes.—The following are the most important of the deep reflexes.

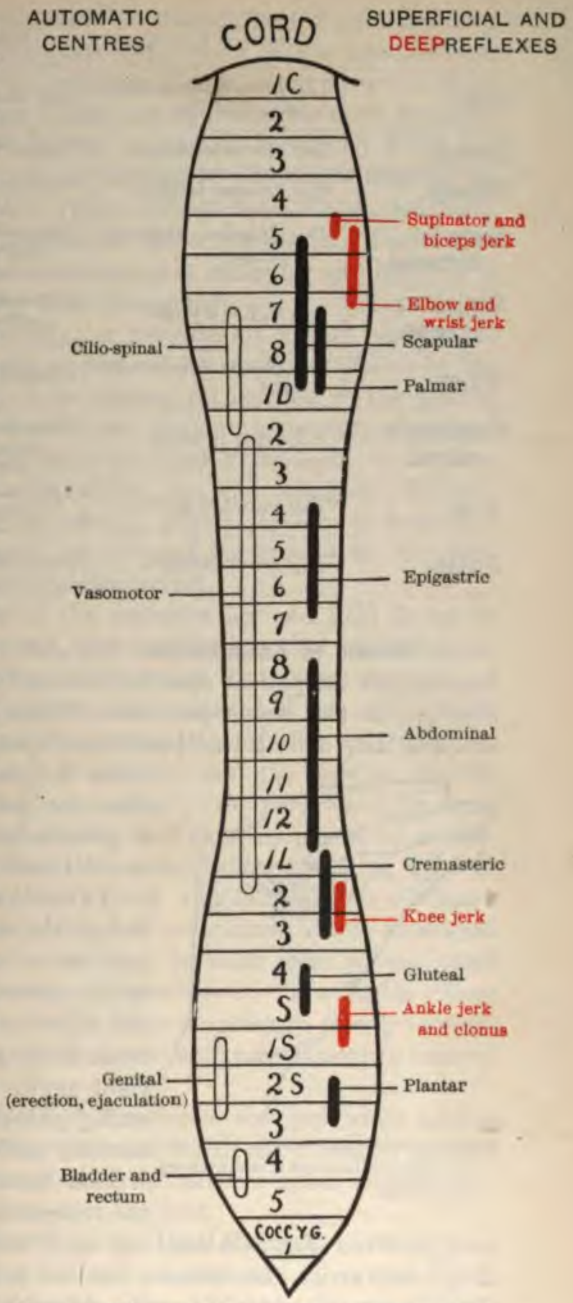


Fig. 223.—Diagram showing the average segmental localization of the automatic centres and the superficial and deep reflexes in the spinal cord.

REFLEX.	METHOD OF ELICITING.	RESPONSE.	SEGMENT.
Jaw.	Tap lower jaw in half-open position.	Jaw closes.	Pons.
Biceps.	Tap biceps tendon.	Biceps contracts.	C5 and C6.
Triceps.	Tap triceps tendon.	Triceps contracts.	C5, 6 and 7.
Scapulo-humeral.	Tap inferior angle of scapula.	Scapular muscles contract.	C5 and C6.
Supinator longus.	Tap radial styloid.	Supinator longus contracts.	C5 and C6.
Wrist.	Tap flexor tendons at wrist.	Fingers are flexed.	C6 to C8.
Carpometa-carpal.	Tap back of wrist.	Fingers are extended.	C6 to D1.
Knee.	Tap patellar tendon.	Leg suddenly extended.	L3 and L4.
Ankle.	Tap tendo Achillis.	Foot plantarflexed.	S1 to S4.

(*a*) **Method of Examination.**—(1) *Jaw Jerk.*—Place a finger upon the chin, let the patient open his mouth, but not too widely, and deliver one or two strong percussion strokes upon the finger; or percuss similarly upon a lead pencil laid upon the lower teeth. If this reflex is present the muscles which close the jaw will suddenly contract. It is dependent upon the motor nucleus of the trigeminus.

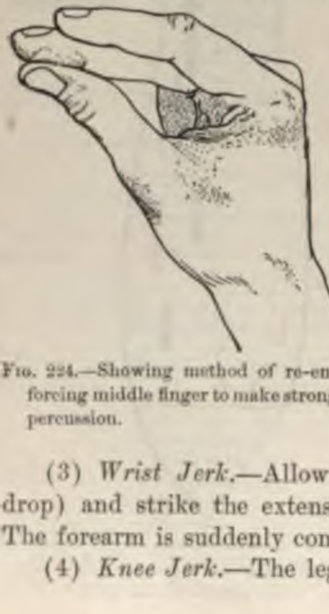


FIG. 224.—Showing method of re-enforcing middle finger to make strong percussion.

(2) *Elbow or Triceps Jerk.*—Bring the arm out from the body and let it rest upon the examiner's hand, which is placed in the crook of the elbow in such a manner that the forearm hangs vertically downward at right angles to the upper arm. Then strike just above the olecranon. The forearm performs a movement of extension because of the contraction of the triceps.

(3) *Wrist Jerk.*—Allow the hand to hang down (as in wrist drop) and strike the extensor tendons proximal to the wrist joint. The forearm is suddenly contracted by the action of the biceps.

(4) *Knee Jerk.*—The leg should be allowed to hang at right an-

es to the thigh. The knees may be crossed; or the examiner may
pport the leg by placing his hand in the crook of the knee; or push
s hand from the outside far enough under to rest it upon the oppo-
te knee of the patient, thus letting the leg swing upon his forearm;
, if the patient can not sit up, the legs should be slightly flexed and
pported. The patient should be directed or diverted so that the leg
ngs loosely. A sharp blow (with edge of hand, finger, hammer)
ould be struck upon the tendon just below the patella. Under nor-
al conditions there follows an abrupt jerk of the leg and foot. If it
n not be obtained, employ "re-enforcement"—i. e., desire the pa-
ent to clinch his fists strongly; or hooking his fingers together, to
ll, one hand against the other, using all his strength. Either by les-
ning cerebral inhibition, or by causing an increase of the general
uscular tension, re-enforcement may elicit an apparently absent re-
x. In some cases of exaggerated knee jerk it is possible to obtain a
nee clonus equivalent to ankle clonus (see (5) following) by fully
tending the leg, seizing the patella, quickly pushing it downward
nd continuing the pressure. As a result the quadriceps may con-
act rhythmically for a considerable period.

(5) *Ankle Jerk.*—Extend the patient's leg and hold it up by
rasping the foot, at the same time bending the foot upward so as
stretch the tendo Achillis. Then strike the tendon sharply and
serve the resulting contraction of the muscles of the calf. *Ankle
onus* is a peculiar rhythmic contraction of the calf muscles.
artly extend the leg, and it is essential that the knee be slightly
nt. Let the heel rest in the examiner's left hand while his right
nd seizes the front part of the foot and abruptly pushes up or dor-
lexes it upon the leg. The calf muscles contract, forcing the foot
wnward against the examiner's hand, and if the latter continues
press steadily on the sole of the foot a rhythmic, clonic movement
the foot will begin and continue. In some cases a very small
ount of flexion and pressure will produce it. True ankle clonus
st be distinguished from the few clonic movements, rapidly ceasing,
ich may be observed as a result of similar manipulations in cases of
rasthenia and hysteria.

"Paradoxical contraction" is sometimes observed while testing
ankle clonus in a leg the muscles of which are extremely spastic.
consists of a tonic contraction of the anterior tibial muscles pro-
ed by the abrupt dorsiflexion of the foot.

(b) Diagnostic Significance of the Deep Reflexes.—Of the deep
exes the knee jerk or patellar reflex is the most important. It is
ost invariably present in health. Fig. 223 shows the spinal local-
ion of the deep reflexes; Fig. 225 their mechanism.

(1) *Absence of the knee jerk* is caused by a lesion affecting a part of the reflex arc. Like the other reflexes, its presence implies

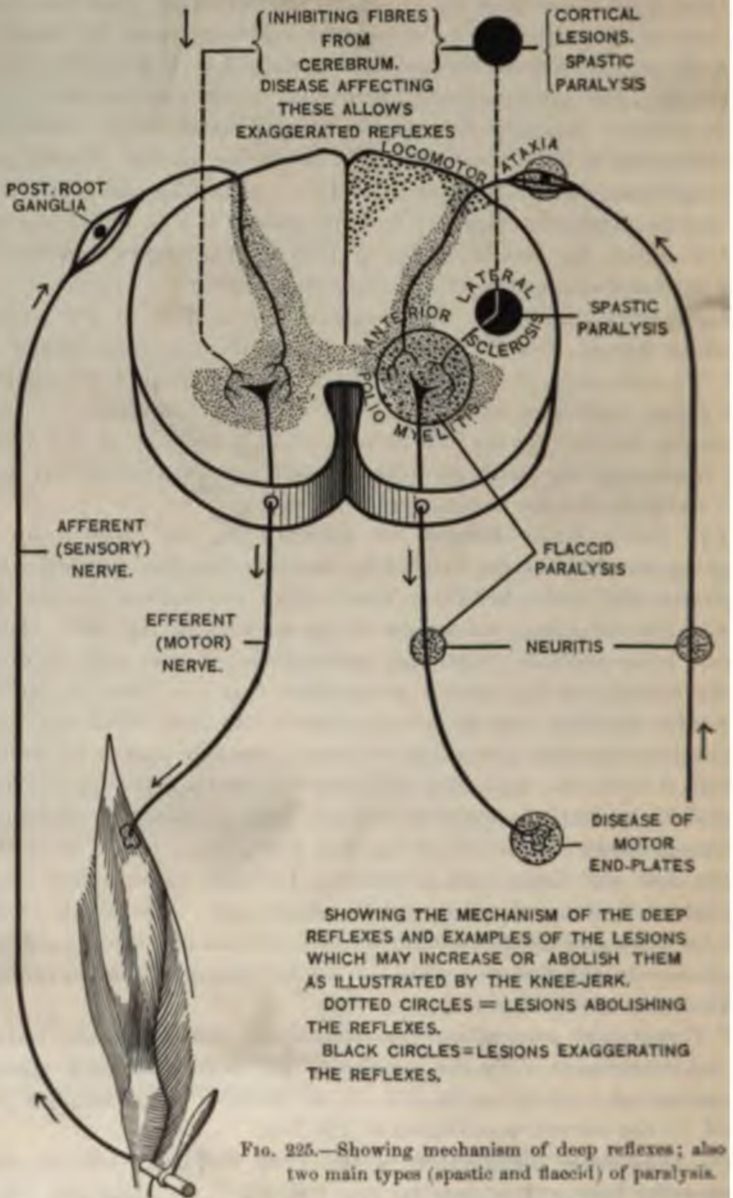

INHIBITING FIBRES FROM CEREBRUM.
DISEASE AFFECTING THESE ALLOWS EXAGGERATED REFLEXES
CORTICAL LESIONS. SPASTIC PARALYSIS
LOCOMOTOR ATAXIA
POST. ROOT GANGLIA
LATERAL SCLEROSIS
ANTERIOR POLIO MYELITIS
SPASTIC PARALYSIS
AFFERENT (SENSORY) NERVE.
EFFERENT (MOTOR) NERVE.
FLACCID PARALYSIS
NEURITIS
DISEASE OF MOTOR END-PLATES
SHOWING THE MECHANISM OF THE DEEP REFLEXES AND EXAMPLES OF THE LESIONS WHICH MAY INCREASE OR ABOLISH THEM AS ILLUSTRATED BY THE KNEE-JERK.
DOTTED CIRCLES = LESIONS ABOLISHING THE REFLEXES.
BLACK CIRCLES = LESIONS EXAGGERATING THE REFLEXES.

Fig. 225.—Showing mechanism of deep reflexes; also two main types (spastic and flaccid) of paralysis.

healthy condition of the afferent (sensory) nerve, the posterior roo the intraspinal paths, and anterior horn of the cord, the afferent (

tor) nerve and the muscle itself. If the function of any portion or element of this circuit is in abeyance the reflex is modified. Consequently, loss of the knee jerk is a symptom of disease affecting either the motor or sensory fibres or both—neuritis; diseases of the posterior roots and columns—locomotor ataxia and Friedreich's disease; diseases of the anterior horns—anterior poliomyelitis (acute or chronic) and Landry's paralysis; and transverse myelitis, if affecting the second and third lumbar segments in which the reflex is localized. It is absent in apoplexy immediately after the shock, in epilepsy immediately after the convulsion, in injuries to the cord immediately after the accident, and in spinal meningitis. It is frequently lacking in the toxæmias of diphtheria, diabetes mellitus (sometimes also insipidus) and chorea.

(2) *Exaggeration of the knee jerk* shows that the reflex arc is intact, but that either the normal restraining influence of the upper (cerebral) motor neurones is destroyed by lesions affecting the cells, or their fibres which run down in the lateral pyramidal columns; or that the irritability of the spinal cord as a reflex centre has been increased. Consequently, taking the intracranial causes first, the patellar reflex is exaggerated in the hemiplegia of apoplexy on the affected side shortly, but not immediately, after the attack; in the cerebral paralysis of children; in hereditary cerebellar ataxia; and in general paralysis of the insane. The lesions affecting the function of the pyramids are multiple, lateral, and amyotrophic lateral, scleroses. Transverse myelitis, injuries to the spinal cord (after the immediate effects of the traumatism have passed), pressure on the spinal cord, and unilateral lesion of the spinal cord, if situated above the level of the second and third lumbar segments, will, by cutting off the inhibiting cerebral impulses, cause exaggeration of the knee and ankle jerk. If the lesion is unilateral the exaggerated reflex will be on the same (paralyzed) side. The patellar reflex may also be exaggerated as a symptom of hysteria, neurasthenia, rheumatoid arthritis, tetanus, and strychnine poisoning.

(3) The ankle jerk is usually present in health; ankle clonus is usually abnormal. *Absence of the ankle jerk* has the same, although less important, significance as absent knee jerk; *exaggerated ankle jerk* and the *presence of the ankle clonus* are of equivalent value to exaggerated knee jerk, the clonus being found especially in lateral amyotrophic and disseminated sclerosis. A brief abortive or false clonus has been mentioned as occurring in neurasthenia and hysteria.

(4) The deep reflexes of the upper extremity are of much less diagnostic value than those of the lower extremities because they are frequently absent in health. The jaw jerk is rarely found under normal circumstances. The presence or exaggeration of jaw, elbow,

or wrist jerk is therefore of more importance than a failure to elic them, and their diagnostic significance, allowing for the difference localization (Fig. 223), is the same as that of the knee jerk.

(*c*) The **excito-motor** or organic reflexes and the significance their altered functions (*q. v.*) have been previously considered, vi Defecation, Urination, Dysphagia, and Respiration.

ELECTRO-DIAGNOSIS (NERVE AND MUSCLE)

An elementary knowledge of the physics of electricity is presu posed.

Apparatus and Technic.—Required are: A *faradic curre* apparatus and a *galvanic current* apparatus. These may run by d cells, wet cells, or the commercial street current. If the latter

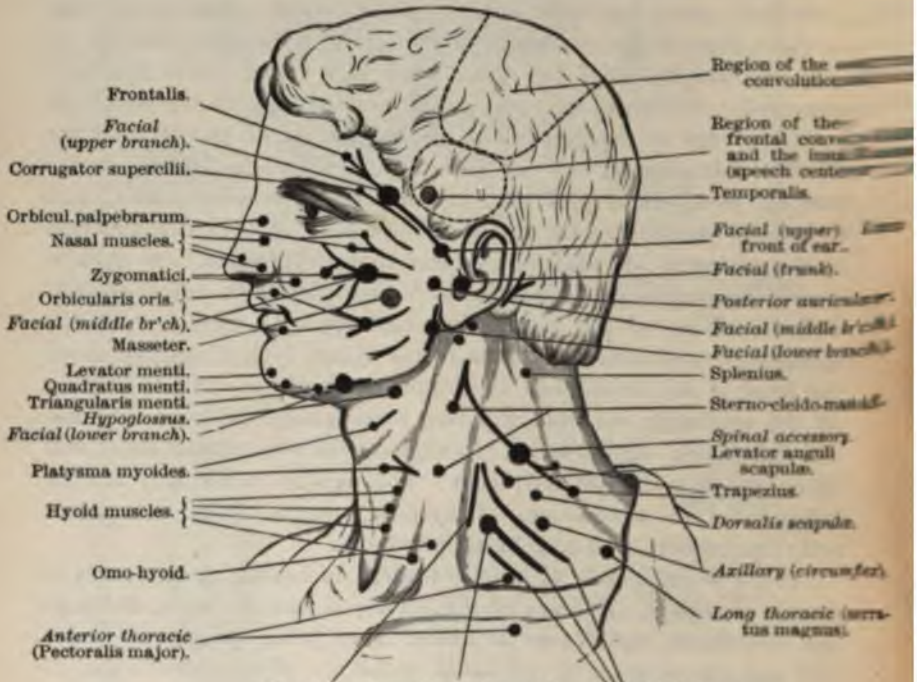


FIG. 226.

employed, guaranteed devices should be used in order to prevent dangerous accidental shocks to the patient. A *rheostat*, for interposing a variable resistance to the galvanic current, so that its strength may be altered at will. A *milliampèremeter*, for measuring the strength of the galvanic current. A *switch*, for changing the direc-

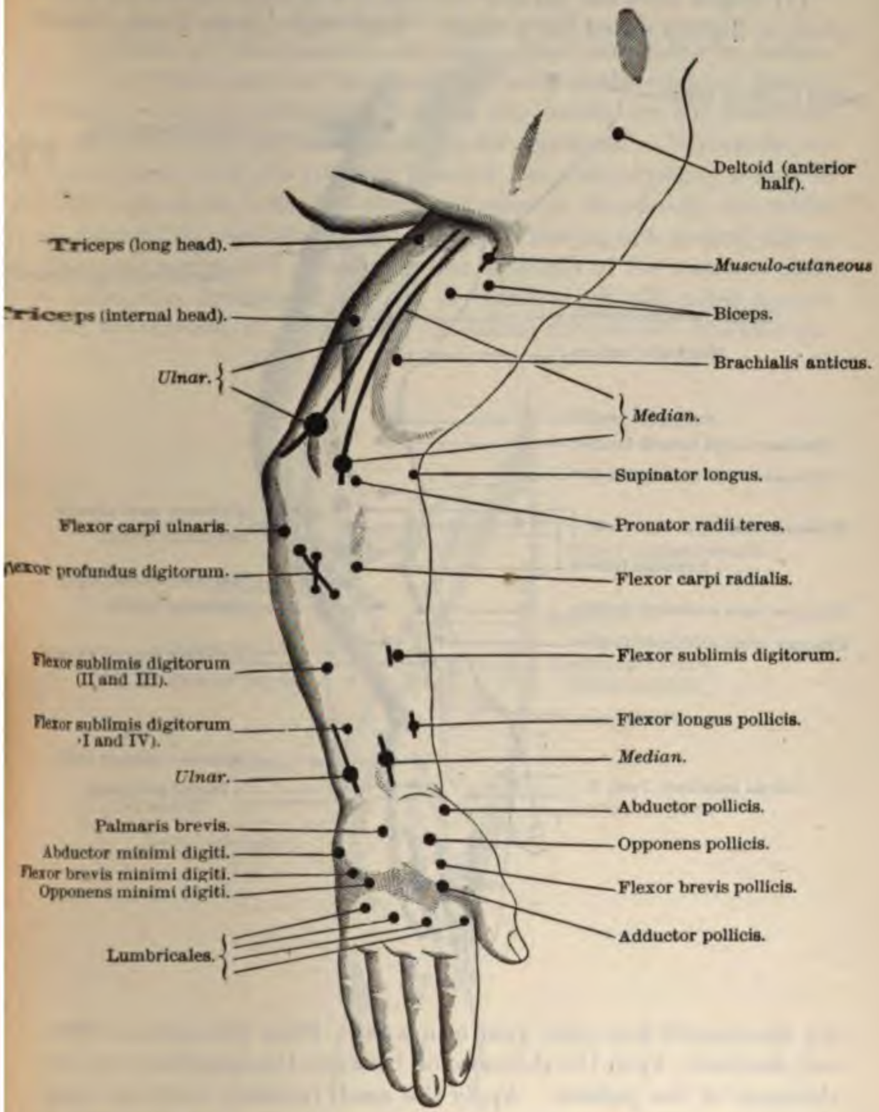


FIG. 227.

tion of the galvanic current—i. e., the polarity of the electrodes—at will. One (*indifferent*) electrode, 2 × 6 inches (5 × 15 centimetres). One (*selective* or *normal*) electrode, 1¼ inches in diameter (about 10 square centimetres). A removable handle for the small electrode, furnished with a button, pressure upon which closes the circuit.

(1) *Begin with the faradic current.*—Wet both electrodes with plain or slightly salted warm water. Start with a weak current, testing its strength first upon your own wrist. Place the large (indifferent) electrode upon the sternum, or between the scapulæ, or on the abdomen of the patient. Apply the small (normal) electrode upon the nerve or the motor points (see 1st paragraph upon page 583) of the muscles to be examined. If the current is not sufficiently strong to cause a visible contraction of the muscle, increase its strength until a contraction is elicited or the current becomes too painful to be borne. Note the minimal strength of current requisite to obtain

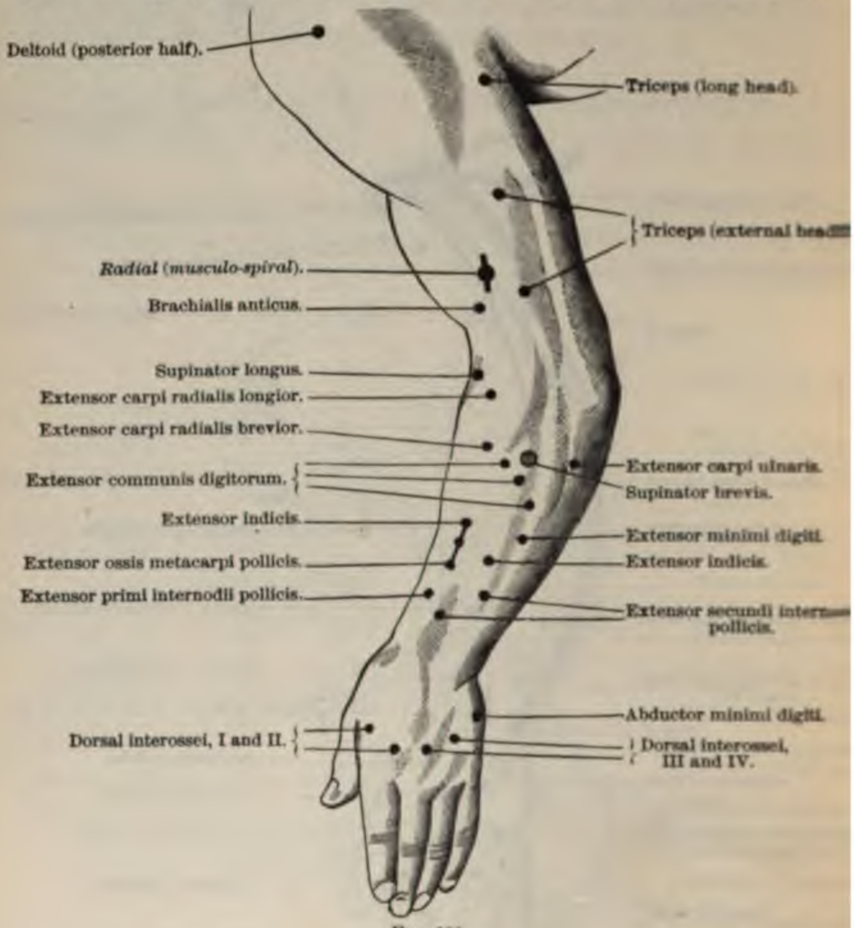

Deltoid (posterior half).
Triceps (long head).
Triceps (external hea
Radial (*musculo-spiral*).
Brachialis anticus.
Supinator longus.
Extensor carpi radialis longior.
Extensor carpi radialis brevior.
Extensor communis digitorum.
Extensor carpi ulnaris.
Supinator brevis.
Extensor indicis.
Extensor minimi digiti.
Extensor ossis metacarpi pollicis.
Extensor indicis.
Extensor primi internodii pollicis.
Extensor secundi inter
pollicis.
Abductor minimi digiti.
Dorsal interossei, I and II.
Dorsal interossei, III and IV.

Fig. 228.

a contraction, as indicated by the scale showing the distance to which the inner and outer coils overlap (CD = coil distance). Go over the nerves and muscles systematically, comparing each muscle with its fellow on the opposite side of the body.

(2) *Then use the galvanic current.*—Place electrodes as before. Turn the switch so that the small electrode is the negative pole or *cathode.* Close the circuit by pressing the button on the handle of the electrode, and let the current run for a moment. It must be understood that with the galvanic current the contraction of a muscle occurs *only at the instant of closing or opening the circuit*, not while the current is passing. Open and close the circuit several times, slowly increasing and decreasing the strength of the current, until the minimum strength of current is found which will cause a muscular contraction at the instant of *closing* the circuit—the cathode closure contraction (CaCC). Note the reading of the milliampèremeter. Change the direction of the current, thus making the small electrode the positive pole or *anode*, repeat the same tests, and ascer-

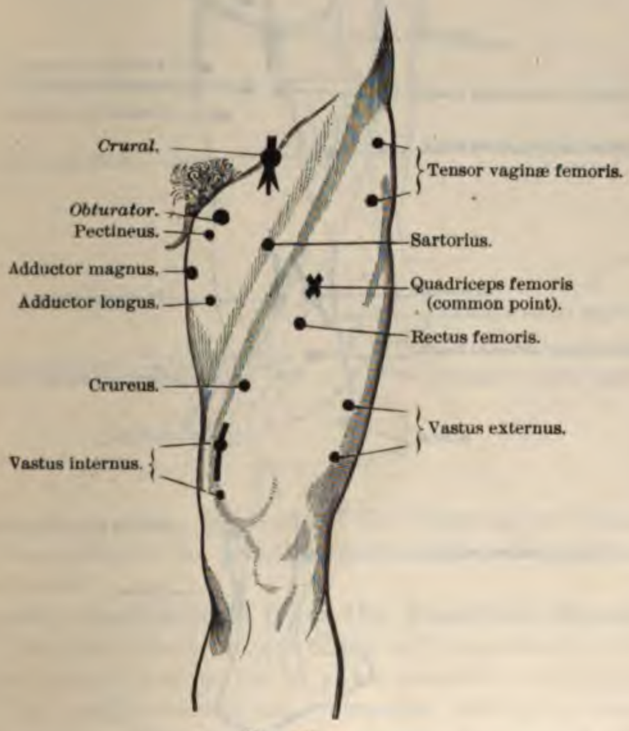


Fig. 229.

tain the same facts with regard to the anode closure contr
(AnCC). Then, with the same strength of current in each ca
termine whether CaCC is equal to, stronger, or weaker, than
Similar tests (rarely required) may be made with the *opening*
cathodal circuit (cathode opening contraction = CaOC), and
anodal circuit (AnOC). The *character* of the muscular contr
is of great importance—i. e., whether it is quick and sharp, sl
and slow, or continuous and tetanic.

It is further to be borne in mind that the testing electrod
be placed directly over the nerve going to the muscle, or ov
body of the muscle itself as far as possible from the nerve.

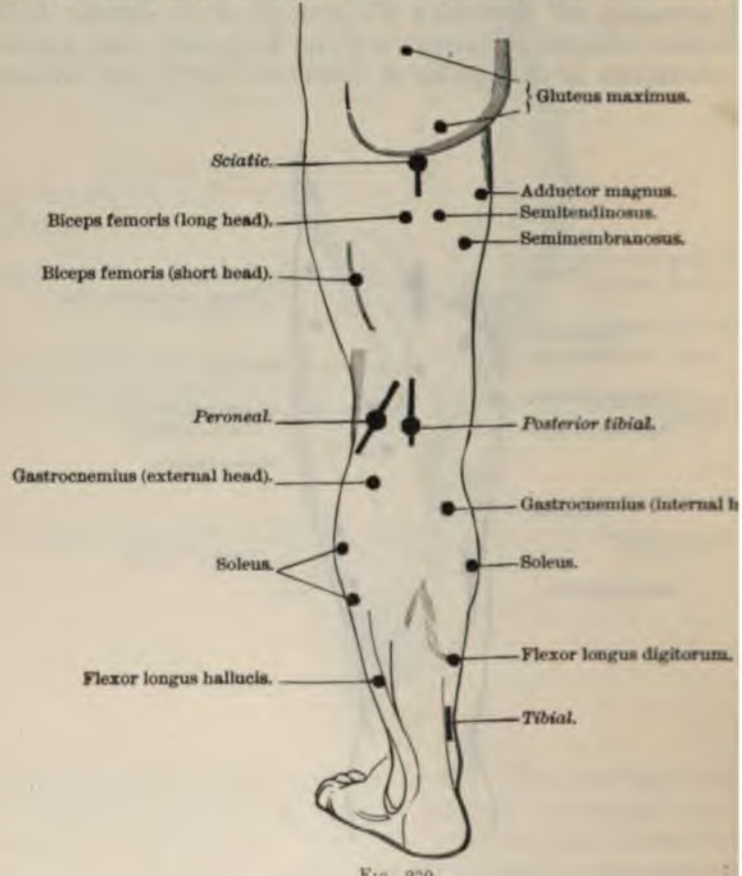


FIG. 230.

matter of fact, it is well-nigh impossible to limit the action
electric current to the muscle alone without also influencin
nerve fibres embedded in it. In either case both the capabil

the nerve as a conductor and the preservation of the contractility of the muscle are demonstrated by the occurrence of visible muscular contraction.

Figs. 226 to 231 show the situation of the accessible nerve trunks, and the motor points—i. e., the points at which the branches of the nerve enter the muscles. By study of the diagrams in connection with the living subject the principal nerves and motor points may readily be learned.

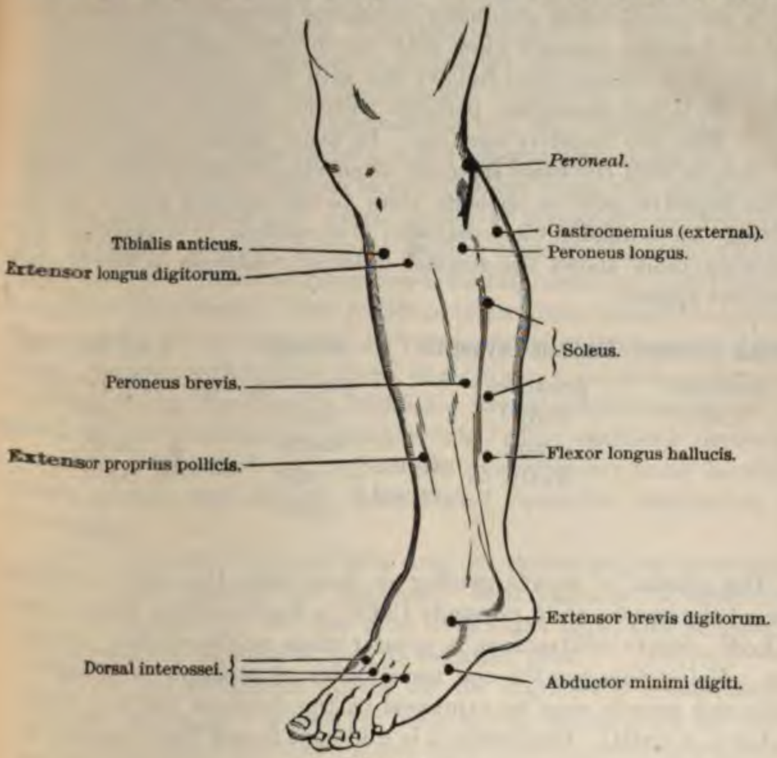


Fig. 231.

Diagnostic Indications from the Electrical Examination.—(1) In **health** the behaviour of the nerve and muscle is as follows: A ready sharp contraction to either electrode (or pole) upon closure of the *faradic current*, the contraction continuing while the current is passing. The stronger the current the stronger is the contraction. With the *galvanic current* it is otherwise. The occurrence of a contraction depends upon the pole which is applied to the nerve, and whether the circuit is closed or open. If a weak current is passed, with the negative pole on the nerve, it will be found that no contraction occurs *while the current is passing.* But if the c rent is just sufficiently strong, a quick, sharp contraction will occ at the instant of *closing* the circuit but not at its opening; and the positive pole is applied, with the same strength of current, th will be no contraction at all, either upon opening or closing. If t test is continued, using gradually increasing strengths of current, t next contraction appears when with the positive pole on the ner the circuit is opened, then follows one with the positive closing, a finally with the strongest, perhaps painful, current, a contracti occurs with the negative opening. In other words, a healthy ner and muscle with the same strength of current responds more readi to the negative pole or cathode than to the positive pole or anod Letting An = anode, Ca = cathode, C = closure, O = opening, th following table shows the normal order of readiness in which co tractions appear:

Weak Current	**Medium Strength**		**Strong**	**Very Strong**
produces CaCC	produces CaCC and AnOC	often reversed	produces CaCC AnOC and AnCC	produces CaCC AnOC AnCC and CaOC

The symbol $<$ means greater or less than, the apex pointing toward the lesser; thus normally CaCC $>$ AnCC, which means that cathode closure contraction is greater than anode closure contraction. Using this symbol, the usual order of reaction of a healthy nerve and muscle may be expressed by the formula CaCC $>$ AnOC $>$ AnCC $>$ CaOC. Practically it is sufficient to test CaCC and AnCC.

(2) In **disease** these reactions may be altered as follows: The response to faradism becomes sluggish, lessens, or disappears. The response to galvanism is changed, so that the contraction is slow, sluggish, and wormlike; the AnCC becomes equal to or even greater than CaCC; and possibly no contraction can be obtained with either faradism or galvanism, no matter how strong the current may be.

The variations from the normal which have just been outlined constitute the reaction of degeneration (R. D.). This reaction depends upon the changes which occur in a motor nerve and muscle which have been cut off from their trophic centre in the spinal cord or medulla, either by disease of the centre or disease of the nerve itself. The terminal nerve fibres, their end plates in the muscle, and finally the muscle itself will degenerate. During this process the electrical reaction progressively alters, affording, according to its degree, "partial" or "complete" R. D. Thus within 3 or 4 days after a spinal nerve has been deprived by injury or disease of the trophic influence of the motor cells of the anterior horn, it becomes less responsive to either galvanic or faradic stimulation, and the faradic response may soon disappear altogether. In 10 days or 2 weeks, however, the response to galvanism may increase beyond the normal. During this time the changed behaviour to the poles and the sluggish contraction of the muscles will make their appearance in varying degrees. Subsequently, if the cause is permanent, in a period varying from several months to two or more years, the galvanic irritability also becomes nil. If the lesion is temporary and removable the paralyzed muscles begin to regain their power, and within a short time the electrical reactions show improvement and gradually return to the normal. The response to faradism, which is composed of a number of excessively short rapid currents, is the first to disappear, because, as the nerve fibres degenerate, it requires a current of relatively long duration to stimulate them—conditions better fulfilled by the continuous galvanic flow. The reactions depending upon the strength of the current are *quantitative*; those involving polar changes and altered character of muscular contraction are *qualitative*.

The *reaction of degeneration* may be epitomized as presenting: With faradic current—no response. With galvanic current—sluggish contraction (by far the most important sign) and as good or better response to positive than to negative pole.

(3) As the peripheral nerves do not degenerate unless they or their cell bodies are diseased (in other words, unless there are lesions of the lower motor neurone), if one finds that the muscles respond to faradism, and that the galvanic reactions are normal, he can exclude diseases of the anterior horns and roots and the peripheral nerves, but can not negative even extensive disease of the higher (central) nervous system. If the reactions of degeneration are found, he can exclude disease of the brain, functional paralysis, and, because of the fact that R. D. occurs very late in the disease, primary affections of the muscles (dystrophies); and can affirm disease of the anterior horns and roots or the peripheral nerves.

VASOMOTOR AND TROPHIC DISTURBANCES

Vasomotor Disturbances.—The vasomotor nerves may be paralyzed or irritated.

Vasomotor paralysis (*angioparalysis*) allows the arterioles to dilate, whereby the capillaries of the part supplied become distended with blood and the circulation is increased. The objective evidence of vasomotor paralysis, therefore, is a more or less marked redness of the cutaneous surface involved, attended not only by a subjective but by an actual increase of temperature.

Vasomotor irritation resulting in a contraction or spasm (*angiospasm*) of the arterioles is evidenced, according to its degree, by pallor, blueness (local asphyxia), or mottling of the skin, with coldness (subjective and actual), formication, or actual pain. If the constriction of the vessels is extreme, gangrene may occur, as in some instances of Raynaud's disease. Angio-ataxia or *vasomotor ataxia* is a condition of unstable tension in which there are more or less rapid variations and irregularities in the tone of the vessels, spasm alternating with paralysis.

As the vasomotor nerves belong for the most part to the sympathetic system, and the latter is simply a division of the peripheral nervous apparatus, receiving its motor fibres from the brain, medulla, and cord, angioparalysis or angiospasm may result from various lesions of the cerebrum, medulla, spinal cord or sympathetic nerves. Angio-ataxia is one symptom of neurasthenia, hysteria, exophthalmic goitre, the menopause, and lesser nervous disturbances.

Trophic Disturbances.—Whether or not there are nerve cells and fibres devoted exclusively to the control of nutritive influences is still an open question. Nevertheless there are numerous trophic disturbances which result from and indicate disease of the nervous system. Some of these involve also vasomotor irregularities. The more important trophic disturbances are the atrophy of muscles from disease of the cranial nuclei or anterior horns of the spinal cord, facial hemiatrophy, myxœdema, cretinism, acromegaly, urticaria, angioneurotic œdema, erythema exudativum, acute bedsores, scleroderma, morphœa, vitiligo, herpes zoster, and glossy (atrophied) skin, as well as changes in the nails and hair. In tabes dorsalis, *mal perforans* and arthropathies occur.

CRANIAL NERVE FUNCTIONS

An important part of the examination of a case of nervous disease relates to disturbances of the functions of the cranial nerves. The condition of each nerve is indicated by the manner in which it performs its functions, as follows. Disturbances of certain cranial nerves have been described elsewhere.

First or Olfactory Nerve.—See Sense of Smell, page 233.

Second or Optic Nerve.—See Vision, page 219.

Third or Oculo-motor Nerve.—See Ocular Paralyses, page 214.

Fourth Nerve or Patheticus.—See Ocular Paralyses, page 216.

Fifth Nerve or Trigeminus.—The *motor portion* of the trigeminus supplies the muscles of mastication, viz., the two pterygoids, the temporal, masseter, mylohyoid, and anterior belly of the digastric.

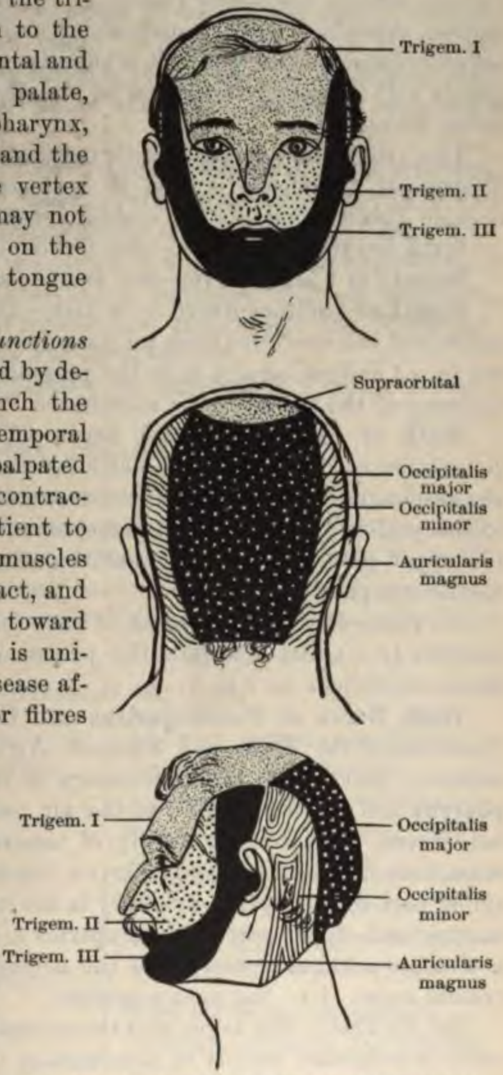


Fig. 232.—Showing sensory supply of skin of face and head. (Hasse.)

The *sensory portion* of the trigeminus affords sensation to the face, conjunctiva, nose, frontal and maxillary sinuses, teeth, palate, tongue, part of upper pharynx, external auditory meatus, and the scalp as far back as the vertex (Fig. 232). It may or may not supply the sense of taste on the anterior two thirds of the tongue (Fig. 58).

To Test.—The *motor functions* of the trigeminus are tested by desiring the patient to clinch the teeth firmly, while the temporal and masseter muscles are palpated in order to observe their contraction. Also desire the patient to open his mouth. If the muscles on one side fail to contract, and the opened jaw deviates toward the paralyzed side, there is unilateral paralysis due to disease affecting at least the motor fibres of the inferior maxillary division of the trigeminus. Spasm of these muscles constitutes trismus or lockjaw (*q. v.*).

The *sensory functions* of the trigeminus are examined in the ordinary manner. Whether one or all of its divisions are involved may be determined by comparing the areas of anæsthesia found with the cuts showing its distribution. If there is complete unilateral anæsthesia the patient when drinking can feel the contact of the cup or glass on one side only, affording a sensation as if half the vessel was missing. As the sense of taste is partly conducted through the medium of the trigeminus, it also should be examined. There are certain inflammatory (herpes zoster), vasomotor (flushing, pallor), and secretory (lachrymation, salivation) disturbances which may arise from disease of this nerve, because of the fact that it supplies the lachrymal and salivary glands with secretory fibres and is also the channel by which vasomotor fibres run.

The trigeminal nerve is involved in neuralgias and migraine. Trigeminal anæsthesia is seen in hysteria and in various forms of organic disease involving the cranial nerve centres.

Sixth Nerve or Abducens.—See Ocular Paralyses, page 214.

Seventh or Facial Nerve.—See Facial Paralysis, page 185.

Eighth or Auditory Nerve.—*To Test.*—Disturbance of the *auditory portion* of the nerve requires an examination of the power of hearing (*q. v.*) and an inquiry as to the presence of tinnitus (*q. v.*). Disturbance of the *space sense* is manifested by vertigo (*q. v.*).

Ninth or Glossopharyngeal Nerve.—The glossopharyngeal (in connection with the vagus) is *motor* for the pharyngeal muscles and the esophagus. Its *sensory* functions are to supply the sense of taste to the posterior third of the tongue, as well as general sensation to the upper part of the larynx, and, together with the vagus, to the tonsils and pharynx.

To Test.—Examine the sense of taste (*q. v.*), the sensibility of the pharynx (*q. v.*), and ascertain the presence of dysphagia (*q. v.*) and globus hystericus (*q. v.*).

Tenth Nerve or Pneumogastric, and the Spinal Accessory.—(1) *Functions of the Tenth and Eleventh Nerves.*—The vagus *plus* the accessory part of the spinal accessory is *motor* for the soft palate, pharynx and larynx, as well as the air passages, heart and abdominal viscera. The *sensory* supply of the vagus has to do with the sensations from the pharynx, larynx, trachea and esophagus. The spinal part of the spinal accessory is the motor nerve of the sternomastoid and upper part of the trapezius muscles. Physiologically it is a motor cervical nerve, while the accessory portion is a part of a cranial nerve—i. e., the pneumogastric.

(2) *To Test.*—The vagus *plus* the accessory portion of the eleventh nerve is examined mainly by determining the condition of its motor and sensory branches. To determine its *motor* condition, ascertain whether the objective and other signs of paralysis of the palate (*q. v.*), pharynx (*q. v.*) and laryngeal muscles (*q. v.*) are present. Inquire also for the existence of laryngeal spasm (laryngismus stridulus, laryngeal crises) or pharyngeal spasm (dysphagia from spasm of the

pharyngeal constrictors). Disorder of the cardiac branches is manifested by altered rhythm and rapidity of the pulse (*q. v.*); of the pulmonary branches by alterations in the respiratory rhythm (*q. v.*); of the esophageal and gastric branches by esophageal spasm, and attacks of vomiting or gastric crises. It is to be borne in mind that the lesion (or irritation) causing the symptoms just mentioned may concern the peripheral portion of the nerve, but more commonly, perhaps, involves its nuclei.

The *sensory* condition of the nerve is determined by ascertaining the presence of anæsthesia (rarely hyperæsthesia) of the larynx (*q. v.*); and the presence of unpleasant cardiac sensations or neuroses.

The spinal portion of the eleventh nerve, being purely motor, is tested by ascertaining the presence of paralysis or spasm (wryneck, *q. v.*) of the sterno-mastoid muscle, and paralysis of the upper portion of the trapezius, raising the shoulder, etc.

Twelfth or Hypoglossal Nerve.—See Paralysis of Tongue.

CEREBRAL LOCALIZATION

A brief statement of the main facts in regard to the localization of functions in the brain is here given. (See Figs. 61, 62, and 63.) As some of the convolutions have two or more synonyms, the indifferent use of which may cause confusion, a brief list of equivalent names is here given for reference. The bracketed terms are synonymous.

CONVOLUTIONS OR GYRI

First frontal convolution.
Superior frontal convolution.
Gyrus frontalis superior.

Second frontal convolution.
Middle frontal convolution.
Gyrus frontalis medius.

Third frontal convolution.
Inferior frontal convolution.
Gyrus frontalis inferior.

Quadrate lobule.
Præcuneus.

Fusiform lobe.
Lateral occipito-temporal lobe.

Lingual lobe.
Median occipito-temporal lobe.

Anterior central convolution.
Precentral convolution.
Ascending frontal convolution.
Gyrus centralis anterior.

Posterior central convolution.
Postcentral convolution.
Ascending parietal convolution.
Gyrus centralis posterior.

First occipital convolution.
Superior occipital convolution.

Second occipital convolution.
Middle occipital convolution.

Third occipital convolution.
Inferior occipital convolution.

Fissures or Sulci

Fissure of Rolando.
Central fissure.

Interparietal fissure.
Parietal fissure.

Second temporal fissure.
Middle temporal fissure.

Postcentral fissure.
Sulcus retro-centralis.

First temporal fissure.
Parallel fissure.

Frontal Lobes.—That portion of the frontal lobes lying anterior to the precentral convolution probably has to do with psychic functions. Disease of this part of the brain may give rise to irritability, mental weakness, frontal ataxia and loss of self-control.

Central Convolutions.—This region is the great motor area of the cortex and the centre for the cutaneous sensations of the portions of the body whose muscles are supplied by it—the *sensori-motor* area. The centre for the leg is highest, then comes that for the arm, and the area for the face lies lowest. The motor centres overlap to some extent, the sensory areas to a still greater extent, thus requiring a much more widespread lesion of this part of the cortex to cause anæsthesia than paralysis of an extremity.

It is to be borne in mind that while many muscles are innervated from the opposite side of the brain, certain groups on opposite sides of the body which habitually act together are each represented in both hemispheres. These are the muscles of respiration, of deglutition, of the vocal cords and of the eyelids. If the centre in one hemisphere for any of these is destroyed the corresponding centre in the opposite side of the brain is competent to carry on the action, and paralysis does not result.

Internal Capsule.—Through the capsule pass the great majority of the motor and sensory fibres connecting the cerebral cortex with the periphery. Its functions are shown in Fig. 190. It will be observed that through the knee and the anterior two thirds of the posterior limb, or thalamo-lenticular portion, pass the motor fibres; while the sensory fibres, including the visual and auditory, traverse the posterior portion of the thalamo-lenticular portion and the posterior third, or retrolenticular portion, of the posterior limb.

Crura.—By way of the crura the fibres from the internal capsule pass down. In the substance of the crus run the 3d and 4th cranial nerves, and the optic tract crosses it.

Pons.—The pons contains the nuclei of certain cranial nerves; the tracts from the cortex by way of the crura pass through it; and its transverse fibres connect the two hemispheres of the cerebellum.

Medulla.—In the medulla are the nuclei of certain cranial nerves and the vasomotor, secretory, visceral, respiratory and cardiac reflex and automatic centres.

Cerebellum.—The cerebellum is so connected with the cerebrum, pons, spinal cord and auditory nerve that it receives and sends impulses which regulate the higher automatic and reflex actions and the maintenance of bodily equilibrium.

Special Senses.—The sense of *hearing* has its primary (basal) centre in the posterior tubercle of the corpora quadrigemina and the internal geniculate body; its secondary (cortical) centre in the first and second temporal convolutions. The sense of *vision* has its primary centre in the posterior portion of the optic thalamus, the external geniculate body, and the anterior corpora quadrigemina; its cortical centre in the cuneus and calcarine fissure. The sense of *smell* has its primary centre in the olfactory lobes; its cortical centre is supposed to be in the uncus and hippocampal convolution. The sense of *taste* has its cortical centre in the hippocampal convolution; the primary centre is unknown.

Memory Centres.—These have been described in connection with aphasia (*q. v.*).

Latent Areas.—There are certain portions of the brain which, if destroyed or irritated by disease, cause very complex motor, sensory, or other symptoms. These are the anterior portion of the frontal lobe, portions of the temporal lobes, part of the inferior parietal lobule, portions of the centrum ovale, the corpora striata, the optic thalami, and the lateral hemispheres of the cerebellum.

SUMMARY OF VARIOUS DIAGNOSTIC POINTS BEARING UPON THE NATURE AND LOCATION OF CEREBRAL AND SPINAL LESIONS

It is convenient and useful, from a diagnostic point of view, to epitomize some of the statements scattered through previous pages with regard to the bearing of various combinations of paralysis, anæsthesia and other symptoms upon the nature and localization of lesions of the brain and spinal cord.

(1) Paralysis, if hemiplegic or at least unilateral, is almost invariably of cerebral origin. Light may be thrown upon the *nature of the cerebral lesion* by ascertaining the symptoms preceding and the mode of onset of the paralysis, which may correspond with one of the following symptom groups: (*a*), (*b*), (*c*), (*d*), and (*e*).

(*a*) Hæmorrhage, Embolism, or Thrombosis.—If due to these causes the paralysis is preceded by apoplectic symptoms, viz., sudden coma or convulsions, with subsequent hemiplegia, accompanied or not by aphasia. Hæmorrhage requires the presence of arteriosclerosis and miliary aneurisms; embolism the presence of left-side heart lesions; thrombosis the presence of arteriosclerosis or obliterating syphilitic endarteritis.

(*b*) Brain Tumour.—The paralysis is preceded by more or less prolonged and progressive general symptoms, viz., headache, vertigo, vomiting, optic neuritis, and mental defects.

(*c*) Syphilis of the Brain.—This affords a history of severe and persistent headache, with vertigo, nausea, vomiting, epileptiform convulsions, and apoplectiform attacks, which are followed by various paralyses (hemiplegia, paralyses of the cranial nerves, especially the ocular). These symptoms are usually due to a gummatous meningitis affecting the base of the brain.

(*d*) Abscess of the Brain.—There is a history of antecedent head injury or suppurative disease of ear, nose, or lung, followed by headache, vomiting, vertigo, delirium or mental dulness, optic neuritis, local tenderness and heat of scalp, septic symptoms with or without irregular fever, and a slow pulse.

(*e*) Hysteria.—Emotional manifestations (laughing, crying, globus hystericus), the presence of anæsthesias, contraction of the visual fields, and other hysterical manifestations. The age and sex may enable a recognition of the functional nature of the paralysis.

(2) Hemiplegia is almost invariably of cerebral, pontine, or medullary origin. Hysterical hemiplegias, however, are frequently encountered.

(3) Evanescent or temporary hemiplegia may be due to embolism or thrombosis and premonitory of a more severe attack; or to uræmia; or occur in the extreme weakness of the final stages of carcinoma and phthisis.

(4) Hemiplegia in young adults should arouse a strong suspicion of syphilis.

(5) Hemiplegia which disappears, leaving only a monoplegia, may be due in rare instances to a lesion of the entire motor cortex of one side.

(6) Ordinary hemiplegia (face, arm, and leg on the same side) is most frequently caused by a lesion of the posterior limb of the internal capsule (*B* and *G*, Fig. 219). If the paralysis is permanent the motor track is destroyed; if temporary, the lesion is slight or the capsule has been functionally affected, perhaps by disease in its neighbourhood. Complete hemiplegia is very rarely of cortical origin, implying as it does a very extensive lesion.

(7) If in a hemiplegia the arm is paralyzed to a much greater extent than the leg, with little or no anæsthesia, the lesion is in the anterior portion of the posterior limb of the internal capsule (*G*, Fig. 219).

(8) If in a hemiplegia the leg has lost its power to a much greater extent than the arm, and with it is found hemianæsthesia, hemiopia, and disturbances of hearing, perhaps also of taste and smell, the lesion is well back in the posterior third of the posterior limb of the internal capsule (*G*, Fig. 219).

(9) Paralysis of the face and leg on the same side, the arm escaping, is a rare combination, due to an irregular linear lesion of the internal capsule, the line of the lesion curving in such a manner as to pass outside of the arm fibres.

(10) Hemiplegia *plus* hemiathetosis, hemichorea (post-hemiplegic), or marked tremor, usually indicates a lesion of the internal capsule involving the optic thalamus (*G*, Fig. 219).

(11) Hemiplegia of one side with paralysis of the oculo-motor nerve on the

opposite side (crossed or superior alternating paralysis), perhaps also with hemiopia, indicates a lesion of the crus cerebri (*C*, Fig. 219).

(12) Hemiplegia of one side with paralysis of the face on the opposite side (alternating or crossed facial paralysis) indicates a lesion of the lower pons (*D*, Fig. 219).

(13) Hemiplegia (right) with motor aphasia indicates a lesion involving the third left frontal convolution.

(14) Hemiplegia with speech disturbances (anarthria, not aphasia) and difficulty in swallowing indicates a lesion of the medulla.

(15) Paralysis of the face and arm or arm and leg (associated monoplegias) may in some cases be due to a lesion of the cortex involving these neighbouring centres. If the paralysis is of the face and arm on the right side, motor aphasia may also be present.

(16) Oculo-motor paralysis of one side with loss of weight sense and posture sense on the opposite side of the body (hemiataxia) is suggestive of a lesion of the tegmentum of the crus on the same side as the ocular paralysis.

(17) If a paralyzed limb exhibits convulsive movements from time to time, or if a convulsed muscle becomes powerless, it may indicate a progressive lesion (tumour, localized meningitis, small hæmorrhage, or abscess) of that part of the cortex from which the limb or muscle receives its motor fibres.

(18) A sense of motion, pain, or tingling preceding a localized spasm of an extremity (Jacksonian epilepsy) is a "signal symptom" indicating the cortical seat of the causative lesion.

(19) Paraplegia of the spastic (cerebral) type is almost invariably due to a spinal lesion involving the motor tracts (*F*, Fig. 219).

Paraplegia of the flaccid (atrophic, peripheral) type may be due to spinal-cord lesions (anterior horns, Fig. 225; and *E*, Fig 219) or lesions of the peripheral nerves (neuritis, Fig. 225). If bladder or rectal disturbances are present the paraplegia is almost always caused by lesions of the cord.

(20) Monoplegia, local and multiple paralysis may be caused by either cerebral or spinal-cord disease, or disease of the peripheral nerves. If the paralysis is due to a cerebral lesion, the latter is almost always cortical (*A*, Fig. 219); if to a spinal-cord lesion it is almost invariably an affection of the anterior horns.

(21) To determine whether a localized paralysis is due to a lesion of the spinal cord or to a lesion of the peripheral nerves :

(*a*) If the paralysis is preceded by a history of exposure to cold or trauma, lead, arsenic, alcohol, etc., it is probably peripheral.

(*b*) If the paralyzed muscles and the associated area of anæsthesia correspond to the area of distribution of one (or more) individual cerebro-spinal nerves, the paralysis is due to disease of the nerve (or nerves).

(*c*) If the paralysis and anæsthesia correspond to the segmentary representation in the cord, the lesion is in the latter.

(22) Lateral deviation of the tongue when protruded, or an inability to move it freely to the same side, indicates paralysis of one hypoglossal nerve.

(23) Anarthria, dysphagia, bilateral atrophy of the tongue, lips, palate and throat muscles indicates a lesion of the medulla (bulbar paralysis).

(24) Anarthria—disturbances of speech involving the muscles of articula-

tion—is to be distinguished from aphasia in which the muscular apparatus is intact but the cerebral centres are affected.

(25) A purely motor aphasia indicates a lesion of the third left frontal convolution.

(26) If the muscles of mastication are paralyzed it indicates a lesion of the motor (inferior maxillary) division of the trigeminus.

(27) Paralysis of the sterno-mastoid and upper portion of the trapezius implies a lesion of the spinal portion of the spinal accessory.

(28) Marked and more or less rapid muscular atrophy occurs in all lesions of the lower neurone (anterior horn cells, cranial nuclei, and their peripheral fibres); it does not occur in disease of the motor tract above the anterior horn cells and cranial nuclei, or in muscular dystrophies and functional paralyses.

(29) Hemianæsthesia of the skin and the special sense organs is due to a lesion well back in the posterior limb of the internal capsule (*G*, Fig. 219) or, more commonly, to hysteria.

(30) Partial (limited to a part of one side of the body) hemianæsthesia with partial hemiplegia on the opposite side is found in unilateral lesions of the spinal cord.

(31) In transverse cord lesions the paralysis and anæsthesia below the lesion are due to the destruction of the columns or tracts which should transmit voluntary impulses and sensory impressions to and from the parts which have been cut off (*F*, Fig. 219).

(32) Disease of the cervical cord will cause sensory disturbances in the fingers, hand, and arm; of the dorsal cord, in the back, trunk, and thighs; of the lumbar cord, in the feet and legs.

(33) In transverse lesions of the spinal cord the anæsthesia begins at a level three or four inches below the site of the external lesion in the cord.

(34) If paralysis is suspected to be of cortical origin the presumption is strengthened by the existence of paræsthesias and vasomotor disturbances in the paralyzed part.

(35) Disturbances of taste in the posterior part of the tongue indicate a lesion of the glossopharyngeal nerve.

(36) In anæsthesia of cerebral origin the reflexes are preserved although the patient may not feel the touch which causes them.

(37) Hemiopia may be due to a lesion of the occipital lobe, the pulvinar of the optic thalamus, the anterior corpora quadrigemina, or optic tract; or, with associated hemianæsthesia, to a lesion of the internal capsule. See (8) and (29).

(38) Word deafness is due to a lesion of the first, and a part of the second, temporal convolution.

(39) The preservation of the knee jerk does not forbid the presence of poliomyelitis, as the lesion may lie at a higher point than the knee-jerk centre.

(40) Ataxia of the cerebellar type, drunken, staggering gait, with little or no ataxia of the extremities while lying in bed, with vertigo, vomiting, headache, optic neuritis, and forced movements, is indicative of a lesion of the vermis or middle lobe of the cerebellum, usually a tumour.

(41) Ataxia of the cerebellar type *plus* ocular paralysis, auditory disturbances and paralysis of the muscles of mastication, points to a lesion of the corpora quadrigemina.

(42) In endeavouring to determine the site of lesions in the brain and spinal cord it is helpful to prepare a written summary of the signs and symptoms, to be compared with the various tables referring to motor and sensory spinal localization.

SECTION XXXVIII

EXAMINATION OF THE BLOOD

A PROPER *clinical* examination of the blood is in all cases useful, and in some indispensable, for diagnosis. A full and *scientific* examination reveals certain facts, as yet of little or no clinical value, and does not fall within the scope of this volume.

THE TECHNIC OF THE CLINICAL EXAMINATION OF THE BLOOD

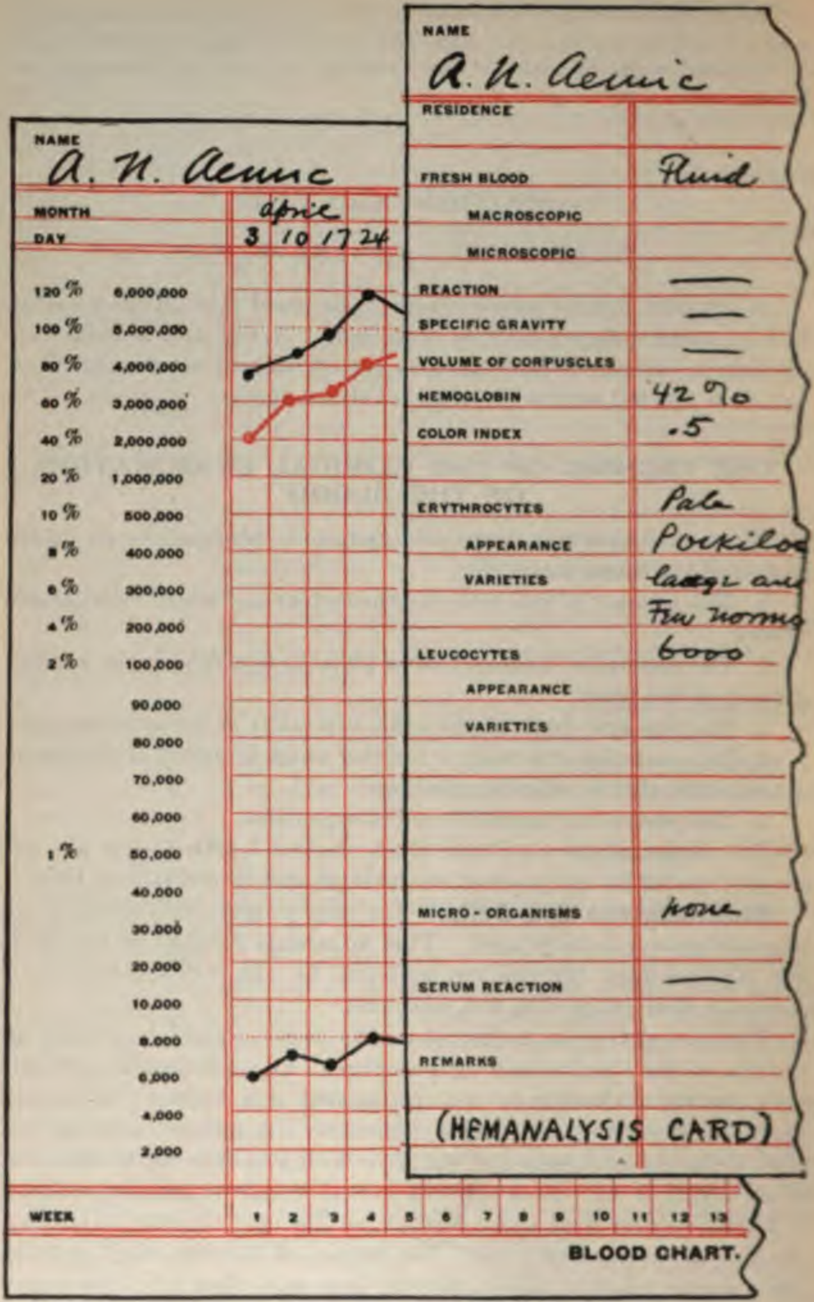
The most important diagnostic facts to be obtained by an examination of the blood relate to:

1. The number of red cells (erythrocytes) and white cells (leucocytes).
2. The amount of hæmoglobin *or* (what is equivalent) the specific gravity of the blood.
3. The size and shape of the cells, especially of the erythrocytes.
4. The varieties and relative number of each variety of the leucocytes as revealed by staining methods.
5. The presence of parasitic micro-organisms.

The hæmanalysis card and chart devised by De Forest are extremely useful for purposes of observation and record (Chart IX).

Counting the Red Cells.—For this purpose the Thoma-Zeiss hæmocytometer is to be used. This apparatus consists of two mixing pipettes (one for the red, and one for the white, cells) and a counting slide (Figs. 234, 235, and 236).

The lobe of the ear, rather than the finger, should be chosen as the site of the blood-obtaining puncture. As an invariable preliminary, ascertain whether or not the patient is a bleeder. If hæmophilia be present a very slight puncture will suffice. Cleanse the lobe of the ear with soap and water, or with a mixture of alcohol and ether, drying it with brisk rubbing so that it may become hyperæmic. A 3-sided (glover's) or spear-pointed needle may be used. Holding the lobe of the ear firmly with the fingers of the left hand, stab its lower border quickly (thus inflicting less pain than by a slow puncture) to the depth of ⅛ to ¼ of an inch. The hæmatospast (Fig. 233)



NAME a. n. aemic

MONTH april

DAY 3 10 17 24

BLOOD CHART.

NAME	a. n. aemic
RESIDENCE	
FRESH BLOOD	Fluid
MACROSCOPIC	
MICROSCOPIC	
REACTION	—
SPECIFIC GRAVITY	—
VOLUME OF CORPUSCLES	—
HEMOGLOBIN	42 %
COLOR INDEX	.5
ERYTHROCYTES	Pale
APPEARANCE	Poikilo
VARIETIES	large an
	Few normo
LEUCOCYTES	6000
APPEARANCE	
VARIETIES	
MICRO-ORGANISMS	none
SERUM REACTION	—
REMARKS	

(HEMANALYSIS CARD)

Chart IX.—Blood chart and hæmanalysis card for recording blood examinations.

is, perhaps, an improvement over the needle. This instrument, devised by V. A. Moore, consists of a nickled brass cylinder, 2½ inches long, in which a spring lancet with a flat spear point is concealed. When drawn up, the knob is caught in a shallow depression, and is released by a touch. The broad thin incision is almost painless, bleeds freely, and heals quickly. It is particularly useful with nervous persons and children. The cap A may be screwed up or down to regulate the size of the cut, and can easily be removed to clean the blade. The deeper depression in the slot locks the lancet securely, so that it may be carried in the pocket. Do not squeeze the lobe to make it bleed (except, perhaps, to start the flow) as the blood will then be diluted by lymph from the surrounding tissues and is worthless for a blood count. When the blood issues spontaneously wipe away the first 3 or 4 drops. Into the next drop insert the tip of the "red" pipette and suck up blood exactly to the 0.5 mark; but if, as may happen, the blood column slightly overpasses this mark it may be carried down by gentle blowing, or by tapping the point of the tube upon a towel or a piece of blotting paper.

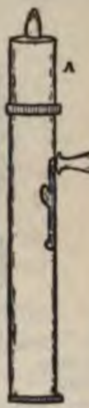


FIG. 233. Moore's hæmatospast.

Now dilute the blood with Toisson's solution. Filter before using, as yeasts may be mistaken for mononuclear leucocytes.

Methyl violet, 5 B	0.025	grm.
Sodium chloride	1	"
Sodium sulphate	8	grms.
Neutral glycerin	30	"
Distilled water	160	"

This stains the leucocytes violet and permits of their being counted in the same preparation with the red cells, which will remain in good condition for several hours.

Wipe the pipette clean of blood, immerse its tip in the bottle of solution and immediately suck up the fluid (meanwhile rolling the pipette between finger and thumb) until the mixing chamber is filled and the fluid reaches the mark 101. The dilution will be 1:200. Next close the ends of the pipette with finger and thumb and shake it actively for one minute in order to thoroughly mix the blood and the diluent, the little glass ball in the mixing chamber greatly assisting. Then blow until the clear diluting fluid contained in the tube below the mixing chamber is expelled and 3 or 4 drops of the mixed blood and fluid have issued. The next drop may be placed upon the counting slide.

In the centre of this slide there is a small ruled glass disk, surrounded by a trench or moat, which in turn is bounded by a wall or slab upon which rests the cover glass (Fig. 236). The relative thickness of the disk and the slab is such that the distance between the upper surface of the disk and the under surface of the cover glass is exactly $\frac{1}{10}$ millimetre. Slide and cover glass must be thoroughly clean and dry. Upon the centre of the disk place a droplet of the diluted blood of a size to be learned by experience. The usual mistake of the beginner is to use too large a quantity. Then place the cover glass at once in position. The drop must contain no air-bubbles, and should almost or quite cover the disk, but if it spills over into the trench the slide is to be washed and a new drop placed. If the cover glass and slide are quite clean and in proper contact one may be able to see (by looking almost horizontally) a series of concentric coloured (Newtonian) rings between the cover and the slab upon which it rests. If not present at first, they may be produced by slight pressure on the cover, but should remain visible after the pressure is remitted.

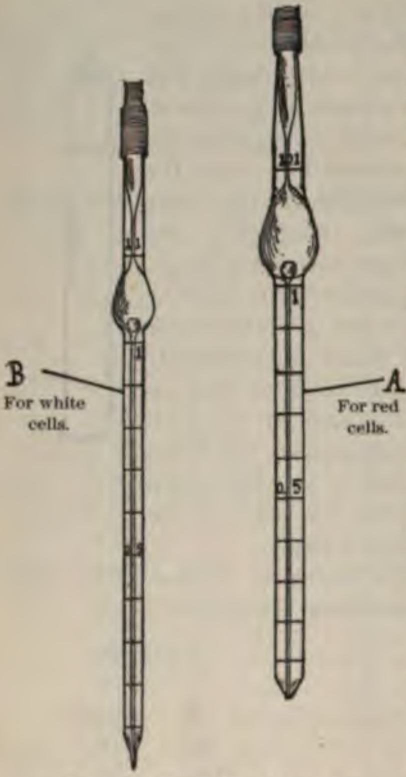


Fig. 234.—Thoma-Zeiss pipettes.

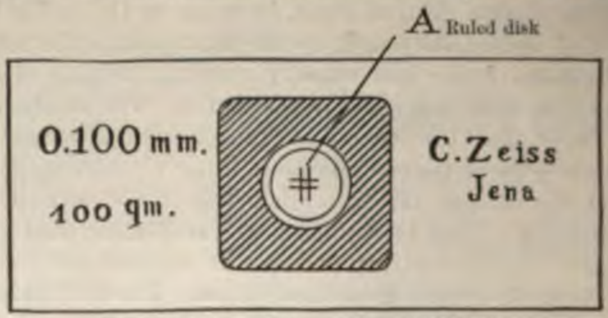


Fig. 235.—Thoma-Zeiss counting slide (plan).

Place the slide upon the stage of the microscope and wait 5 or 10 minutes for the corpuscles to settle. A moderately high power, with a rather dim illumination, is desirable. A mechanical stage, while not a necessity, is of great assistance in all mechanical work, as its uniform movement is less trying to the eyes and therefore tends to greater accuracy.

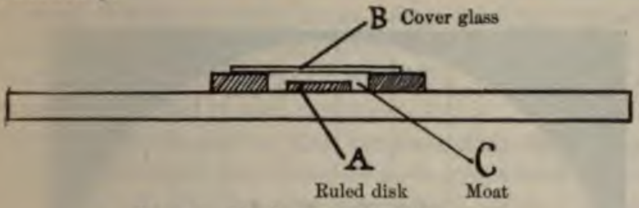


FIG. 236.—Blood counting slide (elevation).

There are 400 small squares, each $\frac{1}{20} \times \frac{1}{20}$ millimetre, which, by means of extra vertical and horizontal lines, are divided into 16 groups, each containing 16 squares (Fig. 240). If the corpuscles lying in each of the 16 small squares of each group are counted, one will have traversed 16 × 16 = 256 small squares. In normal blood diluted in the proportion here employed (1:200) the total number of corpuscles counted will be 1,200 to 1,500, and the chance of error will not be over 2 to 3 per cent in either direction. Some hæmatologists consider a count of 100 small squares to be sufficient for practical purposes, and the average number of cells to each square is readily obtained by placing a decimal point. The squares are conveniently counted in the order shown in Fig. 240. Of the corpuscles which lie upon the line between two small squares, one should count *in* all those which lie upon or touch the *upper and left-hand lines* of each square as indicated in the same figure.

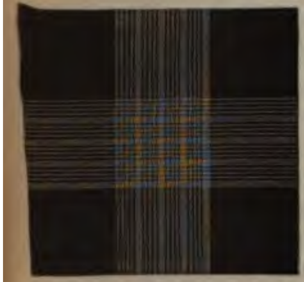


FIG. 237.—Thoma counting chamber.

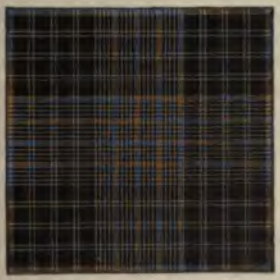


FIG. 238.—Türck counting chamber.

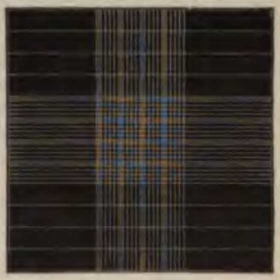


FIG. 239.—Zappert-Ewing counting chamber.

One of the more recent modifications of the Thoma-Zeiss cell may be advantageously employed. The slides are identical in construction, but are ruled somewhat differently (see Figs. 237, 238, 239). Of the three here illustrated, Thoma, Türck, and Zappert-Ewing, the latter may especially be recommended. The ruling of the central square is the same in all, but the eight additional squares provided by the Zappert-Ewing makes possible the counting of the leucocytes simultaneously with the erythrocytes.

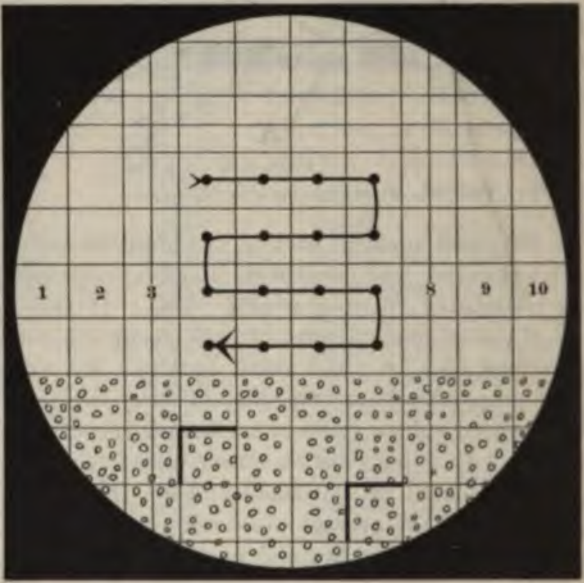


FIG. 240.—Showing one group of sixteen small squares, inclosed by extra vertical and horizontal lines; *also*, by the dotted angulated arrow, a convenient order of counting the squares; *also*, the (shaded) upper and left lines, cells lying *on* which should be counted as *in* the square; *also* (lower part), the average number of cells to each square in normal blood with a dilution of 1:200; and, finally, the method of measuring the size of the field by counting the number (as shown) of squares in its horizontal diameter, each square equalling $^1/_{20}$ millimetre.

Having ascertained the total number of red cells contained in 256 squares, divide the total by 256, which gives the average number of cells to each small square. In normal blood this average is 6 or 7. As the blood is diluted 1:200 and each small square is $\frac{1}{10} \times \frac{1}{20} \times \frac{1}{20}$ mm. = 1-4,000 cu. millimetre, the average number is to be multiplied by 800,000 (= 200 × 4,000), which gives the number of cells in a cubic millimetre of blood. *To summarize:*

Divide the total number of corpuscles by the number of small squares counted.

Multiply the average thus obtained, if, as presumed, the dilution is 1 : 200, by 800,000. If the dilution is 1 : 100, multiply by 400,000.

The pipette must *at once* be cleansed by sucking into and blowing out from the mixing chamber successively water, alcohol, and ether, and finally sucking air through until the glass ball ceases to adhere to any part of the wall of the chamber. If this precaution is delayed or neglected it may be necessary to use a horsehair or bristle to remove coagulated blood; or in bad cases to dissolve it away with strong alkali or acid, or digest it out with a solution of pepsin. The cleansing process is greatly facilitated if the tubing be reversed so that it covers the pointed end of the pipette. Even dense fluids may then rapidly be sucked in and blown out.

If the ruled lines on the disk are extremely faint they may be rendered very distinct by scraping off some graphite from a soft lead pencil, rubbing the powder over the surface of the disk, and then polishing it off with a soft handkerchief.

As a rule it is better (in counting the red cells) to dilute the blood 1 : 200 by drawing the blood to the 0.5 mark, than, by sucking blood to the 1 mark, to use the dilution of 1 : 100.

The "white" pipette may also be used for counting the *red* cells (CABOT) by drawing the blood up to the first line above the tip of the pipette (i. e., ½ of the usual distance) and then sucking the diluting fluid up to mark 11. This gives a dilution of 1 : 100, and the calculation must be made accordingly.

Durham's Hæmometer.—Durham has devised a modification of the older hæmocytometers as follows: (*a*) A number of 5 and 10 millimetre capillary tubes mounted in pipettes (Fig. 241). The rubber nipple is perforated at the side. (*b*) Some small glass test tubes graduated for 1 and 0.5 centimetres for mixing the blood and diluting fluid, and containing glass beads. (*c*) Some 1 and 0.5 centimetre pipettes graduated at 995 and 990 millimetres, and 495 and 490 millimetres respectively, for measuring the diluting fluid. Combined with the appropriate blood tubes these afford dilutions of 1 : 200, 1 : 100, and 1 : 50.

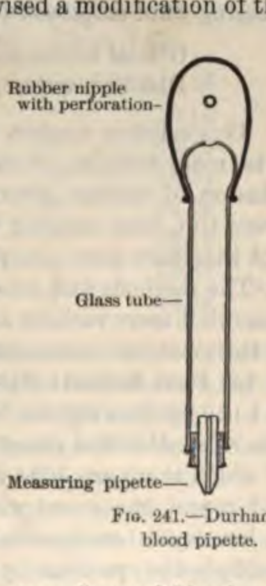


FIG. 241.—Durham's blood pipette.

To Use.—Having determined the desired dilution, the proper amount of diluent is drawn up in the measuring pipette and discharged into the mixing tube. The capillary pipette is then applied horizontally to a drop of blood and fills automatically. All superfluous blood is wiped away and the contents expelled into the mixing tube by covering the hole in the nipple and compressing it. By uncovering the hole the pipette again fills with air without sucking back any of the blood. The tube is then filled by touching it to the diluting fluid and emptied as before, the process being repeated until all blood is rinsed out. Agitation of the mixing tube, aided by the glass beads, insures thorough distribution of the blood through the fluid. A drop is then placed on the Thoma or Zappert stage and counted in the usual way. Simplicity of construction, ease in cleaning, and accuracy, recommend the apparatus.

Counting the White Cells.—If the leucocytes are greatly increased in number, or if special calculations are made, they may be enumerated with the "red" pipette. Ordinarily it is desirable to use the "white" pipette.

The technic of the white pipette is the same as for the "red," except that, as its calibre is greater, a much larger drop (as large as will stay upon the ear without falling off) is required; and the pipette must be kept approximately horizontal or its contents will flow out. Consequently the bottle of diluting solution must be slanted sidewise while the solution is sucked in. Moreover, a much gentler suction is to be employed in order not to overshoot the desired mark. In using the white pipette the degree of dilution is either 1 : 20 or 1 : 10, according as the blood is drawn to the 0.5 or 1 mark. The diluting fluid employed (filter before using) is:

Glacial acetic acid	0.5 to 1 c.c.
Distilled water.....................	100 c.c.

This solution renders the red cells almost invisible and the leucocytes more distinct. Some observers also add sufficient of an aqueous solution of methyl green to make the diluting fluid of a markedly green tint, thus causing the nuclei of the white cells to be stained and therefore more conspicuous; or a drop of Ehrlich's stain.

The methods and calculations involved in the leucocyte count are somewhat more variable and confusing than with the red cells. Three of the most convenient and serviceable are here given:

(*a*) **First Method: Using the "White" Pipette.**—Secure a dilution of 1 : 10 by drawing the blood up to the mark 1. Place a drop upon the ruled disk and count all the leucocytes in *all* the small squares, of which there are 400 in the entire ruled space. Cleanse the slide and repeat the count with a second drop. Divide the total number of leucocytes enumerated by the number of squares counted, and multiply the quotient by 4,000 (since the cubic area of 1 square = $\frac{1}{4000}$ cubic millimetre), and this in turn by 10—i. e., the degree of dilution (1 : 10). For example, suppose the blood to be normal, in case about 70 leucocytes will have been found in each set of mall squares—i. e., the entire ruled space. As two sets of 400 es have been gone over one has

$$\frac{\text{otal leucocytes}}{\text{otal number small squares}} \quad \frac{140}{800} = .175 : \text{and } .175 \times 4{,}000 \times 10 =$$

, the number of leucocytes in 1 cubic millimetre of undiluted

.

) **Second Method: Using the "White" Pipette.**—Another simpler pparently convenient method is as follows (Ostheimer), omitting athematical calculations upon which it is based: Ascertain the er of *small* squares which are contained in the transverse diamf the *field* of the microscope (Fig. 240), if necessary moving the g tube up or down until the right and left edges of the field ide accurately with the outer boundary lines of the two outer es. The number of squares contained in one diameter of the will of course vary with the lenses used and the distance to the sliding tube is drawn out, which points must be noted. e the blood 1 : 20 (by drawing it only to the 0.5 mark), as the s to be given apply only to this degree of dilution. Then count ucocytes in 25 *fields*. These fields may be taken in any part of isk, disregarding the ruled space entirely. Having ascertained otal number of white cells contained in 25 fields, multiply it by f the following factors according to the number of squares cond in the diameter of the field employed. Thus if the

Diameter of the field is	5	squares, multiply by	162.96
" "	6	" "	113.16
" "	7	" "	83.12
" "	8	" "	63.54
" "	9	" "	50.28
" "	10	" "	40.74
" "	11	" "	33.67
" "	12	" "	28.29

or example, if with the required dilution of 1 : 20 a total of 172 cytes has been obtained from 25 fields and the diameter of the is 10 *squares*, the desired result is 172 × 40.74 = 7,007.

) **Third Method: Using the "Red" Pipette.**—The least dilution employed is 1 : 100—i. e., sucking blood up to the mark 1. It essary first to determine the cubic contents of one field of the scope, using the lenses which are to be employed for the count- Ascertain the diameter of the field by means of the number of squares seen, as described in (*b*) preceding. If it has been sary to move the sliding tube of the instrument, a mark should be scratched on it so that it may be set at the same point when required for subsequent observations. As the width (and length) of each small square is $\frac{1}{20}$ millimetre, the exact diameter of the field is known. The depth of the cell is $\frac{1}{10}$ millimetre. If the radius (one half the diameter) of the field is squared and multiplied by $\frac{1}{10}$, and the result is then multiplied by the factor 3.1416, the product will be the cubic contents of the field. For instance, the diameter of the field is 10 squares $= \frac{10}{20}$ millimetre, the radius is therefore $\frac{5}{20}$ millimetre, and $\frac{1}{10} \times (\frac{5}{20})^2 \times 3.1416 = .0196$ cubic millimetre = the cubic contents of the field.

Count the total number of leucocytes contained in twenty-five fields. The fields may be taken from any portion of the disk without reference to the ruled space. If more accuracy is required fifty fields may be counted, using, if necessary, a second drop of blood. Then letting $D =$ the dilution—i. e., 100,

$N =$ the number of leucocytes counted,
$F =$ the number of fields counted,
$C =$ the cubic contents of one field (already known),

the formula $\frac{D \times N}{F \times C} =$ the number of leucocytes in each cubic millimetre of undiluted blood. For example, if 36 leucocytes have been counted in 25 fields with a dilution of 1 : 100, the solved equation will read $\frac{100 \times 36}{25 \times .019} = 7{,}578$. This method is of course a makeshift in the absence of a "white" pipette.

Estimating the Volume of the Cells.—It was anticipated that the hæmatocrit of Hedin as modified by Daland would supplant the more tedious methods of counting the blood corpuscles which have just been described. But while its accuracy in determining the relative *volume* of cells and plasma is conceded, there seems to be some doubt among hæmatologists whether it affords trustworthy information with reference to the *number* of cells. The hæmatocrit is essentially a machine whereby a column of blood contained in a glass tube can be centrifugalized at a speed of 9,000 or 10,000 revolutions per minute for 2 or 3 minutes. At the end of this time the corpuscles are packed solidly toward one end of the tube, the red cells nearest the outer extremity and the leucocytes showing at the free end of the red column as a faint gray line. The tube is marked, each division being supposed to represent a certain number of cells. It is the value of each degree of the scale which is in dispute, varying according to different observers from 99,500 to 123,000. Until this margin of error can be eliminated it is thought best not to include the hæmatocrit as a blood *counter.*

Estimating the Hæmoglobin.—It is quite as important to estimate the percentage of hæmoglobin as to make a count of cells. Several excellent instruments are available. The hæmatometer of Fleischl, or Meischer's modification of it, Gower's, Oliver's, Dare's, and Sahli's instruments are all reliable, while Talquist's paper scale varies from them not more than 5 to 10 degrees. In the absence of apparatus the specific gravity of the blood may be utilized.

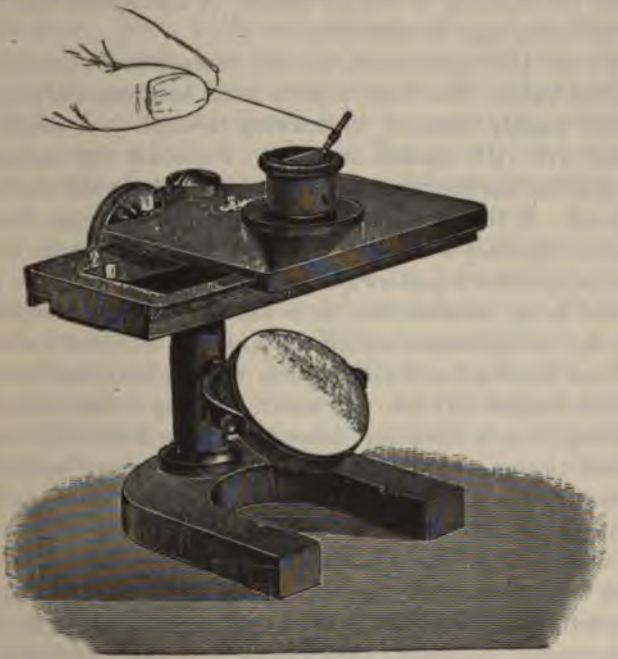


FIG. 242.—Von Fleischl's hæmometer.

(*a*) **V. Fleischl's Hæmometer.**—This apparatus comprises a stage with a central opening, in which is fitted a short glass-bottomed cylinder divided into hemicylindrical compartments by a vertical partition (Fig. 242). A small capillary pipette open at both ends and furnished with a wire handle accompanies the apparatus. Beneath one of the two compartments lies a wedge-shaped strip of coloured glass held in a frame, which is moved by a screw so that any part of the wedge can be brought under the compartment. The colour of the glass changes uniformly from clear white at the thin end to a deep red at the base of the wedge. The sliding frame is furnished with a scale graduated from 0 to 120 which is read through an opening in the stage. Underneath the stage is a white disk to act as a light reflector. *Artificial light is essential*, but the following device renders it unnecessary to use a darkened room. Open a large book at its middle, holding the leaves smooth by a rubber band on each side. Stand it on the examining table and place a small piece of lighted candle well in the angle. Back of this place the hæmoglobinometer, and then close in the triangle with a second book or a piece of cardboard. By covering in the top with a third book, a triangular dark room is obtained, through a small slit at the top of which the reading is readily made. A light-proof box, as described by Da Costa, may be employed.

To use the instrument, fill one compartment one quarter full of distilled water. The little pipette must be thoroughly clean and dry, an end readily obtained by drawing through it a needle carrying a thread wet with alcohol or ether. Puncture the ear, and keeping the pipette horizontal, apply one of its ends to the side of the drop of blood. If the pipette is dry and clean it will immediately fill with blood by capillary attraction. Any blood on the outside of the pipette must be quickly wiped away, and, after glancing at the ends of the pipette to be assured that it is exactly filled with blood, plunge it into the compartment which contains the distilled water. Holding the wire handle, swish the pipette rapidly back and forth until the blood is washed out into the water. If any difficulty is experienced in doing this, a medicine dropper may be filled with water, its tip applied to one end of the pipette and the water squirted through. Then with the handle of the pipette mix the blood and water very thoroughly. No time must be wasted between filling the pipette and emptying it into the water, as undue tardiness may allow the blood to coagulate in the tube. Then with the dropper fill both compartments level to the brim, and turn the cylinder so that the compartment containing the clear water lies over the strip of coloured glass. Adjust the white reflecting disk so that the light is thrown upward through the cylinder, and, placing the eye in the proper position, turn the screw back and forth until the tint of the coloured glass exactly matches the tint of the diluted blood. That figure of the scale seen in the stage opening will be the percentage of hæmoglobin contained in the blood examined.

Certain precautions are to be taken (CABOT). View the cylinder through a proper-sized tube of black paper; use as little light as practicable; sit at one or other side of the instrument so that the light from the two halves of the cylinder may fall on the right and left halves of the retina, as the upper and lower halves of the latter are unequally sensitive to colour; and as the colour sense is readily fatigued, use one eye alternately with the other, taking snap shots rather than a prolonged inspection. The screw should be turned quickly rather than slowly, the turns becoming shorter and shorter, the eye appreciating a sudden change of tint much more readily than a gradual shifting.

(*b*) **Miescher's Modification of Fleischl's Hæmoglobinometer.**—This is the most accurate instrument we now have. It has a graduated pipette in which the blood can be diluted in the proportions of either 1:200, 1:300, or 1:400. The principal divisions on the capillary tube corresponding to these dilutions are marked by complete rings encircling the stem. Short lines, each corresponding to 1:100 part of the entire capillary tube, are placed above and below the principal annular gauge marks. These save time in the adjustment of the blood-column in case the blood fails to reach the principal gauge mark or passes a little beyond it. The point of the pipette is polished so that the lower end of the blood-column can plainly be seen.

The blood is examined in two cells, one having a depth of 15 millimetres, the other of 12 millimetres, and the final reading of the scale gives the number of milligrammes of hæmoglobin in 1,000 cubic centimetres of the diluted blood. The instrument must be in a perfectly dark room and only yellow light used (gas, oil, candle). The stage of the instrument and the eyes of the observer should be protected from direct light by means of a cardboard screen. The pipette is filled with blood to the desired mark, all superfluous blood removed with a cloth, and its point dipped immediately into the diluent, a filtered 1-per-cent solution of sodium carbonate. The diluent is quickly aspirated until it reaches the upper line marked on the stem above the bulb. While filling the pipette it should be gently twisted between the fingers in order to prevent coagulation and to ensure a thorough mixing of the blood with the diluent. The most accurate readings can be taken in the intermediate regions of the glass wedge, so the degree of dilution should be such that the observer is enabled to remain within these limits.

The next step is to fill the largest of the chambers supplied with the instrument. One half of the chamber is filled with distilled water until a convex meniscus protrudes above the edge. The pipette is well shaken, 1 or 2 drops of fluid are blown out, the rubber tube is removed, and the other half of the chamber is filled drop by drop with the dilute blood solution by blowing into the capillary end of the pipette. The surface of this fluid should also form a convex meniscus. Care should be taken to prevent the two fluids from flowing together.

The cover-glass, with the groove downward, should now be carefully slid along the edge of the chamber. Finally, the tubular screen is gently placed on the chamber so as not to move the cover-glass, and it is turned round until the long axis of the opening is accurately at right angles to the partition in the chamber. The latter is now placed upon the circular opening of the stage of the hæmoglobinometer, and its partition is set exactly parallel to the outer edge of the glass wedge.

The observer should sit in a rather high chair, so that the eyes are about 10 inches above the stage of the instrument, and make his readings with both eyes open. The coloured glass wedge is now moved until no difference is noticeable in the intensity of the colour tints of the two surfaces. The adjustments should be made quickly, and the eyes rested after each fifteen seconds of colour-matching. Five or ten readings should be made and the mean—not the average—used. The blood should now be transferred to the smaller chamber and a similar series of readings made. The mean of these readings obtained with the smaller chamber should approximate to $\frac{4}{5}$ of the mean value obtained with the larger chamber, and the readings with the two cells, both calculated for the 15-millimetre cell, should not vary over 2 points. For example, let the following be the mean value of ten readings:

For the larger chamber	64.0
For the smaller chamber	50.0

If the observation had been absolutely correct, the mean value obtained with the smaller chamber should have been 51.2, since,

$$\tfrac{4}{5} \times 64 = 51.2,$$

or, assuming 50.0 to be the correct number, then the former reading should have been 62.5, since,

$$\tfrac{5}{4} \times 50 = 62.5.$$

In this case the mean of 64.0 and 62.5—i. e., 63.25, or, roughly, 63.0—should be taken as the value obtained with the larger chamber. A table goes with each instrument which gives the absolute quantity of hæmoglobin in milligrammes contained in 1,000 cubic centimetres of the blood solution.

Example.—Let 54 be the mean of a series of readings taken with the large chamber. The table gives 430 as the corresponding quantity of hæmoglobin—i. e., 1,000 cubic centimetres of the blood solution contain 430 milligrammes of hæmoglobin. Assuming the degree of dilution to be 1 : 300, then 1,000 cubic centimetres of undiluted blood would contain 300 × 430 milligrammes = 129 grammes of hæmoglobin. The percentage of hæmoglobin in this case would therefore be 12.9 per cent.

In estimating the hæmoglobin by calorimetric methods 100 per cent is considered normal, and corresponds to 14 per cent of hæmoglobin by weight, which is the amount contained in normal blood. Hence, in the last example we should have

$$\frac{12.9}{14} = 92.4 \text{ per cent.}$$

(*c*) **Oliver's Hæmoglobinometer.**—This involves the use of disks of red glass mounted in frames and graded to Fleischl's scale. The blood is mixed in a circular chamber of similar size to the disks, and the reading made by comparison. Intermediate shades are obtained by "riders" placed over the disks.

(*d*) **Gowers' Hæmometer.**—This apparatus consists of two small test tubes of exactly equal size, a small platform for holding them, and a 20-cubic-millimetre capillary pipette (Fig. 243). One tube is capped, and contains a certain amount of coloured fluid as a standard of comparison. The colour of the fluid is that of normal blood when diluted 1 : 100. The other tube is open, and graduated from 0 to 120. The instrument is now made with two colour tubes, one for use with artificial light, the other with sunlight.

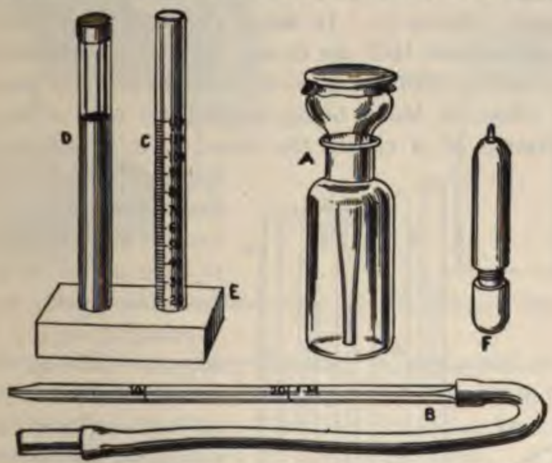


FIG. 243.—Gowers' hæmometer: showing standard test tube, graduated test tube, bottle for distilled water, puncturing lancet, and 20-cubic-millimetre pipette.

To use the instrument, place (with a medicine dropper) 2 or 3 drops of distilled water in the graduated test tube. Then by puncture secure a *large* drop of blood and suck it into the pipette up to the mark. Wipe the tip of the pipette, dip it into the distilled water in the tube, and expel the blood by gentle blowing. Add distilled water drop by drop until the tint of the mixed blood and water is found by frequent comparison to correspond to that of the fluid in the standard tube. When the tint of the blood mixture is the same as that of the standard, read off the height of the fluid in the graduated tube. The mark attained—e. g., 70—indicates that the blood examined contains 70 per cent of the normal 100 per cent of hæmoglobin. The comparison of tints should be made in good daylight, the tubes being placed against a white (paper) background. The standard of this instrument is probably set too high, as many healthy persons will show only 95, or even 90, per cent.

(*e*) **Sahli's Hæmoglobinometer.**—This apparatus, while it resembles Gowers', is based on a different principle. The colour standard is obtained by reducing 20 cmm. of normal blood with HCl and suspending it in glycerine in a sealed tube (Fig. 244 *a*). The rest of the apparatus consists of a graduated tube, *A*; a 20-cmm. pipette, *F*; an ebonite stand, *C*; backed with milky glass, *D*; and a bottle containing decinormal HCl solution, *E*, carried in an ebonite box, *H*, together with a diluting pipette, *G*. In using the apparatus the tube, *A*, is filled with decinormal HCl up to 10; and 20 cubic millimetres of blood are sucked up into the pipette, *F*, and carefully expelled into *A*, the last traces of blood being washed out with a few drops of water. Agitation of *A* causes the blood to mix with the HCl, the hæmoglobin is reduced to methæmoglobin, and a clear brown fluid results. Water is then added drop by drop from the pipette, *G*, the fluid thoroughly mixed by inverting the tube *A*, care being taken to avoid the formation of bubbles, and the colours compared by placing both *A* and *B* in the frame *C*. This is held up to the light—either sunlight or artificial light—and the reading taken. The black sides and milky glass back cut off all but direct transillumination and insure accurate matching, which is further facilitated by the *brown* color, that being an easier comparison tint than red. In order to avoid prejudice do not consult the percentage scale until the colours are the same. It is a further aid to accuracy to cut off with a piece of paper, or the hand, the unfilled portion of the tubes. Should a sediment form in *B*, it will readily disappear on shaking.

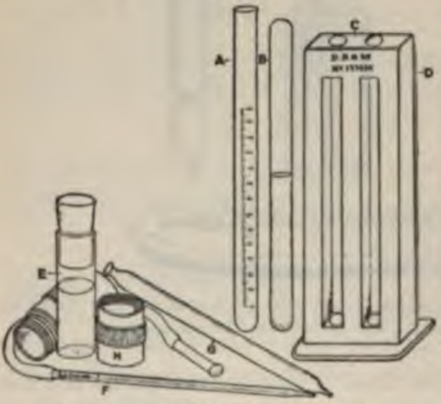


FIG. 244 *a*.—Sahli's hæmoglobinometer.

(*f*) **Dare's Hæmoglobinometer.**—This, like Fleischl's, uses a coloured wedge which is, however, semicircular. As shown in the cut (Fig. 244 *b*), the coloured portion is cemented to a circular disk of white glass, the edge of which is marked in degrees from 10 to 120. This is revolved by the milled screw *R* in the hard rubber box *Z*. A telescope, *U*, is arranged to include in its field both the blood film and the colour scale. The capillary chamber *W*, composed of a plate of clouded and one of colourless glass, so ground as to include a narrow chamber, is placed at the back of the instrument so that both scale and film are equally illuminated by the candle flame. It is used as follows: The front of the box is revolved outward to act as a screen, the telescope is fitted in its place, the blood chamber detached and touched to a drop of blood which immediately, by capillarity, fills the narrow slit, any excess being carefully removed. The chamber is replaced, the candle lighted and turned so that the curve of the wick is toward the instrument to insure equal illumination. On looking through the telescope two red disks are seen side by side, and by turning the milled screw the scale moves to and fro until the colours match. It is advised that the observations be taken in broken peeps, as shades of red are difficult to appreciate when viewed continuously. The percentage of hæmoglobin is then read at a slot in the side of the box. It is not necessary to use a dark room, for in a subdued light

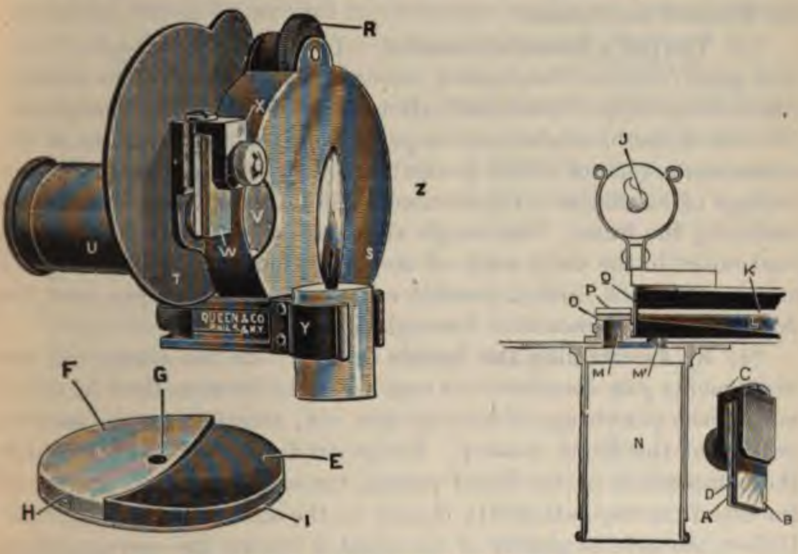


Fig. 244 *b*.—Dare's hæmoglobinometer and cross section of same.

with a black background (e. g., a coat sleeve) the reading is accurate. The plates of the blood chamber are readily separated for cleaning with water and alcohol, and must be kept scrupulously clean. The instrument is to be recommended for its convenience of carriage—it packs in a small case—for the speed with which the readings may be made, and for the fact that undiluted blood is used. The construction of the coloured glass wedge lays it open to the same objections as the Fleischl instrument.

(*g*) **Talqvist's Hæmoglobinometer.**—This consists of a book of bibulous paper, with a lithographed colour scale. A piece of the paper is touched to a drop of blood, and, after the gloss of moisture disappears, the tint of the blood-stain is compared with the various tints of the colour scale, each of which is numbered in accordance with the percentage of hæmoglobin represented by it. Daylight is requisite for matching the tints. This simple and inexpensive device is extremely convenient in the daily work of the practitioner as it offers a quick and ready means, with a possible error of but 5 to 10 per cent, for determining the amount of hæmoglobin.

(*h*) **By Ascertaining the Specific Gravity.**—In the absence of the instruments just described one may use an indirect method of determining the percentage of hæmoglobin, viz., ascertaining the specific gravity of the blood (CABOT). Except in dropsical cases (in which the composition of the blood plasma varies) the specific gravity of the blood corresponds pretty closely to the amount of hæmoglobin. If then the specific gravity of the blood is known the corresponding percentage of hæmoglobin can be inferred.

The specific gravity can be quite readily determined by the following method (ROY-HAMMERSCHLAG). Provide an accurate urinometer (such as most physicians possess), which must be dry and clean, a pipette or medicine dropper, a glass rod, a bottle of chloroform (which is heavier than blood), and a bottle of benzol (which is lighter than blood). Put the urinometer in the footed tube and pour in alternately chloroform and benzol in such quantities that the glass is moderately filled with a mixture of the same specific gravity as normal blood—i. e., 1.059. Remove the urinometer, puncture the ear, take up some blood with the medicine dropper and expel 1 or 2 small drops into the mixture of chloroform and benzol. The blood drop must not contain air bubbles. The blood does not diffuse, but floats in the shape of a small red globule. If it rises to the top, add a few drops of benzol and stir the mixture thoroughly with a glass rod. If it sinks, add chloroform. Continue thus until it neither rises nor falls, but remains stationary, floating about midway between top and bottom. When this is accomplished it is obvious that the specific gravity of the liquid is the same as that of the suspended blood globule. Use then the urinometer to determine the specific gravity of the chloroform-benzol mixture—i. e., the specific gravity of the blood. The mixture may be used indefinitely by filtering out the blood.

Having ascertained the specific gravity of the blood, the percentage of hæmoglobin may be found by consulting either of the following tables, which are subject to correction by further investigations:

Hammerschlag

(By the method described)

Spec. Grav.	Hæmoglobin.
1033–1035 =	25–30 per cent.
1035–1038 =	30–35 “
1038–1040 =	35–40 “
1040–1045 =	40–45 “
1045–1048 =	45–55 “
1048–1050 =	55–65 “
1050–1053 =	65–70 “
1053–1055 =	70–75 “
1055–1057 =	75–85 “
1057–1060 =	85–95 “

Schmalz

(By a direct weighing method)

Spec. Grav.	Hæmoglobin.
1030 =	20 per cent ±
1035 =	30 “ “
1038 =	35 “ “
1041 =	40 “ “
1042.5 =	45 “ “
1045.5 =	50 “ “
1048 =	55 “ “
1049 =	60 “ “
1051 =	65 “ “
1052 =	70 “ “
1053.5 =	75 “ “
1056 =	80 “ “
1057.5 =	90 “ “
1059 =	100 “ “

Rosenberg has devised a float for estimating the specific gravity of blood. It is similar to other hydrometers except that it is provided with an open bulb at its lower extremity into which 1 c. c. of blood can be drawn. The orifice is then closed with a brass cap, the instrument immersed in pure water and the scale read.

Microscopical Examination of the Blood.—In order to ascertain the size, shape, and varieties of the morphological elements of the blood, as well as the presence of parasitic organisms, it is necessary to examine both fresh and dried specimens, the latter either stained or unstained. The microscopical search may be made fairly well with a good $\frac{1}{4}$ objective, but a $\frac{1}{12}$ immersion lens is almost indispensable. All slides and cover glasses used in preparing a specimen of blood for examination must be perfectly clean and dry. The complicated methods of cleaning which have been recommended are quite unnecessary. The application of either ordinary or green soap with the fingers, followed by a thorough washing with water and a subsequent rubbing with a handkerchief fresh from the laundry, will afford perfectly clean and polished slides and covers.

(*a*) **Preparing a Specimen of Fresh Blood.**—Take up a clean cover glass, holding it either by forceps, or (if one touches the edges only, not the surfaces, of the glass) by the fingers. Apply its centre to a fresh globule of blood, without allowing the glass to touch the skin of the ear, and let it fall upon a slide. The drop of blood on the cover should not be larger than a good-sized pinhead. If the slide and cover are dry and clean and have not come into contact with the skin, the blood will at once spread out in a thin layer without the undesirable aid of pressure. A better method consists in painting upon the slide with vaseline a square or ring corresponding to the size and shape of the cover glass, and dropping the latter in such a manner that its edges coincide with the narrow band of vaseline. The thin layer of blood is thus protected from the air, and if kept in a warm place neither crenation nor coagulation will occur for several hours.

The fresh blood can be examined with reference to the shape and size of the red cells, their tendency to form rouleaux, the presence or absence of the *Plasmodium malariæ*, the *Filaria sanguinis hominis*, and the spirillum of relapsing fever. If the examiner has had a large experience he may also form a reasonably accurate opinion as to an increase of fibrin, a decrease of hæmoglobin, or the presence of a marked anæmia or leucocytosis, perhaps of leucæmia.

(*b*) **Preparing Blood Films.**—These may be made either on cover glasses—the method most commonly used—or on slides.

(1) *Cover-glass Films.*—Provide several square cover glasses, perfectly clean and dry. Lay them upon a clean surface in such a manner, by propping up or otherwise, that they can be readily taken hold of with the fingers by their opposite edges or angles, bearing in mind that their surfaces are not to be touched. Apply the centre of one cover glass to the tip of a fresh drop of blood and let it fall upon a second cover glass in such a relation that the angles of the squares do not coincide but lie diagonally to one another, which device renders it practicable to seize them separately by their projecting corners. As soon as the drop of blood has spread between their surfaces, which it should do immediately if the covers are clean, slide the upper cover quickly and steadily off the lower cover, carefully maintaining their parallelism and avoiding any separation by prying or leverage. Dry quickly by waving in the air.

(2) *Slide Films.*—Place upon a slide a drop of blood about half an inch from one end. Take a second slide (with ground and polished edges) and with one of its ends, used like a plane, push the

drop along the slide upon which it lies. A thin film of blood will be left in the rear of the pushing slide, much longer than if made on a cover glass, or select a slide having an even polished end (Ewing has devised a heavy plate glass spreader with rounded, polished ends for this purpose), touch it to the blood drop, being careful to take up only a little, and then *draw* it firmly but lightly the length of another clean slide. This will secure a large, thin, even smear.

(*c*) **Fixing the Blood Films.**—The films require to be fixed—i. e., the albumin coagulated so that the films will adhere firmly. The fixation may be accomplished in various ways, but the two methods about to be described are quite sufficient for clinical work.

(1) *Fixation by a Hardening Solution.*—Immerse the cover films in a mixture of equal parts of ether and absolute alcohol, in which they should remain for half an hour or longer. Even 24 hours' submersion will do no damage. Cover films may be placed in a watch glass, but slide films require to be plunged in a wide-mouth bottle of sufficient depth to entirely cover them. Hardening by alcohol and ether is best adapted for examination of the red cells (e. g., *Plasmodium malariæ*, using Plehn's double stain), although many workers employ it for the differential leucocyte examination as well. Hardening in formalin has also been done with excellent results. The formalin solution is made by diluting 1 part of formalin with 9 times its volume of water and mixing 1 part of the diluted formalin with 9 parts of absolute alcohol. Absolute methyl alcohol is the most satisfactory routine fixative, requiring only 2 minutes.

(2) *Fixation by Heat.*—If a sterilizing oven is at hand it may be heated to 115° C. and the covers or slides placed in it for 15 to 20 minutes. But ordinarily it is much more convenient to heat them on a strip of brass or copper. This plate should be 12 inches long and 3 inches wide. Support it on a tripod or improvised stand, and under its centre place an alcohol lamp or Bunsen burner. In a few minutes the loss of heat balances the gain, and each point of the plate remains at a pretty constant temperature. Then with a medicine dropper place successive drops of water on the plate, beginning at one end and going toward the flame, until a point is reached at which the water just boils. At or a little inside of this spot the cover glasses or slides are to be placed, film side down. Fifteen to 20 minutes is a sufficient time for fixation, but they may be left 2 hours or more without detriment. With Wright's and Jenner's stains previous fixing is unnecessary.

(*d*) **Staining Blood Films and Fresh Blood.**—Among the various stains and their modifications the 4 following are all that are required for most clinical work. Indeed, the first to be described is usually all-sufficient. The stains manufactured by Grübler (Leipsic) alone should be used. The majority of writers on hæmatology advise the use of home-made staining solutions in preference to those prepared by dealers.

The selective action of the stains upon the various corpuscular elements of the blood is dealt with in subsequent pages.

(1) *Erlich's Triple Stain.*—This is used especially for the important differential leucocyte count, and consists of

Saturated aqueous solution of orange G	40 c. c.
" " " " acid fuchsin	45 "
" " " " methyl green	55 "
Distilled water	50 "
Absolute alcohol	50 "
Glycerin	15 "

The saturated solutions should be made by adding a small quantity of the powder each day for a week, until a permanent sediment is formed. After mixing, the stain should be allowed to stand 5 or 6 weeks before using. In the case of this stain it is much more satisfactory to buy Grübler's preparation ready for use.

To use Ehrlich's triple stain, take up a drop of it on the end of a glass rod and spread it over the blood film. It should be left from 1 to 5 minutes, and then washed off with water. The longer the film has been soaked in the ether and alcohol mixture, or heated on the metal strip, the longer should the stain be allowed to remain. Moreover, as at best the stains vary in power, a few trials are requisite to determine the proper length of exposure. The covers (or slides) are then dried between filter paper and mounted in balsam, although some excellent specimens have been mounted dry.

(2) *Plehn's Double Stain.*—This is employed particularly in cases where the *Plasmodium malariæ* is to be sought for, and is composed of

Concentrated aqueous solution of methylene blue	60 c. c.
One half per cent solution of eosin in 75 per cent alcohol	20 "
Distilled water	40 "
Twenty per cent solution of sodium hydrate	12 gtt.

The films, which for this stain are best hardened in alcohol and ether, should be immersed in the solution for 5 or 6 minutes.

(3) *Wright's Stain.*—(Journal of Medical Research, January, 1902.) Place a sufficient amount of aqueous solution of 0.5 per cent sodium bicarbonate in an Ehrlenmeyer flask. Add 1 per cent of

methylene blue (GRÜBLER), (BX, Koch's, or Ehrlich's, Rectified). Be sure all soda is dissolved before adding methylene blue. Steam in Arnold's sterilizer for 1 hour after steam is up, which renders it polychromatic. Remove from sterilizer and allow it to cool. May place flask in cold water. Without filtering, when cold, pour into large dish or flask and add to it, stirring or shaking meanwhile, a sufficient quantity of a 1 to 1,000 solution of eosin (Grübler, yellowish, soluble in water), until the mixture, losing its blue colour, becomes purple, and a scum with yellowish metallic lustre forms on the surface, while on close inspection a finely granular black precipitate appears in suspension. This will require about 500 c. c. of the eosin solution for 100 c. c. of the alkaline methylene blue solution.

The precipitate is collected on a filter and without washing is allowed to dry thereon. When thoroughly dry a saturated solution in pure methylic alcohol is made. Three tenths of a gramme of dry precipitate will thoroughly saturate 100 c. c. of the methyl alcohol in a few minutes. The saturated alcoholic solution of the precipitate is filtered and to the filtrate is then added 25 per cent of methyl alcohol—e. g., 80 c. c. of saturated alcoholic solution requires 20 c. c. of methyl alcohol. This is the staining fluid. It is permanent. Use care to prevent alcohol from evaporating. The dilution is to prevent precipitation on film when staining.

To Use.—Thin films are dried in air. When dry, flood with stain for 1 minute. Next add to staining fluid sufficient water, drop by drop, until it becomes semi-transparent and a reddish tint becomes visible at its margins, while a metallic scum forms on the surface. Let stand 2 to 3 minutes. Wash in water, best distilled water, until the better spread portions of film appear yellowish or reddish in colour. This takes 1 to 3 minutes. Dry quickly between filter paper.

Red cells are orange or pink. Polychromatophilia and punctate basophilia are well brought out. Nucleated red cells have deep blue nuclei, and the cytoplasm is usually a bluish tint.

Lymphocytes: dark purple-blue nuclei, and robin's egg-blue cytoplasm.

Polynuclear neutrophile leucocytes: dark blue nuclei, granules reddish lilac.

Eosinophiles: blue nucleus, eosin granules.

Large mononuclear leucocytes: blue or lilac nucleus, cytoplasm pale blue (one form); or blue with dark lilac or deep purple granules.

Mast cells: dark purple granules.

Megalocytes: blue nucleus and cytoplasm.

Blood plates: deeply stained and prominent.

Malarial parasites: body blue, chromatin lilac through red to almost black. To bring out dark red granules in red cells containing parasites, stain (diluted) for at least 5 minutes and do not wash so long in water.

(4) *Jenner's Stain.*—

a.	Eosin (aqueous)	1.25	grm.
	Distilled water	100	c. c.
b.	Methylene blue (medicinal)	1	grm.
	Distilled water	100	c. c.

Take equal parts of both. Add *b* to *a* (never the opposite) drop by drop with constant stirring. Filter, dry the precipitate in an incubator at 55° C. and powder in a mortar. Wash in distilled water, dry in the air and preserve in tightly stoppered bottles. For use dissolve .5 grm. in 100 c. c. pure methyl alcohol and filter before each staining.

To Use.—Dry blood films in the air. Flood the film for 3 to 5 minutes, wash in water until a pink colour develops, dry in air, mount in balsam if smears are to be preserved.

Erythrocytes stain terra cotta; leucocytes, nucleus pale green; neutrophile granules, pale pink; eosinophile granules, rose; basophile granules, dark blue; mast cells, purple. Malarial and other parasites, blue, chromatin black.

(5) *Staining Fresh Blood.*—For ascertaining the presence of the *Plasmodium malariæ*, especially its hyaline forms, it may be of great advantage to stain the fresh blood as it flows. A drop of a concentrated solution of methylene blue made with six tenths per cent salt solution is placed upon the lobe of the ear, and the puncture made directly through the globule of stain. A very small drop of the mixed blood and solution is mounted in the manner previously described for fresh blood.

Order of Procedure in the Clinical Examination of the Blood.—Place upon a table beside the patient

The "red" pipette and its diluting solution.

The "white" pipette and its diluting solution.

An hæmometer (v. Fleischl's, Sahli's, or Dare's), a small bottle of distilled water, and a medicine dropper. (In the absence of a hæmometer have a urinometer and its glass, a glass rod, a dropper, and a bottle each of chloroform and benzol, to take the specific gravity.)

Two slides on which circles or squares of vaseline have been painted of a size to fit the cover glasses used.

One dozen cover glasses (preferably square); or, if slide films are to be made, a half dozen smooth-edged slides.

A bottle containing absolute methyl alcohol.

A pair of cover-glass forceps.

A small wooden slide-box.

Some gauze or absorbent cotton.

Two ¼-inch-wide rubber bands.

A puncturing needle.

These articles having been conveniently arranged, puncture the ear and:

Prepare two specimens of fresh blood.

Prepare five to ten cover films (or slides), and lay them aside to dry.

Estimate the hæmoglobin *or* find the specific gravity.

Fill the "red" pipette with the proper amount of blood and solution, and put a rubber band over the ends to prevent evaporation. May be kept 24 hours.

Fill the "white" pipette similarly and band it.

Drop two of the films in the ether and alcohol mixture.

Apparatus and specimens may be carried away, and the blood count, fixation of dry films, staining, and microscopical examination be done at the convenience of the examiner. But a search for the *Plasmodium malariæ* may be more successful if made immediately.

II. THE RESULTS AND DIAGNOSTIC SIGNIFICANCE OF THE CLINICAL EXAMINATION OF NORMAL AND ABNORMAL BLOOD

Having described in part the technic of blood examination, it becomes necessary to study the results of the hæmoglobin estimation, the blood count, and the microscopical examination, together with the diagnostic significance of the findings. The items to be considered comprise the red cells (including the hæmoglobin), the white cells, and parasitic micro-organisms.

The following table presents an outline of the varieties of cellular elements occurring in normal and abnormal blood. Their characteristics and the significance of each are considered with more detail in subsequent pages.

CELLULAR ELEMENTS FOUND IN THE BLOOD

Red Corpuscles (Erythrocytes)

Normal	Circular, non-nucleated, average diameter 7.5 μ.
Pathological	*Poikilocytes*, distorted, large and small. *Microcytes*, non-nucleated, small, 3.5 to 6 μ in diameter. *Macrocytes*, non-nucleated, large, 9.5 to 12 μ in diameter. *Megalocytes*, non-nucleated, very large, 12 to 16 μ in diameter. *Normoblasts*, nucleated, 7.5 to 10 μ in diameter. *Megaloblasts*, nucleated, 11 to 20 μ in diameter.

White Corpuscles (Leucocytes)

Normal	*Small lymphocytes.* *Large lymphocytes.* *Large mononuclear leucocytes.* *Transition forms.* *Polymorphonuclear neutrophiles.* *Eosinophiles.* *Mast cells.*
Pathological	*Myelocytes.*

A. THE RED CELLS (ERYTHROCYTES)

Hæmoglobin.—The amount of hæmoglobin at birth is frequently over 100 per cent, and the same condition exists in some grown persons. A percentage of 95, or even 90, in apparently normal individuals is quite common. A notable diminution, however, is significant of one of the various forms of anæmia.

Normally the amount of hæmoglobin corresponds to the number of the red cells. Thus each red cell contains a certain amount or percentage of hæmoglobin, called variously the hæmoglobin value, *valeur globulaire*, or colour index. This value or index can be mathematically stated by dividing the percentage of red cells into the percentage of hæmoglobin. If the average number of red cells to the cubic millimetre, which is assumed to be 5,000,000, is taken as 100 per cent, and the hæmoglobin per cent is also 100, then the colour index is $\frac{100}{100} = 1$. So long as the hæmoglobin increases or diminishes *pari passu* with the number of red cells the hæmoglobin value of the cells remains 1—e. g., 70 per cent of cells and 70 per cent of hæmoglobin $= \frac{70}{70} = 1$. If the hæmoglobin is destroyed to a greater extent than the red cells the *valeur globulaire* is less than 1 —e. g., with 40 per cent of hæmoglobin and 80 per cent of cells, it is $\frac{40}{80} = \frac{1}{2}$ (or 0.5) of the normal. On the other hand, if the cells have disappeared more rapidly than the hæmoglobin, the hæmoglobin value is greater than 1—e. g., with 50 per cent of cells and 60 per cent of hæmoglobin it is $\frac{60}{50} = \frac{6}{5}$, or 1.2 more than normal. This formulary is of importance. In the majority of the anæmias the hæmoglobin is reduced to a greater extent than the cells. In pernicious anæmia, however, there may be a large loss of cells with a smaller loss of hæmoglobin, and the consequent relative increase in the richness of the individual cell is a valuable element in the differential diagnosis.

Number of Red Cells.—The normal number of red cells in adult men is 5,000,000, in women 4,500,000, to the cubic millimetre.

An *increase*, up to 6,500,000, in the number of erythrocytes (*polycythæmia*) is sometimes observed. It occurs in diseases attended by the loss of a large amount of fluid from the body—i. e., cholera, dysentery, and severe diarrhœa; in general cyanosis (see also Vaquez's disease); in diabetes; and in persons living in high altitudes. In unusually vigorous young men the red cells may number 6,000,000.

A *decrease* in the number of red cells (*oligocythæmia*) is of frequent occurrence. Oligocythæmia when found is always indicative of one of the various forms of anæmia, temporary or permanent. The lowest counts are found in pernicious anæmia, of which the oft-quoted case of Quincke's (143,000 to the cubic millimetre when death was impending) is a striking illustration. The average count in ordinary anæmic individuals runs from 3,000,000 to 4,000,000. In the differential diagnosis between gastric carcinoma and chronic gastritis the presence of a marked oligocythæmia speaks for the former.

When the red cells are decreased in number their tendency to rouleau formation diminishes or disappears, and they may be obviously fewer in number in the field of the microscope.

Shape of the Red Cells.—The normal red cell is not absolutely symmetrical, but in disease extremely irregular shapes (Plate III) may be encountered. They may be ovoid, rodlike, or kidney-shaped; or one pole of the cell may be drawn out and lengthened, making it resemble a long-necked flask. At the same time they may be smaller or larger than the normal. The cells are called *poikilocytes*, the condition *poikilocytosis*.

Poikilocytosis when found is indicative of a high grade of anæmia. It is not pathognomonic of any special variety of the disease, but is most marked and extensive in the pernicious form; next to this in the severe chlorotic cases which exhibit thrombotic and embolic phenomena.

Size of the Red Cells.—The average diameter of the normal red cell is 7.5 μ (μ = micromillimetre = micron = $\frac{1}{25000}$ inch), but may vary from 6 μ to 9 μ without being considered pathological. In disease both abnormally small and abnormally large cells (Plate III) may be found. The small cells or *microcytes* measure from 3.5 μ to 6 μ; the large cells embrace two sizes, *macrocytes*, which are from 9.5 μ to 12 μ in diameter, and *megalocytes*, the diameter of which runs from 12 μ to 16 μ. It is to be borne in mind that abnormally sized red cells are apt to exhibit the variations in shape (poikilocytosis) which have been previously described.

Microcytes and macrocytes are seen in the various anæmias. The severer the case the more abundant they are. The presence of a large number of megalocytes or giant red cells is significant of pernicious anæmia. Exceptionally they may be found in chlorosis. Loss of hæmoglobin causes the appearance of irregular clear patches in the erythrocytes which may simulate malarial organisms.

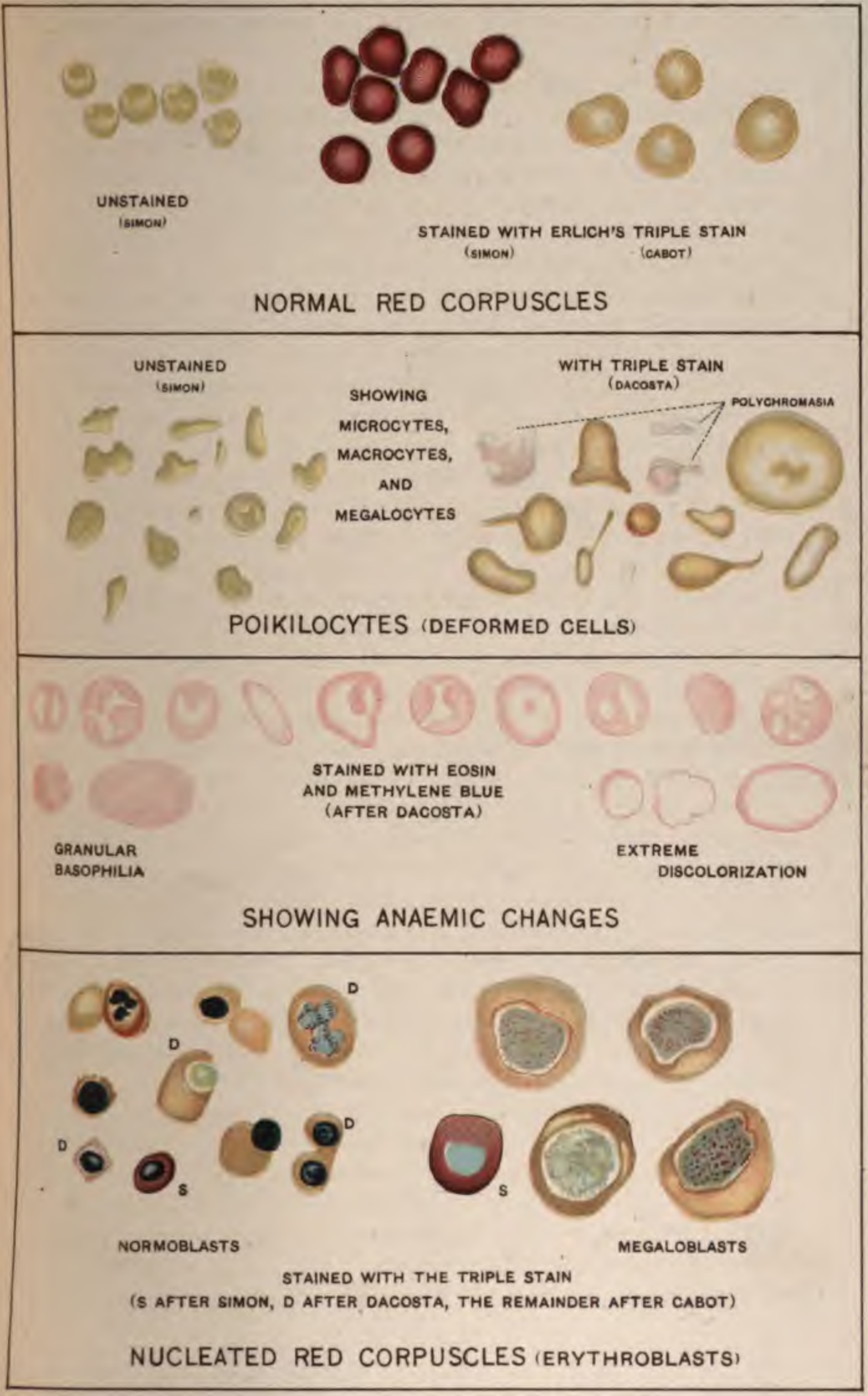
Nucleated Red Cells.—Nucleated red cells may be discovered in stained films. In fresh and unstained specimens it is rarely possible to distinguish them. Two varieties are recognised (Plate III):

(1) **Normoblasts.**—These resemble the ordinary normal red cells in size, form, and character, except in possessing a nucleus, and, indeed, are considered to be simply young and immature red corpuscles. The nucleus is coloured a deep blue or bluish black by Ehrlich's triple stain, sometimes presenting one or more light spots; less commonly its central portion may be bluish gray surrounded by a narrow rim of dark blue. The nucleus as a whole is clearly outlined against the yellow or orange coloured cell body. The nucleus usually lies somewhat to one side, or even partly in and partly out, of the cell; or it may be in two portions, partly or entirely separated.

Normoblasts are normally found in the blood of the embryo, and of infants during the first two or three days of birth; in the blood-making organs—i. e., the bone marrow of children and adults (where they may become very abundant after a hemorrhage), and in the spleen. In these organs they are formed, and, after extruding their nuclei, enter the general circulation as ordinary erythrocytes. But if they are found in the blood subsequent to the second day after birth their presence may be considered indicative of one of the anæmias, particularly if the blood condition is secondary and due to cancer.

(2) **Megaloblasts.**—The megaloblast (or gigantoblast) is an extraordinarily large nucleated red cell, 11 μ to 20 μ in diameter, and of an oval or slightly irregular outline. Its nucleus is also large in pro-

PLATE III.



RED CORPUSCLES, NORMAL AND ABNORMAL

portion to the cell body, and with the triple stain of Ehrlich takes an even, pale green or greenish-blue colour. Not infrequently the cell body or protoplasm, having undergone certain degenerative changes, stains brown, or even a purplish colour, instead of yellow or orange. There is usually a narrow white border, apparently a clear space, lying between the nucleus and the surrounding protoplasm. In view of the existence of atypical varieties of nucleated red cells differing in colour, shape, and relative proportion of nucleus to cell body to an extent which may cause great uncertainty in classification, Cabot has proposed to restrict the term normoblast to nucleated red cells, not exceeding 10 μ in diameter, with a nucleus not over half the diameter of the cell. This nucleus may or may not show signs of division, but must stain deeply. All other nucleated red cells exceeding 10 μ in diameter are megaloblasts.

Normally the megaloblast is found only in the fœtal bone marrow. When discovered in the blood in considerable numbers they constitute an important symptom of pernicious anæmia and leucæmia. In small numbers they may occur in the milder forms of anæmia, primary or secondary. They are generally associated with normoblasts.

Polychromasia.—This condition, characterized by a tendency of the red cells to stain a lighter or darker blue, is regarded by Ehrlich as an evidence of degeneration. On the other hand, some authorities regard such cells as immature and consequently significant of regeneration. They are frequent in chronic anæmias. A similar condition in which the staining occurs scattered through the cell body in specks is known as *Punctate Basophilia*, the "Granular Degeneration of Red Cells" of Grawitz. Schumann calls attention to this in the anæmias due to the intestinal parasite *bothriocephalus latus*, and Behrendt regards it as characteristic of chronic lead poisoning.

B. THE LEUCOCYTES

The diagnostic evidence derived from a study of the white cells or leucocytes relates not only to their total number, but also to the relative proportion of the different varieties of these cells found in normal and diseased conditions.

The Characteristics by which Leucocytes are Differentiated.—The differentiation, as well as the nomenclature, of the recognised varieties of the leucocytes depends upon the number or shape of their nuclei; the character of the protoplasm or substance of the cell body, whether homogeneous, finely granular, or coarsely granular; the behaviour of the protoplasm to certain aniline dyes or stains; and, finally, upon their presumed relative age and birthplace.

In many respects the most important distinction between the various forms of leucocytes is the manner in which different "granulations" exhibit a selective or chemical affinity for certain stains. It should be borne in mind that the granulations are contained in the cell body or protoplasm of the leucocyte, not in its nucleus; although they may surround the latter so completely as to hide it from view in unstained specimens. Some leucocytes have a homogeneous non-granular protoplasm, while in others the granulations are so fine that except with the highest powers their appearance is simply that of a diffuse cloudiness which may entirely obscure the nucleus. In still others the granulations are relatively large, coarse, refractile, and readily seen.

Certain of these granules will take up only acid dyes, others only basic dyes, while some will absorb and hold both acid and basic stains. An "acid" stain is one the acid element of which possesses colouring power; in a "basic" stain the base of the compound is the active dyeing agency. Examples of acid stains are eosin, acid fuchsin, and orange G; of basic stains, methylene blue, methyl green. Granulations which by preference take up acid stains are spoken of as oxyphilic, acidophilic, or eosinophilic; those which select basic stains as basophilic; and those which are stained both by acid and basic dyes as neutrophilic (or amphophilic). The latter are in reality variously oxyphilic.

The Classification of Leucocytes.—Concerning the staining solutions recommended as being sufficient for the clinical examination of the blood the following facts are to be remembered:

Ehrlich's triple stain colours the red cells *yellow* or *orange*—by orange G (acid). Nuclei of red or white cells *blue*—by methyl green (basic). Large (oxyphile, eosinophile) granulations *red*—by acid fuchsin (acid). Small (neutrophile) granulations *lilac* or *violet*—by the combined action of acid fuchsin and methyl green (blue + red).

Plehn's double stain colours the red cells *red*—by eosin (acid). Nuclei of red or white cells *blue* or *violet*—by methylene blue (basic). Large (oxyphile, eosinophile) granulations red—by eosin (acid). The neutrophile granulations *are not stained* by this solution. Malarial organisms *blue*—by methylene blue.

Wright's Stain.—See p. 616, (3).

Jenner's Stain.—See p. 618, (4).

The recognised varieties of leucocytes found in normal blood by the triple stain are as follows. They are arranged according to Uskow's classification, which regarded each as the parent of the succeeding form. Hæmatologists, however, are inclining to the view which considers them as originating from different sources.

(1) **Small Lymphocytes.**—These are leucocytes not larger and often slightly smaller than a red cell (Plate IV). Presumably they are formed in the lymph nodes, hence the name. The lymphocyte is small, mononuclear, and its single round or deeply indented nucleus, which stains very heavily, constitutes the bulk of the cell. Around the nucleus is a narrow rim of protoplasm, homogeneous and *without granules.*

(2) **Large Lymphocytes.**—These (Plate IV) vary from 8 μ to 10 μ in diameter. The nucleus is single, large, and may be round, oval, or kidney-shaped. It stains poorly, its light bluish tint contrasting with the darkly dyed nucleus of the small lymphocytes. It is surrounded by a considerable band of protoplasm, homogeneous and *without granules.* The cell as a whole has a pale transparent appearance. The large lymphocyte is perhaps a later stage in the development of the small lymphocyte, from which it differs only in size, depth of staining, and amount of protoplasm. Both have non-granular protoplasm. It was and still is asserted by some writers that the large lymphocytes are formed in the spleen (hence sometimes called splenocytes) and bone marrow.

It is possible that some difficulty may arise in distinguishing between a nucleated red cell and a lymphocyte, because of a certain similarity in their appearance. The discrimination may be made by remembering that the outline of the latter is more irregular, its border of protoplasm is not so wide, and its nucleus does not stain so evenly throughout as that of the normoblast or megaloblast.

(3) **Large Mononuclear Leucocytes.**—These are large cells, two or three times the diameter of a red cell, with a large, oval, eccentric, faintly staining nucleus and a rather abundant cell body.

(4) **Transition Forms.**—These cells resemble the large mononuclears except that the nucleus takes a horseshoe form and stains more deeply. A few neutrophile granules are often present in the protoplasm.

(5) **Polymorphonuclear (or Polynuclear) Neutrophiles.**—These cells (Plate IV) average about 10 μ in diameter. The nucleus is long, twisted, and extremely irregular in shape. Its sinuosities are such that portions of it are hidden by the granulations of the protoplasm, and it appears to be broken or separated into two or more fragments. Hence the name "polynuclear," which was given because of the impression that the cell contained more than one nucleus. But the latest authorities incline to the opinion that the nucleus is "many shaped" rather than that the cell is "multinucleated." The nucleus stains an irregular dark blue or greenish blue, some parts taking up more of the dye than others.

The special characteristic of this cell, from which is derived the second word of its name, consists in the presence of neutrophilic granules in the protoplasm of the cell body. They are extremely fine and, as the cell is spherical, completely envelop the nucleus. They do not stain except with some such combination as the triple stain of Ehrlich; by which, as previously stated, they are coloured violet, lilac, or pink. The polymorphonuclear neutrophiles are the "adult" or "ripe" cells, and constitute the bulk (70 to 72 per cent) of the leucocytes which are found in normal blood, and form the vast majority of pus cells.

(6) **Eosinophiles.**—These cells (Plate IV) are a little smaller and more irregular in shape than (4). The nucleus is polymorphous, like that of the neutrophiles, but stains more evenly although not so darkly. The granules are large, spherical, refractive, of uniform size, and are grouped loosely around the nucleus, sometimes lying free, not enveloping it as do the fine granulations of the preceding variety. They are stained a bright pink, red, or copper colour, by acid dyes (e. g., eosin, acid fuchsin). There are those who believe that the eosinophiles originate from the bone marrow only and for that reason call them "myelogenic" leucocytes.

(7) **Basophilic Leucocytes** (*Mast Cells*) are cells usually having a polymorphous nucleus and granulations which do not take either Ehrlich's triple or Plehn's double stain, but which have an affinity for dahlia, a basic dye, in the following mixture:

Filtered saturated alcoholic solution of dahlia..	50.00 c. c.
Glacial acetic acid..........................	10.00 "
Distilled water	100.00 "

They may be found in very small numbers in normal blood. Although they have been met with rather more abundantly in leucæmia, their clinical significance is as yet *nil.*

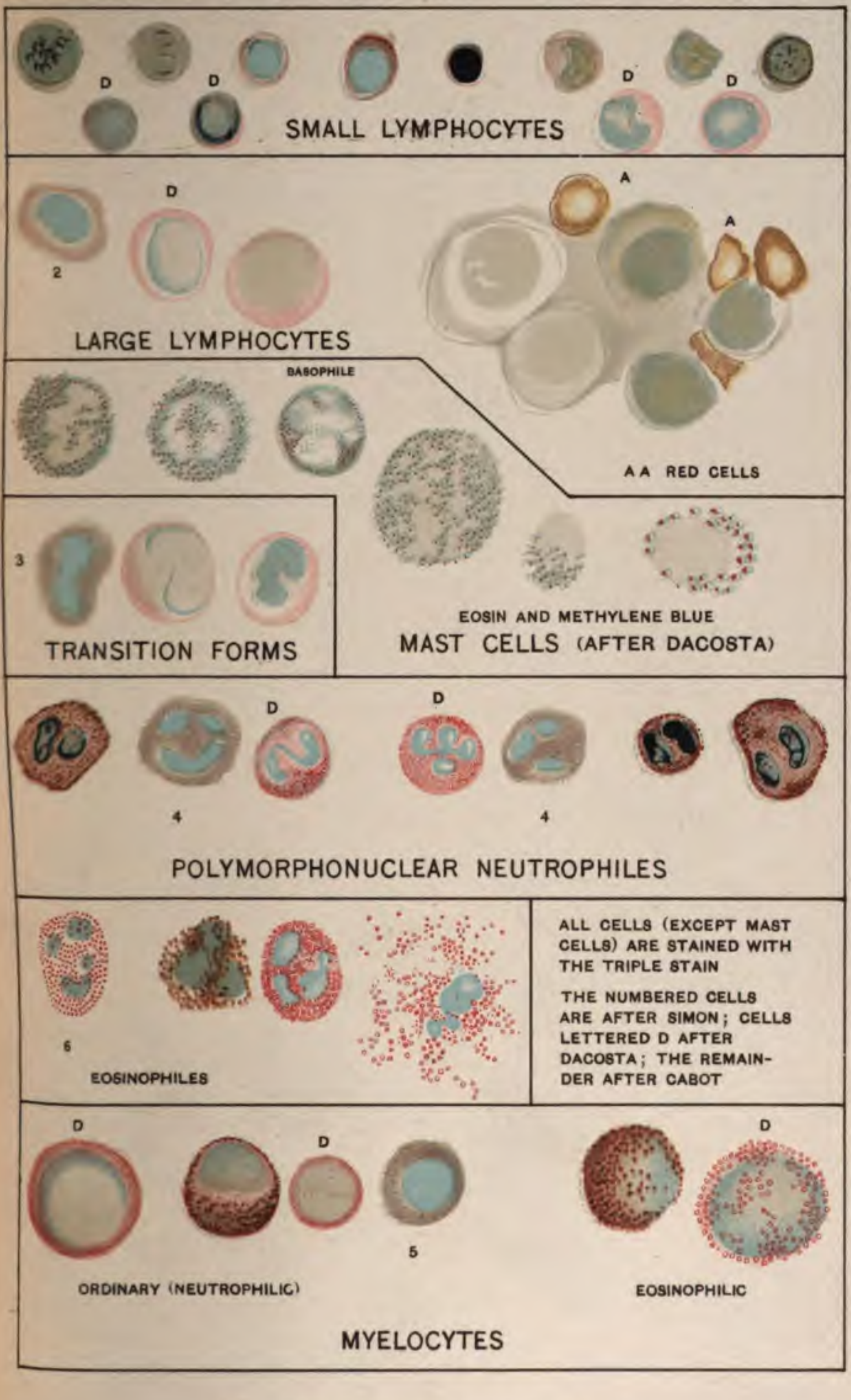
The foregoing varieties of white cells are normally found in the circulating blood, but there are certain leucocytes which make their appearance in the blood drops only in pathological conditions, viz., the

(8) **Myelocytes.**—These cells belong to the bone marrow, whence the name. Two forms are recognised (Plate IV).

The first, called simply a *myelocyte*, has the nucleus of a large lymphocyte and the granulations of a polymorphonuclear neutrophile. The nucleus is spherical or egg-shaped, placed to one side (excentrically), and stains an even pale-blue colour. The cell body is filled with neutrophilic granules, and these, as already stated, can be detected only by Ehrlich's triple stain.

The second form, the *eosinophilic myelocyte*, resembles the first, except that the granules, instead of being neutrophilic, are eosino-

PLATE IV.



ALL CELLS (EXCEPT MAST CELLS) ARE STAINED WITH THE TRIPLE STAIN

THE NUMBERED CELLS ARE AFTER SIMON; CELLS LETTERED D AFTER DACOSTA; THE REMAINDER AFTER CABOT

THE VARIETIES OF LEUCOCYTES

philic, staining a bright red or copper colour with Ehrlich's triple dye. Like the myelocyte, it inhabits the bone marrow.

Method of Differential Counting.—To obtain accurate results in the colour analysis of the leucocytes it is necessary to count 1,000 corpuscles, although a count of 500 is probably trustworthy. If they are present in normal numbers one will have to examine 2 or 3 cover glasses of the usual size (⅞ inch) before the larger number is enumerated. It is convenient to start at the upper left-hand portion of the film, travel horizontally to the right upper angle, then move downward the diameter of one field, travel back to the left, move downward one field, pass to the right, and so continue until the entire film has been traversed or the desired number obtained. A mechanical stage, although not necessary, will greatly facilitate accurate covering of the film area. As the leucocytes come into the successive fields the type of each one is determined and recorded in properly headed ruled vertical columns by marking a short vertical line in the proper column. The small and large lymphocytes may be estimated separately, but usually they are counted together. The large mononuclears and the transitionals are also counted together.

Leucocytes in Health.—It is necessary next to know the total number of leucocytes, and the percentage of the various forms. In health the leucocytes number on an average 7,000 to the cubic millimetre, a proportion of 1 white to 700 red. They may vary within physiological limits from 5,000 to 10,000; in proportion to the red, 1 : 1,000 and 1 : 500. The percentage of the different varieties of leucocytes in normal blood is as follows (Ehrlich):

Lymphocytes	22 to 25	per cent
Large mononuclear leucocytes	1	"
Transition forms	1 to 3	"
Polymorphonuclear neutrophiles	70 to 72	"
Eosinophiles	2 to 4	"
Mast cells	0.5	"

Leucocytes in Disease.—Pathological changes relate either to alterations (increase or diminution) in the total number of the leucocytes (quantitative), or in the proportion of the different forms (qualitative). An increase in the total leucocyte count is called *leucocytosis* or *hyperleucocytosis.* The latter term is preferred, perhaps with greater propriety, by some writers. A decrease is termed *leucopenia* or *hypoleucocytosis.*

(1) Increased Leucocyte Count.—It is necessary to recognise several forms of leucocytosis, the distinctive differences depending upon the presence or absence of qualitative alterations—i. e., whether the relative percentage of the different varieties remains unchanged, or whether one variety has increased out of all proportion to the normal ratio. The following subdivisions are clinically convenient. The first mentioned is normal; the others are pathological.

1. *Physiological Leucocytosis.*—There is a moderate hyperleucocytosis, the percentage of varieties remaining as it is in normal blood, which occurs in certain physiological conditions. It is found in the newborn infant (averaging 18,000); in pregnancy (from 10,000 to 16,000); after exercise (from 11,000 to 13,000); after cold baths (averaging 13,000); and just before death. The leucocyte count also rises after taking food (digestive hyperleucocytosis) usually to an average of 10,000, sometimes to 13,000 or more. Its presence within 1, 2, or 3 hours after a hearty meal is not pathological. The *absence* of this normal increase is said to be a point in favour of gastric cancer (although the total leucocyte count may be large) as against gastric ulcer, for in the latter disease the increase occurs as usual. It is obvious that the normal digestive leucocytosis must be taken into account in comparing successive examinations on different days in the same individual, and an effort made to take the blood as nearly as possible at the same hour before or after a meal. It is always well even in a single count to mention its relation to the digestive act.

2. *Polymorphonuclear Leucocytosis.*—This name is given when the hyperleucocytosis is due mainly or alone to an increase (relative or absolute) in the number of the polymorphonuclear neutrophiles (adult cells). It is the type of leucocytosis usually seen in pathological conditions. The total count may rise to 100,000, but is most commonly somewhere under 50,000. It occurs in—

All acute inflammatory diseases—e. g., abscesses of all varieties in any part of the body; serous membrane inflammations (pleurisy, peritonitis, etc.); gangrenous inflammations (appendix, cancrum oris); and many of the cutaneous inflammations.

Certain acute infectious diseases, if attended by local inflammatory lesions or complications—e. g., erysipelas, pneumonia, phthisis (only when cavities exist), parotitis, scarlatina, rheumatic fever, meningitis, diphtheria, or pyæmia. If the infection is not accompanied by lesions which give rise to a local reaction there is no leucocytosis —e. g., typhoid fever, in which, if it is uncomplicated by some localized inflammatory process (pneumonia, abscess), the leucocyte count is decreased, not increased—a valuable differential point in many cases. It is especially useful in discriminating between appendicitis and typhoid fever, the absence of hyperleucocytosis pretty positively barring out the former.

The cachexia of malignant disease, especially if the growth is rapid or metastases have occurred. The extent of the leucocytosis is thought by some writers to be commensurate with the amount of local reaction in the tissues surrounding the growth. It is to be remembered that the *absence* of a leucocytosis does not forbid malignant disease, as the neoplasm may be small and of slow growth, under which circumstances (slight local reaction) the number of white cells may remain normal. In the differential diagnosis between malignant and non-malignant disease of the stomach the presence of a leucocytosis points toward the former. Leucocytosis of a moderate degree occurs in other secondary anæmias besides that due to cancerous disease. Subsequent to large hemorrhages there is a temporary leucocytosis (16,000–18,000) lasting from one to several days. The longer duration is in cases where the bleeding is due to traumatism.

"In addition to these various forms of hyperleucocytosis an increase of the neutrophiles is further observed under conditions which do not as yet permit of an appropriate classification; in some cases, no doubt, it is of toxic origin; in others it may be referable to an abnormal distribution of the cells; in still others to a coexistent anæmia, etc. Such conditions are rickets, gout, acute yellow atrophy, advanced hepatic cirrhosis (especially when associated with jaundice), acute gastro-intestinal disorders, acute and chronic nephritis, hydronephrosis, etc." (SIMON.)

3. *Mononuclear Leucocytosis* (*Lymphocytosis*).—In this form the lymphocytes are present in increased numbers. The increase may be *absolute*—i. e., the total leucocyte count is above the normal, the lymphocytes constituting a large proportion of the increase; or *relative*, the total number of leucocytes being normal or even decreased, while the percentage of lymphocytes is above the normal. The lymphocytes may be either large or small. It is important to separate these two conditions, as their diagnostic significance differs widely, as follows:

Absolute Lymphocytosis.—This condition—total number of leucocytes increased, lymphocytes in the majority—is found in but 2 ailments: sarcoma attended with metastases in the bone marrow, and lymphatic leucæmia. In view of the infrequency of the former disease, an absolute lymphocytosis is almost invariably significant of lymphatic leucæmia, and the lymphocyte may be considered the characteristic cell of this disease.

Relative Lymphocytosis.—This condition—total number of leucocytes normal or less, percentage (relative number) of lymphocytes increased—is found in healthy infants and in various diseases of the earliest years of life. It also occurs in pernicious anæmia, chlorosis, typhoid fever, pertussis, broncho-pneumonia, measles, chronic malaria, secondary syphilis, rachitis, hæmophilia, goitre, and certain cases of exophthalmic goitre.

4. *Eosinophilic Leucocytosis* (*Eosinophilia*).—This is an increase in the percentage of eosinophiles. The total number of leucocytes may or may not be greater than normal. A marked eosinophilia is very suggestive of trichinosis, anchylostomiasis, or amœba coli. It occurs also in varying degrees in bronchial asthma, emphysema, phthisis with cavities; in disease of the bones (sarcoma, myelogenic leucæmia); in some diseases of the nervous system (neurasthenia, hysteria, certain psychoses); chlorosis; diseases of the skin (eczema, pemphigus); in the lithæmic or uric-acid diathesis; and, during early convalescence, in the acute febrile infections, except measles and scarlatina.

5. *Myelocytosis.*—This term may be employed to indicate the presence of myelocytes, cells which are never found in normal blood. When found in large percentage (20–60) containing either neutrophilic or eosinophilic granules, together with a great increase in the total number of leucocytes, they are extremely characteristic of the spleno-medullary (or lieno-myelogenous) form of leucæmia. Like the normoblast, they are found in small percentage in many diseases, particularly in pernicious anæmia (rarely exceeding 9 per cent), chlorosis, and severe secondary anæmias.

(2) **Decreased Leucocyte Count.**—The absence of an increase, perhaps even a diminution (leucopenia, hypoleucocytosis), in the number of leucocytes is of much value in certain differential diagnoses. Thus, if it becomes a question whether a given case is one of appendicitis or of uncomplicated enteric fever, the presence of an increased number of leucocytes speaks against the latter, as it is one of the infections in which a leucocytosis is notably absent.

The diseases in which there is either an absence of leucocytosis, the number of white cells remaining within normal limits, or an actual leucopenia, are typhoid fever, measles, epidemic influenza, malaria, miliary tuberculosis, and tuberculous affections of the meninges, lungs, pleura, pericardium, peritonæum, and bones. Leucopenia is found in splenic and pernicious anæmia, sometimes in the secondary anæmias, and, associated with lymphocytosis, in conditions of malnutrition and starvation.

There is little to be said concerning a decrease, either relative or absolute, in any one variety of leucocyte. The *eosinophiles* may totally disappear during the febrile stages of acute infectious diseases, except scarlet fever and malaria, to return after the fever has subsided; and are relatively diminished during digestion, after castration, and in most leucocytoses. The *lymphocytes* are relatively diminished in spleno-medullary leucæmia.

C. BLOOD PLATES AND MÜLLER'S BLOOD DUST

Blood plates are small rounded bodies frequently agglutinated into bunches. They number from 200,000 to 500,000 under normal conditions. As they promptly disintegrate on the slightest exposure to air they are rarely seen unless the blood is immediately mixed with Hayem's fluid, the composition of which is as follows :

Bichloride of mercury	0.5 gramme.
Sodium sulphate	5.0 grammes.
Sodium chloride	2.0 "
Distilled water	200.0 "

A drop of the fluid is placed upon the lobe of the ear, a puncture made through the drop, and the mixed fluid immediately examined. Variations in the number of blood plaques have as yet so little clinical significance that there is nothing to be gained by counting them.

The "hæmoconien" or blood dust of Müller consists of round, colourless, highly refractile granules varying from ¼ to 1 μ in diameter. They are not motile, but exhibit rapid molecular motion. There is evidence to show that they are the extruded granules of the eosinophilic and neutrophilic corpuscles. As yet they have no clinical significance.

D. PARASITES IN THE BLOOD

The three micro-organisms of greatest clinical importance to be found in the circulating blood are the *Plasmodium malariæ*, the *Spirochætæ* of relapsing fever, and the *Filaria sanguinis hominis*.

Plasmodium Malariæ.—(1) **The Life History of the Plasmodium.**—There are three forms or varieties of this organism : the *tertian*, *quartan*, and *æstivo-autumnal*. The cycle of the growth and development of each form taken separately is briefly as follows :

Tertian Variety.—In the early stage (Plate V) it is a small colourless "hyaline" body, with somewhat indistinct outlines, occupying only a small portion of the interior of the red cell and situated excentrically. If living, it exhibits distinct and rapid amœboid movements. The hyaline body rapidly increases in size, small groups of actively moving pigment granules appear within it, and the containing red cell becomes pale and swollen. When the parasite has attained its full growth the outlines of the red cell are hardly distinguishable; the activity of the amœboid movements of the organism diminishes, and the pigment tends to collect along the circumference. The final stage of development then begins. Signs of segmentation are visible around the periphery of the parasite, the pigment granules pass inward to be collected in the centre, and finally, when the division is complete, a "rosette" is formed by the symmetrical arrangement of the segments (10 to 20) radiating from the central rounded mass of pigment. These segments lose their regular arrangement and form a confused group or are held loosely together by an apparent envelope. The tertian parasite passes through these various stages in 48 hours.

Quartan Variety.—The description of the tertian cycle just given answers also for the quartan form of the organism (Plate V), except that the latter is smaller, its outlines are more sharply defined, its amœboid movement is less active, its pigment is coarser, darker and less motile; the segments number from 5 to 12, and the rosette arrangement is more perfect and symmetrical. It requires 72 hours for its development. The containing red cell becomes shrunken rather than swollen, and brassy rather than pale in colour.

Æstivo-autumnal Variety.—This variety begins, as do the tertian and quartan forms, with a "hyaline" body (Plate V) which is actively amœboid and develops in a similar manner, but the organism is much smaller and the pigment granules are scanty and motionless. The segmenting forms are rarely seen in the blood obtained from puncture of the ear or finger, as this stage in its development takes place in the internal organs, particularly in the spleen and bone marrow. The erythrocyte host is shrunken and brassy like that of the quartan parasite. The æstivo-autumnal form requires from 24 to 48 hours for its development. Finally, if the disease has lasted for at least a week, pigmented crescents and spherical or oval bodies may be found which appear to be pathognomonic of this variety of the organism. The crescent contains pigment, and the remains of the red corpuscle are frequently seen clinging to some portion of its border.

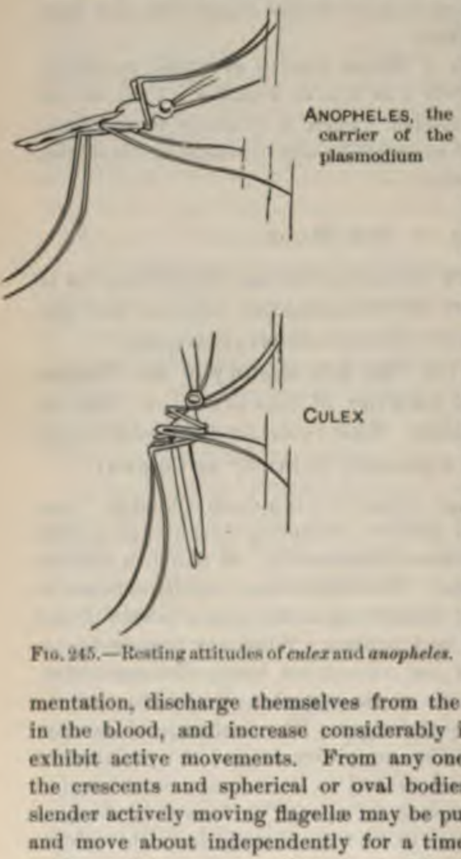
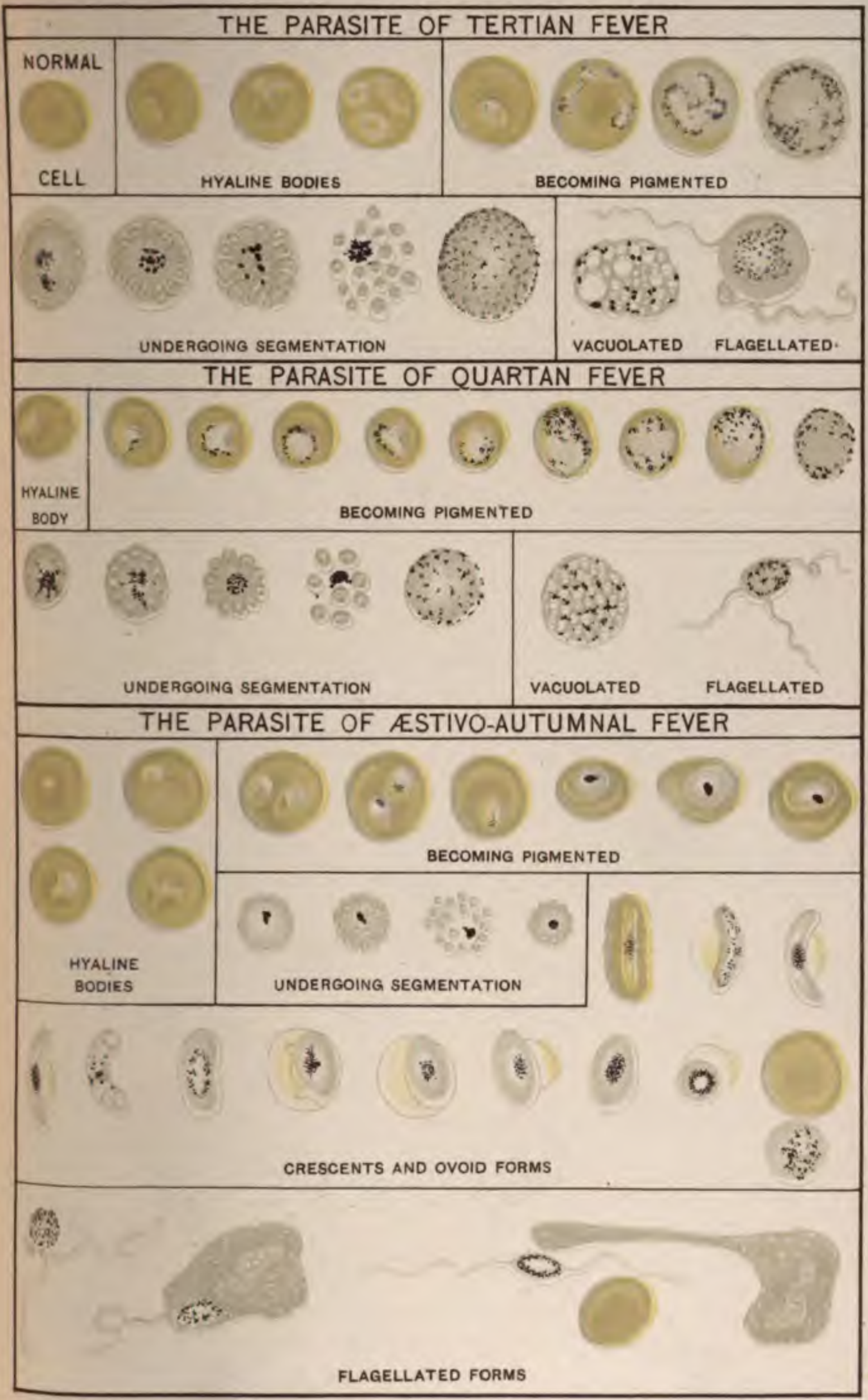

ANOPHELES, the carrier of the plasmodium
CULEX

FIG. 245.—Resting attitudes of *culex* and ***anopheles.***

It is seen that all three varieties begin as a hyaline body, which becomes pigmented and proceeds to segmentation, but that each form presents certain peculiarities in development and morphology which enable the expert, but not the occasional examiner, to distinguish one from the other. Furthermore, each variety may give rise to extra-cellular, vacuolated, and flagellate bodies. Some of the tertian and quartan organisms, instead of undergoing segmentation, discharge themselves from the containing red cell, thus lying free in the blood, and increase considerably in size while the pigment granules exhibit active movements. From any one of these free bodies, and also from the crescents and spherical or oval bodies of the æstivo-autumnal organism, slender actively moving flagellæ may be put forth. The flagellæ may break off and move about independently for a time. The flagellate organisms of the tertian variety are larger than those of the quartan and æstivo-autumnal forms.

It may be considered as proved (THAYER) that the malarial parasite passes through two cycles of development: one, usually asexual, in the human body,

PLATE V.



THE PLASMODIUM MALARIÆ
SELECTED AND REPRODUCED FROM THAYER'S PLATES

ending in segmentation; the other, reproductive, taking place in the mosquito, in which the male elements undergo flagellation and penetrate the female elements. The newly formed parasites enter the salivary glands and are inoculated into the human being by the bite of the insect. Only one genus of mosquito (*anopheles*) appears to be the active agent, the ordinary mosquito (*culex*) not participating in the work. It has been stated that the attitude of the two varieties when at rest upon a wall furnishes a ready method of recognition (Fig. 245, reproduced from the British Medical Journal of September 30, 1899), but unfortunately only a few species of anopheles rest with the axis of the body at right angles to the wall, so that this method of identification is not trustworthy (Sambon, Low).

(2) **How to Detect the Plasmodium.**—In searching for the malarial organism it is always best to examine fresh unstained blood, at the bedside if possible; but if this is impracticable, warm the slide, seal the cover glass with vaseline, and the organism will retain its vitality for one or two hours at least, in summer weather perhaps for several hours. If a considerable time must elapse before examination, one should make films, fix them by ether and alcohol (not by heat), and stain with Plehn's solution, by which the malarial organisms are coloured blue, the nuclei of the leucocytes blue, and the red cells pink; or with Wright's stain which colours the red cells pink, the parasites blue, and the chromatin lilac through red to almost black. The blood may be obtained and coloured simultaneously by puncturing through a drop of staining solution.

As one of the two principal reasons for a failure to find the parasite lies in having too thick a layer of blood, it is well for the observer to satisfy himself that the red cells lie flat and one deep, without excessive overlapping or rouleau formation. This desideratum is best assured by following the directions already given relative to the use of a very small drop of blood in preparing the specimen. The second reason for non-discovery of the parasite is a too brief search. At least half an hour should be devoted to the quest before the presence of the organism is denied. In well-marked cases one organism may be found in every field; in slighter attacks a number of fields may be examined before the search is rewarded. Although the examination may be made at any period, it is probable that the best time for taking the blood is from 8 to 12 hours before or after a chill, as it is then that the parasite is found most abundantly in the peripheral blood. If quinine has been given it may be impossible to find them. The specimen having been prepared, the *red cells* should be examined with a $\frac{1}{12}$-inch objective, using a very moderate illumination.

Look for: 1. Red cells containing actively moving black specks or dots—i. e., pigment granules in the living organism. 2. Unusually pale red cells containing clear areas, these areas of irregular and changing shape—i. e., hyaline forms with amœboid motion. 3. Extra large red cells—i. e., corpuscles swollen by the growth of the parasitic organism. Unless the slide, cover, and punctured surface have been thoroughly cleansed, the particles of dirt which remain may be mistaken for pigment granules, especially if the organism is dead and its movements have ceased. If, under such circumstances, the distinction can not be made, a fresh preparation must be secured.

After a little experience the pigmented organisms are readily identified. Attention is directed to the flagellæ, if such be present, by the commotion among the red cells caused by the active whipping movements of the former. For the beginner the hyaline bodies or youngest forms are the most difficult of recognition. This arises from the fact that the white circles which are to be seen in many of the normal red cells under varying conditions of illumination or partial desiccation are likely to be mistaken for the hyaline bodies. The discrimination can usually be made by attention to the following points: The apparent but not real body (artefact) may be found in much larger numbers, a dozen or more to each field; it lies in the centre of the erythrocyte, it is circular, its edges are sharply defined, it is white and brilliant, it increases or diminishes in size as one focuses up or down, and its motion, if any, is wavy and undulating. On the other hand, the hyaline organism is usually found in much less abundance, one to one or several fields, it is placed excentrically, its shape is irregular and branching, its edges ill defined, it is pale with a slight yellowish tinge, if the focus is altered it simply becomes more or less distinct, and finally it undergoes changes of shape and position.

One more point is to be remembered, viz., that pigment-bearing leucocytes may be encountered in the blood of malarial cases. These leucocytes, usually of the polymorphonuclear (neutrophilic) variety, take into themselves (phagocytosis) the masses of pigment and pigmented fragments of the old segmenting or disintegrating organisms.

(3) **The Clinical Relations of the Plasmodium.**—The relation of the events of the life history of the organism to the clinical events of a malarial paroxysm is of importance. The segmentation of the organism immediately precedes the chill and the fever, these latter phenomena depending perhaps on the formation or liberation of some toxic material at the time of segmentation. Consequently the segmenting forms, if present in the peripheral circulation, will be found at the beginning or the height of the paroxysm, while the hyaline forms are discoverable during or subsequent to the seizure.

As the *tertian parasite* requires 48 hours to complete its cycle of development, segmentation and paroxysm will occur every other day, provided that only 1 group or brood of the parasite is present. More commonly 2 groups of different ages exist (double tertian), segmenting on alternate days and causing the quotidian (daily) type of fever.

The *quartan parasite*, requiring 72 hours to reach maturity, gives rise to paroxysms separated by 2 days of apyrexia, provided 1 brood only is present; if there are 2 broods there will be paroxysms on 2 successive days followed by 1 free day (double quartan); while 3 groups will cause daily or quotidian attacks like the double tertian type.

As the *æstivo-autumnal parasite* has a variable period of development (24 to 48 hours), segmentation and fever may be extremely irregular, and sometimes continuous, organisms at different stages of development coexisting in the blood.

Owing to the rapid destruction of the red cells and the transformation of the hæmoglobin into pigment, the patient becomes quickly and characteristically anæmic. If pigmented leucocytes are found the existence of malaria is to be suspected, as practically the only other conditions in which they occur are relapsing fever and melanotic neoplasms. The periodicity of the malarial paroxysm is obviously due to the natural history of the parasite.

Spirochætæ of Relapsing Fever.—The specific organism of relapsing fever may be found in a specimen of fresh blood prepared in the same manner as in the search for the malarial parasite. The spirilla or spirochætæ are narrow spiral filaments 36 to 40 μ in length and are actively motile, attracting attention by the movements imparted by them to the red cells. They are present only during the fever and may then be abundant, 20 or more appearing in a single field of the microscope. In the apyrexial periods small glistening bodies, believed by some to be the spores of the organism, are to be found free in the blood.

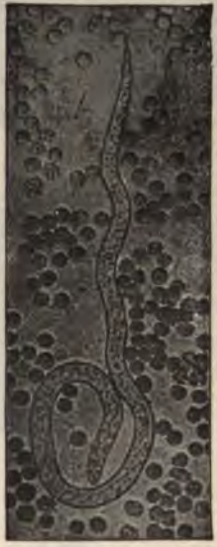


Fig. 246.—Filaria alive in blood. Photomicrograph × 400 diameters. (Henry.)

Filaria Hominis Sanguinis.—There are several varieties of this parasite, for the distinguishing points of which special works should be consulted. The most important of these is the *Filaria nocturna* (Fig. 246), which is held responsible for certain forms of chyluria, elephantiasis, and lymph scrotum. The adult or parent organisms are slender and hairlike, varying from 3 to 6 inches in length, and inhabit exclusively the lymphatics and tissues. The embryos, of which the female adult discharges a large number, live in the circulating blood, which they enter by way of the lymphatic vessels. The average length of the embryo is $\frac{1}{75}$ of an inch and its width about that of a red cell. In this variety they make their appearance in the peripheral blood only at night, unless the habits of sleeping and waking are reversed, when the contrary holds good; in another only during the day (*F. diurna*); while a third variety may be found at any time (*F. perstans*). The search for the embryos in a suspected case should therefore be made both in the daytime and at night. They are to be sought for in a fresh drop of blood, using a low power. As they move pretty actively their presence is usually first noted by the commotion which they create among the red corpuscles. When they finally come to rest it will be seen that each embryo is contained in a sheath, within which it contracts and extends, and that its body is somewhat granular and transversely striated.

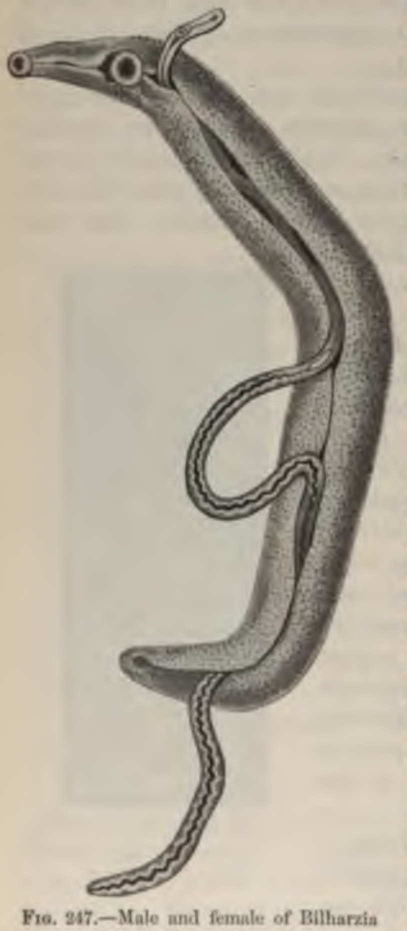


FIG. 247.—Male and female of Bilharzia Hæmatobia. (Looss.)

Piroplasma Hominis.—Tick Fever, or Spotted Fever, is a disease peculiar to certain sections of Oregon and other Western States. J. F. Anderson and others have described an amœboid, non-pigmented parasite in the red cells—the *piroplasma hominis*. It is regarded as entering the circulation from the bite of certain forms of wood-tick.

Distoma (Bilharzia) Hæmatobium.—This trematode is most abundant in the blood of the portal system, while the ova (Fig. 248) lodge in the capillaries, particularly of the bladder, urinary organs, rectum, and lungs. They may frequently be identified in blood-clots from the bladder as whitish, melon-seed-shaped specks about $\frac{1}{100}$ inch in length. The ciliated embryo can be seen through the shell (Fig. 249). The parent worms differ according to sex (Fig. 247). The male is about 12 mm. long by 1 mm. broad. At the extreme anterior end is a circular sucker. A second oval one is just posterior to it on the ventral surface, which is grooved to receive the female, a much longer and thinner organism than the male. The disease is seldom seen outside of northeastern Africa and Arabia.

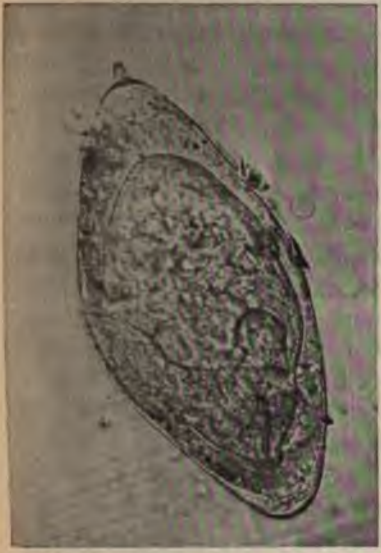


Fig. 248.—Egg of Bilharzia Hæmatobia.

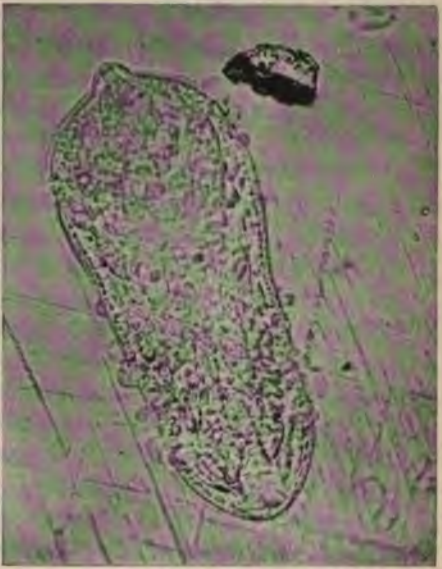


Fig. 249.—Free swimming embryo of Bilharzia Hæmatobia.

Trypanosomiasis.—The mammalian trypanosomes may be divided into a (1) non-pathogenic, and (2) pathogenic group. Among the latter may be mentioned Tr. evansii, the cause of surra; Tr. brucei, the cause of nagana; Tr. equiperdum, the cause of dourine; and Tr. gambiense, the cause of sleeping-sickness in man.

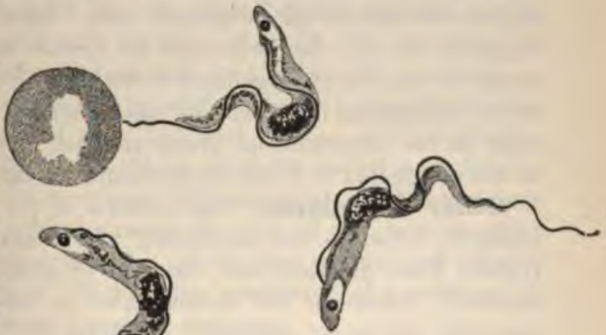


Fig. 250.—Trypanosoma gambiense in human blood (Dutton).

These diseases are conveyed to warm-blooded animals and man by the bites of certain insects, generally flies. It has been definitely proved that sleeping-sickness is conveyed to man by the bite of a tsetse-fly (*Glossina palpalis*). In certain cases the flies seem to act as mere mechanical carriers of the parasites, while in other instances they may act as true intermediate hosts, multiplication forms of the trypanosomes having been found in the stomachs of certain of the flies.

The *Trypanosoma gambiense* is about 18 to 25 μ long and 2.5 μ wide, with one flagellum anteriorly and an undulating membrane which extends the whole length. Near the posterior extremity of the parasite is seen a body known as the micronucleus, and a true nucleus can also be made out. When stained by the Romanowsky or Wright method, the nucleus and flagellum are stained red, the micronucleus dark red or almost black, and the body of the parasite blue. The enveloping membrane may also take a red stain.

When fresh blood is examined the parasite is seen in the plasma, moving with a screwlike motion among the red cells. Their number varies, but generally only a few are present in the blood of man. At the present time simple fission is the only mode of multiplication known. The parasite can generally be found in the enlarged glands which are a constant feature of early cases of human trypanosomiasis. The Leishman-Donovan bodies which are found in the disease known as kala-azar are small, oval, or round bodies from 2.5 to 3 μ in diameter, with a nucleus and micronucleus, but no undulating membrane. They are not seen in the circulating blood, but are easily found in blood obtained by splenic puncture. Most of them are intracellular, within the endothelial or splenic cells. The exact position of the organism can only be determined by further study. Rogers has succeeded in cultivating flagellated forms, but on account of the absence of the undulating membrane he has concluded that the organism belongs to the herpetomonas group and not to the trypanosomes, and he has designated it as the herpetomonas of kala-azar.

Other Organisms.—The bacteria of the following diseases are regularly found in the circulating blood: anthrax, bubonic plague, typhoid fever, glanders, and the cocci of septic infections; while occasionally organisms are demonstrable in cerebrospinal meningitis, beri-beri, influenza, malignant endocarditis, Malta fever, measles, pneumonia, rheumatism, and scarlatina. The gonococcus may sometimes be isolated in septic cases. As the recognition of most of these organisms calls into play the methods of the bacteriological laboratory they can only be mentioned here. For the technic of obtaining blood for such examinations, see page 639.

E. DIRECTIONS FOR PERFORMING WIDAL TESTS

With Living Cultures.—*Collecting Blood.*—From a free puncture in the ear or finger the blood is allowed to flow into a glass capsule until it is two-thirds full. Pressure may be used to increase the flow. The capsule should be about 2 inches long and $\frac{1}{4}$ inch in diameter, and drawn out into a capillary at either end.

Obtaining Serum, Preparing Dilutions.—The capsule of blood is placed in an ice-box to allow coagulation, or it may be centrifugated. When the serum has separated the tube is filed and broken at a point just beyond the clot, and the serum collected by means of a capillary pipette. The serum is measured with a 1 c.c. pipette graduated in $\frac{1}{100}$ths, and diluted fifty times with a 0.8 per cent salt solution. In case the amount of serum is so small that the mixture does not equal 2 c.c., then the test is to be made on a cover-glass as follows:

Microscopical Test.—A drop of the 1:50 dilution of serum is placed on a cover-glass and there is added to it as much of a 10- to 24-hour agar typhoid culture as will go on the point of a platinum needle. The organisms are to be thoroughly mixed with the serum, and the drop should show only a slight cloud. The cover-glass should now be examined in a hanging-drop slide which has been ringed with vaseline. Should typical clumping take place with this dilution the test is to be repeated with a dilution of 1:100. The reaction is to be considered positive if in every field there are numerous clumps and the bacilli have lost their motility. The presence of a few non-motile bacilli in the spaces between the clumps does not vitiate the test. Control experiments should always be made, using salt solution instead of serum. Care should be taken that not more than the specified needle-point of bacteria be mixed with the drop of serum. In reporting the result the strength of the dilution and the time should be stated.

Macroscopical Test.—Should the original dilution equal more than 2 c. c., it is further diluted to 1:100, and 0.5 c. c. of this dilution placed in a small test-tube. In this is carefully rubbed up 1 loopful (1 mg.) of the 10- to 24-hour agar typhoid culture. The organisms should be thoroughly rubbed against the side of the tube in order to produce an even emulsion. The tube is then shaken and allowed to incubate for 3 hours at 37° C. The appearance of a sediment at the end of this time shows a positive reaction.

The Widal test may be made with dried blood, but its only recommendation is the convenience with which blood may be transported.

With Dead Cultures.—*Formalized Cultures.*—The Widal test may also be made with formalized cultures (Dreyer, *Journal of Pathology and Bacteriology*, January, 1909).

Ordinary veal peptone bouillon, without sugar, is used as a culture medium. It should be as pale as possible, and should not be autoclaved for more than 15 minutes at 115° C. The strain of *B. typhosus* should be passed through a number of bouillon tubes day by day for several weeks before it is used in the preparation of the culture for agglutination, since these passages augment its agglutinability and result in the production of a more homogeneous strain. After inoculation the flask of bouillon is kept at 37° C. in the incubator for 22 to 24 hours. It is then well shaken up and 0.1 per cent of the ordinary 40-per-cent solution of formalin is added. After another careful shaking it is placed in the ice-chest at about 2° to 6° C. It is kept there for 3 days, and shaken every day. It is always sterile at the end of the 3d day. Should it happen that the culture does not form an entirely homogeneous emulsion it must be filtered through sterilized cotton wool or coarse filter paper. Finally, it is put in a sterile flask, with a rubber cork, and kept in the cold and in the dark.

To make this test the finger or ear is pricked and about 15 drops of blood are collected in a dry sterilized test-tube. The tube is then closed with a cotton plug. In a few hours the blood will have coagulated and enough serum collected. Four drops of this serum are next measured out into another test-tube. The pipette is then washed out in tap water, and 36 drops of water are added to the serum and the two are carefully mixed, this giving a serum diluted 1:10.

Five small sedimentation tubes are placed in a rack, and counting from left to right water is added as follows:

Tube 1	0 drops of water.
" 2	5 " " "
" 3	8 " " "
" 4	9 " " "
" 5	10 " " "

Next the serum diluted 1:10 is dropped from the pipette:

Tube 1	10 drops of the 1 : 10 serum.
" 2	5 " " " "
" 3	2 " " " "
" 4	1 " " " "
" 5	0 " " " "

The pipette is again washed in water, and to each tube is added 15 drops of the sterile *B. typhosus* culture. Each tube is then well shaken and the rack placed in a water-bath or incubator at a temperature between 37° C. and 55° C. for 2 hours. Test-tubes 1 to 4 now contain serum in dilutions of 1:25, 1:50, 1:125, 1:250. The 5th tube contains no serum and serves as a control for the detection of spontaneous agglutination. It should never be omitted. If a measurable amount of agglutinin is present in the serum, one or more of the tubes will become clarified and less opalescent. A light flocculent deposit will collect at the bottom of such tubes in the course of 2 or 3 hours, or even sooner, while the control tube remains diffusedly turbid. A positive result in tube 1 (serum 1:25) must be taken as a somewhat doubtful reaction. Well-marked agglutination at a dilution of 1:50 or higher is nearly always definitely diagnostic of the presence of active typhoid infection.

Ficker Diagnostic.—Ficker has devised a modification of the Widal test which does not require the employment of a living culture of the typhoid bacillus. The method is thoroughly reliable, and is particularly useful for those who for any reason can not avail themselves of laboratory facilities. A sterilized suspension of the bacilli is the essential agent, and this, together with the necessary apparatus and directions for its use, is furnished, by at least two large firms, under the respective names of "Typhoid Diagnostic," and "Typhoid Agglutometer."

In general, the test is as follows: A puncture is made and about 1 c. c. of blood obtained in a small blood-tube. When the serum has separated, it is diluted in stated percentages in the test-tubes furnished, and the diagnostic reagent added. If positive, the bacilli, within 10 or 12 hours, agglutinate and sink to the bottom of the tube, leaving the supernatant fluid clear.

Diagnostic Value of Widal Reaction.—A negative result with these tests should not be interpreted as meaning that the patient is not suffering from a typhoid infection. In this case the serum should be tested with paratyphoid bacilli. A positive reaction seldom appears before the 7th day, and is sometimes delayed until the 2d or 3d week. Therefore, in case of a negative result, further tests should be made at intervals of several days.

The latest statistics indicate that from 95.5 to 97 per cent of cases of typhoid afford a positive reaction, while in non-typhoid cases 1.5 per cent may show it. If the serum is obtained from a patient *in articulo mortis* the reaction may be absent.

A positive diagnosis may often be made during the first week of the disease by means of a blood culture, since the organisms are most numerous in the blood at that time, and diminish in numbers as the disease progresses.

F. BLOOD TEST FOR DIABETES

Williamson's test.—Place in a small test-tube 40 c. mm. of distilled water. Puncture the finger; obtain 20 c. mm. of the suspected blood (using Gowers' hæmatocytometer pipette), and expel it into the water. Then add 1 c.c. of an aqueous, 1:6000, solution of methylene blue and 40 c. mm. of a 6 per cent aqueous solution of potassium hydrate. Prepare another tube, using blood known to be non-diabetic. Place both tubes in boiling water for 4 minutes. A diabetic blood mixture will have turned a dingy yellow; the control mixture remains bluish green.

G. IODINE REACTION IN SUPPURATION

Make a mixture as follows (Goldberger and Weiss):

Iodi. sublim	1
Potass. iodati.	3
Aq. Dest.	100
Gummi ad syrupam.	

Paint some of this solution upon a slide, and press a dried cover-glass film upon it. Or the dried films may be exposed for a few minutes in a vessel containing iodine crystals. After they have turned brown they are mounted in a drop of saturated levulose, when the iodine colour is seen on examination in the red cells. The so-called glycogen granules are mahogany coloured. If suppuration is not going on the red cells stain dark yellow, the white cells light yellow, and their nuclei a citron yellow. If *acute* suppuration is in progress the protoplasm of the white cells will be stained brown, diffusely or as a network.

Barnicot says that a positive reaction is evidence of a toxæmia, bacterial or non-bacterial in origin. The reaction bears no relation to leucocytosis, when both are present in the same blood, other than that of a common cause; the reaction may be absent in leucocytosis, present with a normal leucocyte count, or even with leucopenia. The continued presence of an iodine reaction in pneumonia after crisis is a further aid to other clinical signs which lead one to suspect delayed resolution or some complication. When an accumulation of pus is thought to exist the absence of a reaction is of very great negative value, but with the exception of the two conditions just mentioned the iodine reaction is so inconstant in manifestation or so variable in degree that it can not be granted a serious place among the means at our disposal for the diagnosis or prognosis of disease.

H. OBTAINING BLOOD FOR BACTERIOLOGICAL EXAMINATION

For this operation it is necessary to have a syringe capable of holding 10 c.c., and a flask containing 100 to 250 c.c. of dextrose broth. In place of the broth, 2 tubes of agar, each containing 10 c.c., and 2 sterile Petri dishes can be used. The syringe and needle should be boiled for 15 minutes in a 1-per-cent solution of carbonate of soda.

The arm at the bend of the elbow should be scrubbed with soap and water, followed by alcohol and some antiseptic. If the veins are not prominent a bandage may be tied around the arm near the shoulder. In order to avoid infection from the skin it is necessary to incise the skin over the vein. The needle is now pushed into the vein in the direction of the blood-stream, and the blood enters the syringe, which should fill with very moderate suction.

The needle is now withdrawn and the blood carefully expelled into the flask of broth. If agar is used it must first be melted and then cooled to 45° C. Two or three c. c. of blood are then emptied into each of the two agar tubes. The contents of the tubes should be carefully mixed and then poured into the 2 Petri dishes and allowed to solidify. The cultures are incubated at 37° C. for from 1 to 5 days, and if a growth appears the identity of the organism may be ascertained by the usual bacteriological procedures.

I. DETERMINING THE COAGULATION-TIME OF THE BLOOD

Milian's Method.—A simple method is that of Milian. Place a large drop of blood on the centre of a clean, dry glass slide. In a minute or two tilt the slide on end. If the blood is completely coagulated the profile of the drop is symmetrical and bluntly conical; if not, it is tear-shaped (Fig. 251). The tilting is repeated at short intervals until the coagulation shape appears. The average period from the collection of the drop to complete clotting is, for normal blood, about 5 minutes.

Wright's Method.—In connection with the diagnosis of purpuric eruptions it is often of service to observe the degree of coagulability of the blood, as this is often delayed in the graver forms. For this purpose the apparatus devised by Wright is convenient.

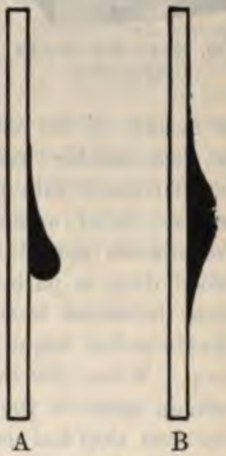


FIG. 251.—A. Incomplete coagulation. Tear-shaped drop. B. Complete coagulation. Convex drop. (Da Costa.)

The essential parts of this instrument are a cylinder for water, surrounded by a jacket of felt and leather filled with 9 pockets to hold a thermometer and 8 coagulation tubes. These are of 10 cm. length and have a lumen of 0.25 mm. The can is filled with water at about 100° F. and the apparatus allowed to stand for a little time until warmed through.

The tubes, which should be numbered consecutively, are then filled in order at intervals of 1 minute with the blood to be tested and replaced in the pockets. After each has remained for 3 minutes the blood is blown out. The first one to show a clot is selected as showing the coagulability, which is expressed in minutes. The normal coagulation-time is from 3 to 5 minutes.

Biffi-Brooks Coagulometer.—The case or metal jacket of the instrument (Fig. 251 *a*) is filled with water at a temperature slightly above that of the body. The glass tube is then half filled with water at the same temperature.

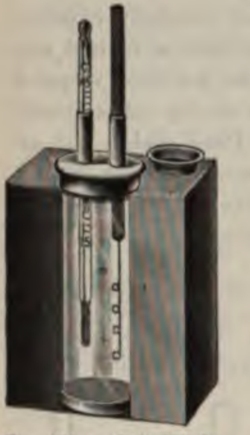


Fig. 251 *a*.—Biffi-Brooks Coagulometer.

For satisfactory results it is necessary to secure a more abundant blood flow than is necessary for counts or ordinary hæmoglobin tests, since all the loops must be quickly and simultaneously filled at the same step. The specimen is therefore better obtained by a small cut with a clean scalpel or cataract knife than by a puncture, and the lobe of the ear is obviously better suited for this purpose than the finger tip.

A large drop of blood is allowed to collect, and if it does not exude rapidly it is to be wiped away and a larger incision made. The loops are then rapidly passed over the blood so that each is filled by a drop of blood which is exactly of the same size in each instance. The preparation rod is then quickly inserted in its tube or carrier, and the drops of blood entered into the chamber filled with moist air just above the water. After a length of time considerably less than the normal coagulation period for that species the first loop with its enclosed blood drop is pushed down into the water; if clotting has not begun the blood is immediately dispelled throughout the water, but if fibrin has begun to form, more or less clot will adhere to the loop. Where the clotting is complete the whole mass of blood will remain intact in the platinum circle even for a considerable time. If the first loop has not coagulated at intervals of one half minute, or at such other spaces as may be desired, according to the nature of the case, the other drops are forced down into the water, and the coagulation-time is computed from the appearance of the blood to the time when fibrin formation is first evident in the loop.

Where the approximate time has been thus determined a second reading where desirable at 10- or 20-second intervals will enable one to accurately determine the exact clotting time of any blood.

The instrument will serve for from 4 to 6 determinations without changing the water in the warming jacket, but for accurate readings it is advantageous to change the water in the glass tube after every two tests. The platinum loops are readily cleansed by passing them through a Bunsen flame.

With this instrument the coagulation-time of normal blood is between 3 and 5 minutes.

J. WASSERMAN (NEISSER, BRUCK) TEST FOR SYPHILIS

Theory.—The serum diagnosis of syphilis is based upon the principle of hæmolysis and the Bordet-Gengou phenomenon (Fox).

Hæmolysis means the ability of the serum of one species of animal to dissolve the blood corpuscles of another species. This power can be greatly increased by injecting, several times, the corpuscles—e. g., of sheep, into a rabbit. As the result of introducing the sheep's corpuscles (an antigen) there forms in the rabbit's blood a *hæmolytic* amboceptor (an antibody). To produce hæmolysis a third substance, complement, is needed, and this is present in all normal fresh blood serums. "Amboceptor" means that this element possesses an affinity both for corpuscles and for complement. The latter is so named because it completes the action of the amboceptor. If the fresh serum of a rabbit thus immunized against sheep is placed in a test-tube and sheep's corpuscles added, the amboceptor which has formed in the rabbit's blood unites with the complement already present and with sheep's corpuscles, dissolving the latter. Hæmolysis is indicated by the change from an opaque or cloudy, to a clear or transparent, red colour. All three substances (a *hæmolytic system*)—(1) corpuscles, (2) hæmolytic amboceptor, and (3) complement—must be present or hæmolysis does not occur.

It is to be borne in mind that an antigen is any substance (corpuscles, tissue cells, bacteria) which, if injected into a living organism causes the formation of antibodies (amboceptors).

Under corresponding circumstances there may develop in the blood-serum a bacteriolytic amboceptor (antibody) which destroys bacteria, as the hæmolytic amboceptor destroys corpuscles. For its formation the antigen consists of bacteria, and complement is needed exactly as in hæmolysis. As an example of bacteriolysis one may employ (1) a culture of *B. typhosus* (as antigen), (2) the serum of a typhoid-fever patient in which has been formed a *bacteriolytic* amboceptor (antibody), and for complement (3) fresh guinea-pig serum. If these three substances are mixed in a test-tube at a proper temperature they will unite, and the bacilli will be dissolved in this "bacteriolytic system" just as the corpuscles were destroyed in the "hæmolytic system." But while in the latter there was an indicator (hæmoglobin) which showed by colour-changes whether or not the corpuscles were dissolved, it is not possible to ascertain from appearances whether the bacteria have been destroyed or remain intact.

Fortunately, it is possible to utilize two elements of a hæmolytic system in conjunction with the three elements of a bacteriolytic system to determine the presence or absence of amboceptors (antibodies) in the serum to be tested. This reaction (Bordet-Gengou) depends upon the fact that the complement actually unites with the other two elements of a bacteriolytic system. By way of illustration: The three components of a bacteriolytic system—e. g., (1) *B. typhosus*, (2) serum of patient (suspected of having typhoid fever) containing or not, as the case may be, the typhoid antibody (bacteriolytic amboceptor), and (3) complement—are placed in a test-tube and incubated for one half hour to allow union. To the tube are added (4) immunized rabbit serum (hæmolytic amboceptor), and (5) sheep's corpuscles. The mixture is again incubated. If now the suspected serum "(2)" contains typhoid antibodies, there will have been a union between "(1)," "(2)," and "(3)," completing the bacteriolytic system and reaction, and none of "(3)," the *complement*, will be left to unite with "(4)" and "(5)," and there will be no hæmolysis. The absence of hæmolysis (no change in colour) indicates the presence of the typhoid antibodies—i. e., the suspected patient has typhoid fever. Conversely, if no typhoid antibody is present in "(2)" with which the complement may unite, it joins "(4)" and "(5)," completing the hæmolytic system and destroying "(5)," as indicated by the colour change. The occurrence of hæmolysis thus demonstrates the absence of typhoid antibodies—i. e., the patient is not suffering from typhoid fever.

The principles involved in this reaction were applied to the diagnosis of syphilis (1906) by Wasserman, Neisser, and Bruck. An extract of a syphilitic liver, containing an abundance of the causative organism (*Spirochæta pallida*), was employed as antigen, taking the place of "(1)," and the serum of a suspected syphilitic patient was substituted for "(2)." Later work showed that a lipoid (fat-resembling) substance, lecithin, was able to combine with the syphilitic antibodies, and it is now used instead of the extract of syphilitic organism-containing tissue.

Noguchi's Simplified Test.—The technic of the Wasserman test proper, because of its elaborateness, is unfitted for general use. Therefore Hideyo Noguchi (Rockefeller Institute) has worked out a much easier, and even more accurate, modification. Slips of bibulous paper (5 mm. × 5 mm.) are impregnated with certain standardized components, and when dried will keep indefinitely. There are 3 of these: *amboceptor* slips (serum from immunized rabbit's blood), *complement* slips (fresh guinea-pig serum) and *antigen* slips (lecithen solution). At present they are not, but doubtless soon will be, manufactured by some good commercial biological laboratory. Having these, prepare a suspension of human blood corpuscles by adding 2 drops of such blood to 8 c. c. of physiological salt solution. Procure 10 drops of blood from a definitely syphilitic patient and allow it to clot and the serum to separate. Do the same with blood from a normal person, and also with the blood of the patient to be tested. Then proceed as follows:

Take 6 clean test-tubes (10 cm. × 1 cm.). Set them in pairs (Fig. 251 *b*). In each of the 1st pair put 1 drop (from a capillary pipette) of the serum to be tested; in each of the 2d pair, 1 drop of the syphilitic serum; in each of the 3d pair, 1 drop of the normal serum. The 2d and 3d pairs are controls. To *each* of the 6 tubes add 1 c. c. of the suspension of corpuscles, *and* 1 complement slip. Then into *1* tube of *each pair* put an antigen slip. Mix well by shaking. Incubate at 37° for 1 hour, *or* in vest pocket for same time, body heat serving for incubation. Label tubes and hold each pair together by rubber band. There can be no possibility of confusion between the 2 forming a pair, as one contains 1 more slip (antigen) than the other.

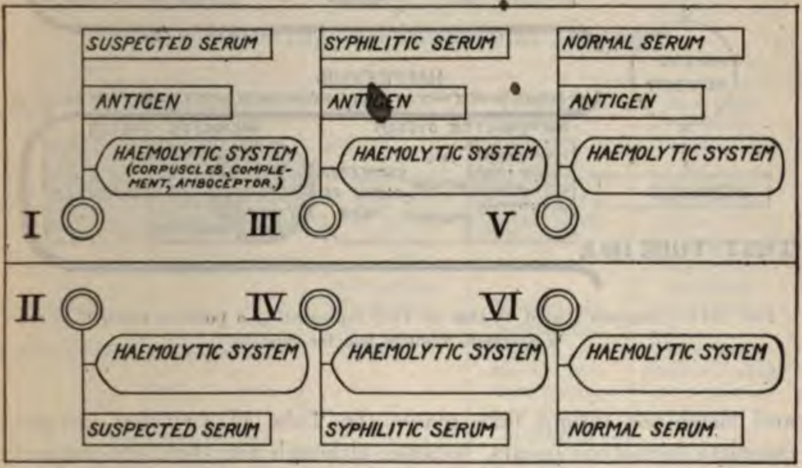


FIG. 251 *b*.—Showing arrangement and contents of test tubes I to VI in making the Wasserman-Noguchi test.

After incubating, add to each tube 1 hæmolytic amboceptor slip. Shake well, incubate again for 2 hours, and set aside at room temperature. Inspect from time to time for 10 or 12 hours.

Reading.—A *positive* reaction (Fig. 251 *c*) is indicated by *absence of hæmolysis* in Tube I (with antigen), for if a syphilitic antibody (amboceptor) is present it will unite with the antigen (lecithin) and the complement. The available complement being thus fixed, there is none to combine with the rabbit amboceptor and human corpuscles, and hæmolysis cannot take place. In Tube II (without antigen) complete hæmolysis occurs, because, although the syphilitic antibody is present, there is no antigen wherewith it may combine, the complement is not bound, but is left free to unite with the rabbit amboceptor and the human corpuscles, with consequent solution of the latter.

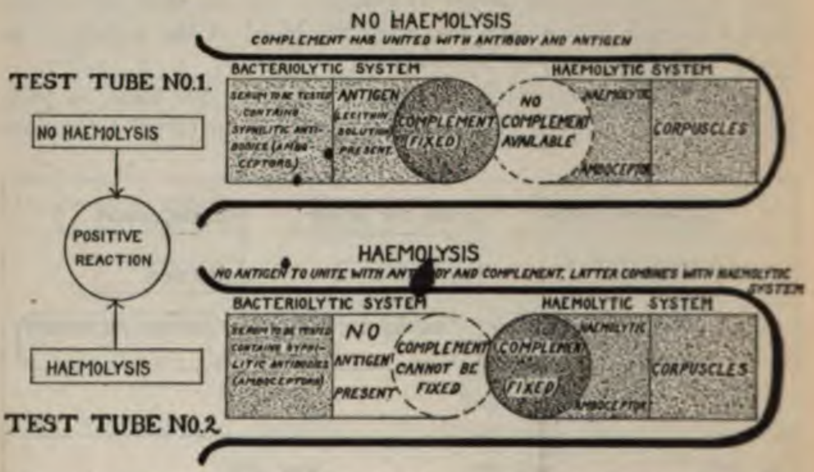

NO HAEMOLYSIS
COMPLEMENT HAS UNITED WITH ANTIBODY AND ANTIGEN
TEST TUBE NO.1.
BACTERIOLYTIC SYSTEM
HAEMOLYTIC SYSTEM
SERUM TO BE TESTED CONTAINS SYPHILITIC ANTIBODIES (AMBOCEPTORS)
ANTIGEN LECITHIN SOLUTION PRESENT
COMPLEMENT (FIXED)
NO COMPLEMENT AVAILABLE
HAEMOLYTIC
AMBOCEPTOR
CORPUSCLES
NO HAEMOLYSIS
POSITIVE REACTION
HAEMOLYSIS
HAEMOLYSIS
NO ANTIGEN TO UNITE WITH ANTIBODY AND COMPLEMENT. LATTER COMBINES WITH HAEMOLYTIC SYSTEM
BACTERIOLYTIC SYSTEM
HAEMOLYTIC SYSTEM
SERUM TO BE TESTED CONTAINS SYPHILITIC ANTIBODIES (AMBOCEPTORS)
NO ANTIGEN PRESENT
COMPLEMENT CANNOT BE FIXED
COMPLEMENT (FIXED)
HAEMOLYTIC
AMBOCEPTOR
CORPUSCLES
TEST TUBE NO.2

FIG. 251 *c*.—Diagram (based on that of Fox) representing a positive reaction to the Wasserman-Noguchi test for syphilis.

A *negative reaction*, explained by similar reasoning, is indicated by complete hæmolysis in both tubes (I and II).

There should, of course, be a positive reaction in pair III and IV, and a negative reaction (within 1 hour) in pair V and VI. A final decision as to the reaction must be postponed until all the tubes without antigen (II, IV, and VI) have undergone complete hæmolysis.

This test may be applied to the cerebrospinal fluid as well as to the blood-serum.

Diagnostic Value.—The value of this test for syphilis—recent, latent, or tertiary—is very great. It is also probable that it will constitute a most important criterion in the treatment of syphilis, the reaction becoming negative when the disease is cured.

TABULATION OF THE RESULTS OF HÆMANALYSIS IN CERTAIN DISEASES OF THE BLOOD

The following table presents in brief compass the salient features of the blood examination in the principal diseases of the blood. It is to be remembered that the table refers to the average or typical examples, and that considerable variations may exist in the individual case.

Blood Examination in Special Diseases

	Normal blood. For comparison.	Chlorosis.	Pernicious anæmia.	Secondary Anæmia.
RED CELLS	4,500,000 to 5,000,000.	Average 4,000,000, and rarely under 2,000,000. Poikilocytosis. Many microcytes.	Average 1,000,000. Microcytes. Megalocytes.	Moderate or marked decrease. Poikilocytes and microcytes common.
Normoblasts...	....	Occasional.	Moderately numerous.	Common.
Megaloblasts...	....	Rare.	Present, constantly predominating over the normoblasts.	Rare, never predominating over normoblasts.
Hæmoglobin...	90 to 95 per cent.	40 per cent. Always relatively low.	Often relatively high.	Relatively low.
WHITE CELLS..	5,000 to 10,000.	Normal or slightly increased. Average 8,000.	Usually decreased.	Usually increased.
Small lymphocytes	20 to 30 per cent.	Increased.	Increased.	Usually diminished.
Large lymphocytes	5 to 6 per cent.	Increased.	Increased.	Usually diminished.
Polymorphonuclear.....	60 to 75 per cent.	Decreased.	Decreased.	Usually increased.
Eosinophiles...	2 to 4 per cent.	Increased.	Often increased.	Vary.
Myelocytes....	....	Rare.	Neutrophilic myelocytes common.	Rare.

Blood Examination in Special Diseases (continued)

	Pathological leucocytosis.	Myelocytæmia or spleno-myelogenous leucæmia.	Lymphæmia or lymphatic leucæmia.	Leucæmia in infancy (Cabot).
Red Cells....	Normal or decreased.	3,000,000, rarely under 2,000,000.	3,000,000 or somewhat less.	May be under 2,000,000.
Normoblasts...	....	Numerous.	Comparatively rare.	Common.
Megaloblasts...	....	Moderately numerous.	Comparatively rare.	Common.
Hæmoglobin...	Normal or decreased.	Averages 50 per cent. Relatively low.	Averages 25 per cent. Relatively low.	Relatively low.
White Cells..	Usually not over 50,000.	Averages 100,000 to 300,000	Average 100,000 or less.	Usually more increased than in any other disease.
Small lymphocytes ...... Large lymphocytes	Diminished, averaging together about 10 per cent.	Diminished, averaging together about 7.5 per cent.	90 per cent, either small or large forms predominating.	Usually increased.
Polymorphonuclear.....	90 per cent.	49 per cent.	Average only 3 per cent.	Usually diminished.
Eosinophiles...	Decreased.	Usually increased.	Scanty. Average 0.7 per cent.	
Myelocytes	....	35 per cent. (characteristic) (Neutrophilic).	Scanty. Average 0.3.	Usually more numerous than in other diseases.

SECTION XXXIX

MICROSCOPICAL EXAMINATION OF THE SPUTUM

The evidence to be derived from gross or macroscopic inspection of the sputum has already been considered (page 276). With the microscope one searches for cells, elastic fibres, casts, spirals, crystals, and parasitic organisms.

Preparation of the Sputum.—It is desirable first to examine a fresh unstained specimen and afterward to make a dried and stained cover-glass preparation.

(1) A *preliminary careful inspection*, either by the unaided eye or by a hand lens, is useful, and is most conveniently made by having two pieces of window glass, one 5 or 6 inches square, the other an

inch or two less in size, the larger piece painted black upon its under surface, or, when in use, laid upon a black ground. A portion of the sputum is placed upon the larger piece and covered by the smaller. Against the black background, cheesy particles, casts, elastic fibres, and spirals may be detected. Yellowish particles are probably caseous or cheesy masses in which tubercle bacilli and elastic fibres are most likely to be found; while grayish-yellow spots may be elastic tissue; sago-like grains, Curschmann's spirals; and whitish, yellowish-brown, or reddish-yellow masses, fibrinous casts. By sliding the upper glass to one side particular objects may be removed for identification and detailed examination by the microscope. A microscope slide or a watch glass placed upon a piece of black paper will serve almost as good a purpose if the sputum is thoroughly spread out. Rarely there are found in the sputum concretions, sometimes of considerable size, which have been formed in vomicæ and bronchiectases, or consist of portions of calcified bronchial glands which have entered the lungs and been expectorated.

(2) The *fresh sputum is examined* by picking out a promising particle, placing it on a slide, and covering. The cover glass should be moved about with slight pressure so as to flatten out the specimen into a layer of sufficient thinness. Examine first with a low, then with a high, power.

(3) *Dried preparations* are made either by putting a suspicious-looking portion of the sputum upon a cover glass and spreading it over the glass by a needle or small spatula; or by placing the chosen specimen between two cover glasses, pressing them gently together to diffuse the material, and then sliding the covers apart as in preparing a blood film (page 614). In either case they must be allowed to dry in the air, after which it is usually necessary to fix the layer of sputum by heat, preparatory to staining. This is best done by passing the cover glass, held in forceps, three times through an alcohol flame, film side up, each passage occupying something less than a second, thereby coagulating the albumin and securing the adherence of the film. Appropriate methods of staining are described in connection with the special end desired.

Methods and Results of the Microscopic Examination.—(*a*) **Red Blood Cells.**—According to the length of time that these cells have been out of the vessel so will they vary in appearance, from the normal to pale, shadowy, or fragmentary forms. A few red corpuscles are of no diagnostic significance, as they may be found in many sputa, particularly if there is a catarrhal condition of the respiratory mucous membrane. They necessarily occur in large numbers in hæmoptysis, but, aside from this, they appear constantly and more or less abundantly in all pulmonary diseases, especially phthisis. The green and yellow sputum of resolving pneumonia owes its colour to altered hæmoglobin, the corpuscles having been destroyed so that they can not be seen.

(*b*) **White Cells.**—Polymorphonuclear leucocytes (pus cells) in various stages of degeneration, granular, fatty, or pigmented, are found in all sputa, especially from a putrid bronchitis, or a perforating empyema, subphrenic abscess, or other collection of pus.

Eosinophilic leucocytes (*q. v.*) may be found in large numbers, often associated with Charcot-Leyden crystals, in the expectoration of bronchial asthma.

(*c*) **Epithelium.**—Epithelial cells from the mouth, tracheal tree, and alveoli are found in the sputum, but their shape is so much altered at the time of examination that no correct inference as to the seat of the abnormal process can be derived from their characters. A round pigmented (hæmatoidin) alveolar epithelial cell is quite characteristic of brown induration of the lungs due to the chronic pulmonary congestion of cardiac valvular disease.

(*d*) **Elastic Fibres.**—To detect particles and shreds of elastic tissue, suspicious lumps in the sputum may be mixed with a 10-per-cent solution of sodium hydrate and a considerable quantity examined under a low power; or the entire amount of sputum may be boiled for a few moments with an equal volume of the sodium hydrate solution. The resulting gelatinous mixture should be diluted with three or four times its volume of water, and either centrifugalized or allowed to stand until settled. The elastic fibres, which resist the alkali except after prolonged boiling, will be found in the sediment. The fibres are undulating, double contoured, and the ends are usually curled. Not infrequently they present an alveolar arrangement (Fig. 252), and if this is present it may be safely affirmed that they originated in the lungs and are not derived from particles of flesh food in the sputum.

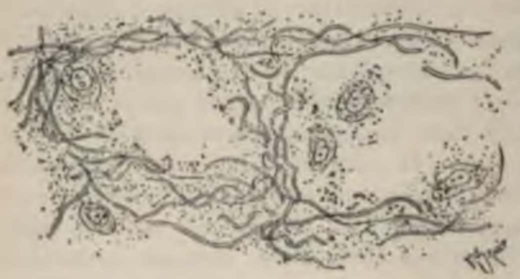


Fig. 252.—Elastic tissue from lung in sputum of a case of phthisis. × 300. (Hutchison and Rainy.)

When elastic fibres are found, it may be considered indubitable proof of a destructive process in the lung—e. g., phthisis, abscess, or bronchiectasis. In many cases of gangrene of the lung the elastic tissue is destroyed (perhaps by a ferment) so that its absence in suspected cases does not disprove the existence of a necrotic process.

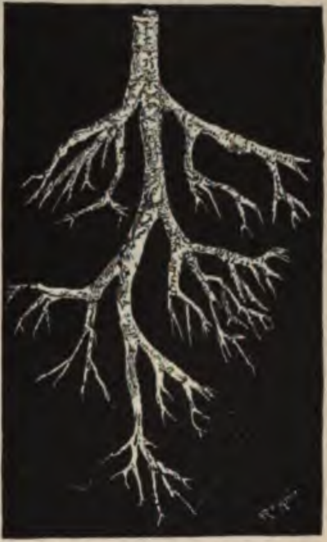


FIG. 253.—Bronchial cast from a case of plastic bronchitis. Natural size. (Hutchison and Rainy.)

(*e*) **Fibrinous Casts.**—These casts (Fig. 253) vary in size from those which constitute a mould of the trachea and larger bronchi—4 or 5 inches in length and of corresponding thickness—to those which are formed in the bronchioles, ½ to 1½ inches long and of threadlike thickness. While casts large enough to attract attention in the naked-eye inspection occur not infrequently, the majority require a low power of the microscope to render them visible. The casts of macroscopic size may be unravelled under water. They are found to branch dichotomously; the slenderer branches are solid and the larger are apt to be hollow.

Fibrin casts are found in three diseases: the largest in diphtheria, the medium sized in fibrinous bronchitis, and the smallest in lobar pneumonia, either just before or immediately after the stage of resolution—i. e., the third to the ninth day.

(*f*) **Curschmann's Spirals.**—These consist of a central core or thread (perhaps fibrinous) which has an undulating course (Fig. 254). Around this central thread are coiled in a spiral manner fibrillary bands (probably tough mucus). Entangled in the spirals are eosinophiles, and frequently also Charcot-Leyden crystals.

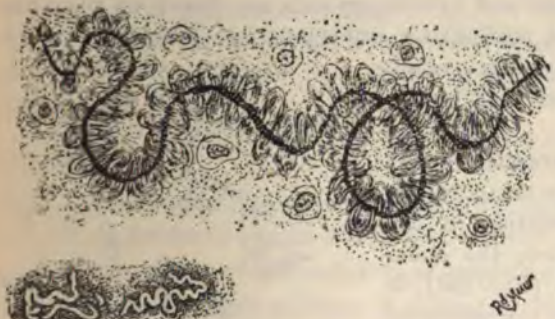


FIG. 254.—Curschmann's spirals in sputum. × 200, and natural size. (Hutchison and Rainy.)

Curschmann's spirals are found especially in the sputum of bronchial asthma, much more rarely in phthisis, bronchitis, and lobar pneumonia. Their presence may be of service in distinguishing between bronchial and the so-called cardiac or renal asthma.

(*g*) **Crystals.**—(1) *Charcot-Leyden crystals* (Fig. 255) (probably spermin phosphate) are fine, colourless, sharply pointed octahedra of varying size, often requiring a high power to make them visible. They are found for the most part in asthma, usually associated with the spirals just described, but occur as a rarity in bronchitis, phthisis, and chronic pneumonia.

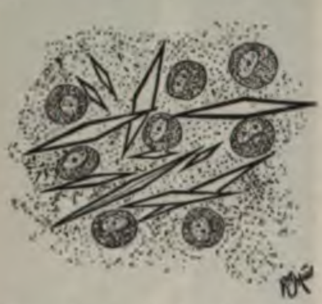


FIG. 255.—Charcot-Leyden crystals (Hutchison and Rainy).

(2) *Cholesterin crystals* are but rarely seen in the sputum, occurring as transparent, colourless rhomboidal plates, with notched and irregular angles and ends. They may be found in old purulent sputum from phthisical or pulmonary abscess cavities, or where old pus accumulations have perforated into the bronchial tubes.

(3) *Hæmatoidin crystals*, derived from hæmoglobin, occur as needles or rhombi of a red or brownish-yellow colour. They are found where there is an old extravasation of blood into the lungs, or where perforation of an empyema or other abscess has taken place.

(4) *Fatty acid crystals*, fine long needles, generally bunched or clustered, occur frequently in putrid bronchitis, pulmonary gangrene, phthisis, bronchiectasis, old sputum, and follicular plugs of the tonsils; consequently their diagnostic value is slight.

(5) *Leucin globules* and tyrosin crystals are found in the purulent sputum from old perforating empyemas and putrid bronchitis. Rarely crystals of calcium oxalate and triple phosphates may be encountered.

(*h*) **Animal Parasites.** — (1) *Echinococcus.*—In the comparatively rare cases of hydatids of the lung or near-by organs, the hooklets of the organism (Fig. 256), or more frequently the fragments of the cyst wall, may be encountered in the sputum, rupture into a bronchus having occurred.

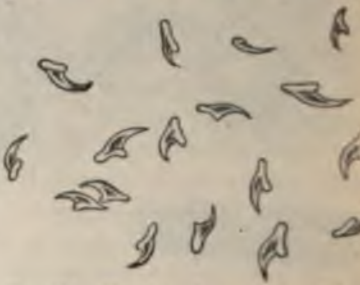


FIG. 256.—Hooklets from tænia echinococcus. × 350.

(2) *Distoma pulmonale.*—This worm when lodged in the lung causes pulmonary hæmorrhage which may be mistaken for that of

phthisis. It is common in China and Japan, but is extremely rare in this country. Its presence is betrayed by the finding in the sputum of its ova, which are brown, oval bodies, 0.1 millimetre long and 0.05 millimetre wide (see Stools, microscopic examination).

(3) *Amœba coli.*—This organism (*q. v.*) when found in the sputum (a very rare occurrence) is proof positive of the perforation into the lung of an hepatic abscess.

(*i*) **Vegetable Parasites.**—(1) *Actinomyces.*—Pulmonary actinomycosis is of infrequent occurrence in this country. I have reported a case terminating in recovery under the use of oil of eucalyptus (Med. News, April 29, 1898). The organism is found in the sputum as sulphur-coloured granules from 0.5 to 2 millimetres in diameter. If a granule is placed under a cover glass and slight pressure applied, it will flatten and be seen under the microscope to consist of club-shaped rods or threads radiating from a common centre (Fig. 257).

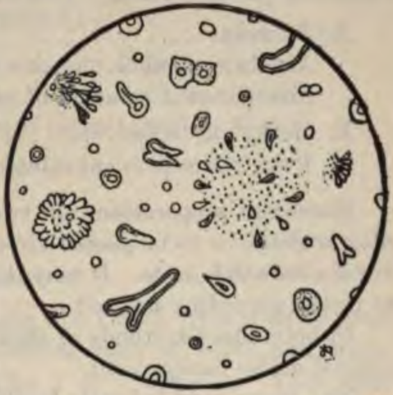


FIG. 257.—Actinomyces in sputum (Hutchison and Rainy).

(2) *Bacillus tuberculosis.*—This is by far the most important organism—clinically, at least—to be found in the sputum. To find it, a dried preparation is made, picking out, if possible, for this purpose, one of the small caseous or cheesy masses usually discovered by careful inspection of the mass of sputum when spread out against a black background. The dried and fixed specimen is to be stained by one of the following methods:

Ziehl-Neelsen Method.—This requires the use of the following solutions:

A. Concentrated alcoholic solution of fuchsin.... 10 c. c.
Five-per-cent aqueous solution of carbolic acid. 90 "

B. Twenty-five-per-cent solution of sulphuric acid.

C. Saturated watery solution of methylene blue.

A sufficient quantity of solution *A* is put in a watch glass, the coated cover glass immersed in it, and gentle heat applied until steam rises. It is best to remove and reheat the solution several times, taking three or four minutes in the process. A more convenient method is to put a few drops of the stain upon the cover glass, which is then held by forceps over the flame, heating and reheating it, as with the watch glass. Wash the cover glass in water to remove the excess of staining fluid, and then place it in solution *B*. The film, which has been stained red, will be decolourized by this solution, the tubercle bacilli alone retaining the colour. The cover glass should be kept in motion to facilitate the bleaching process. Wash in water or 50-per-cent alcohol, and if a contrast staining (i. e., of all elements except the bacilli) is desired, the cover glass should be immersed for one minute in solution *C*. Wash again in water, dry, and mount in a drop of water for examination. To preserve permanently, mount in Canada balsam.

Gabbett's Method.—This is perhaps a still more convenient method than that just described. Two solutions are required:

A.	Fuchsin	1 grme.
	Absolute alcohol	10 c. c.
	Five-per-cent solution of carbolic acid	100 "
B.	Methylene blue	2 grmes.
	Twenty-five-per-cent solution of sulphuric acid,	100 c. c.

Place the preparation for two or three minutes in solution *A*, after which it is to be passed directly into solution *B*, where it should remain for one minute. It may then be washed in water and mounted as in the preceding method.

In both these methods of staining the bacilli appear as *red* rods against a *blue* background.

The presence of tubercle bacilli in the sputum is positive evidence of pulmonary phthisis; their absence does not disprove the existence of this disease unless repeated examinations have given a negative result, as they may be absent at one time and present at another. If the symptoms and signs point toward a phthisical process, and yet the bacilli, presumably because of their scanty number, are not found by the ordinary methods, resort may be had to one or both of the following devices: *First*, the sputum may be stirred with a glass rod in a glass or porcelain capsule until smooth and diffluent, and one or two centrifuge tubes filled by means of a medicine dropper with the thinned material and centrifugalized, a portion of the sediment being prepared and stained as usual; or, *second*, a considerable quantity (2 or 3 ounces) of sputum may be mingled with an equal quantity of water, to which 6 or 8 drops of a 10-per-cent sodium hydrate solution has been added, and the resulting mixture boiled until homogeneous, after which it may either be allowed to settle for one or two days, or centrifugalized. A portion of the sediment is then examined for the bacilli and elastic tissue.

(3) *Influenza bacillus.*—This organism (PFEIFFER'S) may be stained by Loeffler's solution, which is:

Concentrated alcoholic solution of methylene blue	3 c. c.
One-tenth-per-cent aqueous solution of potassium hydrate,	10 c. c.

This should be freshly mixed when needed. Stain the dried and fixed cover-slip preparations for from 5 to 10 minutes, wash in water, and mount in water or balsam. The organisms are coloured blue, and appear as extremely small rods. Their ends stain more readily than the middle portion, and although for this reason they resemble diplococci, they have no capsule. They are found in great abundance in the bronchial secretion.

(4) *Diplococcus pneumoniæ.*—This is best demonstrated by the following modification of Gram's method:

A. Saturated (filtered) alcoholic solution of gentian violet	1 c. c.
Five-per-cent aqueous solution of carbolic acid,	9 c. c.

A should be prepared when needed.

B. Iodine	1 grme.
Potassium iodide	2 grmes.
Distilled water	300 c. c.

Place the dried and fixed cover-slip film (made from a bit of rusty sputum) in solution *A* for 3 or 4 minutes, wash in water, and transfer to solution *B*, letting it remain for 1 or 2 minutes. Then wash in alcohol until the darkly stained film is decolourized. After this put it first in absolute alcohol, then in oil of cloves, and finally mount in balsam. The organism is seen as a dark blue or violet diplococcus. Welch's method is said to be the best for differentiating the characteristic capsule of the organism. A few drops of glacial acetic acid are placed upon the spread and dried cover slip and allowed to remain a minute or two. Then, without washing, the film is treated repeatedly with solution *A*, until it is judged that the acid is removed, after which the specimen is washed with, and mounted in, a 2-per-cent salt solution.

The absence of this coccus in the sputum negatives the existence of a croupous pneumonia; its presence is not altogether conclusive, as it occurs in the sputum of a certain proportion of healthy persons.

SECTION XL

EXAMINATION OF THE STOMACH CONTENTS

THE gross or macroscopic characters of the vomited contents of the stomach have been previously considered (page 123); the method of obtaining the gastric contents by means of the stomach tube has also been described in connection with the physical examination of the stomach (page 488).

I. PHYSIOLOGY OF DIGESTION

Prior to dealing with the chemical and microscopic examination of the stomach contents a brief statement of certain facts in the physiology of gastric digestion may be serviceable.

Stomach digestion is carried on by means of a mineral acid, a proteolytic enzyme (or ferment), and a coagulating enzyme—i. e., hydrochloric acid, pepsin, and rennin, which constitute the active agents of the gastric juice. Practically the gastric juice affects only the proteids (e. g., meat) or the albuminoids (e. g., gelatin) of the food. The gastric juice is secreted in small quantities even when the stomach does not contain food, but the presence of food acts as a prompt and effective stimulus to an abundant formation. Fats inhibit gastric secretion to a degree; starches exert a moderate stimulating action, the juice secreted being of relatively low acidity; proteids exert the most stimulating effect both as to quantity and strength of the juice.

The secretion of gastric juice is apparently the result of nervous impulses which pass to the stomach from the higher centres; these impulses seemingly arise as a result of seeing, smelling, and tasting food. Pawlow has spoken of this as the "psychic juice." The presence of food substances in the stomach later brings about a flow of gastric juice which is presumably the result of chemical stimulation. The meat extractives and peptones seem to be particularly efficacious in such stimulation. These are important considerations in dietetics, and explain the general use of meat soups before meals.

The acid of the juice which has been secreted combines with the gelatins, proteins, and mineral salts of the food. As soon as these affinities are satisfied, free acid begins to appear on the outside of the food mass.

Under the influence of the free acid the zymogens (pepsinogen and chymosinogen) secreted by the gastric glands are transformed into pepsin and rennin. The only action of the rennin is to coagulate the casein of milk. The hydrochloric acid and pepsin act together, changing the proteids and the curdled casein, first into albumoses (proteoses), finally into peptones.

II. THE CHEMICAL EXAMINATION OF THE GASTRIC CONTENTS, AND THE DETERMINATION OF THE MOTOR POWER OF THE STOMACH

At the height of digestion in a normal stomach analysis will show the presence of free hydrochloric acid, acid salts, pepsin, rennin, albumoses, and peptones, besides maltose and its related substances, achroödextrin and erythrodextrin. The object of the examination of the gastric juice is to determine the presence and in some cases the amount of certain of these constituents; and, furthermore, to ascertain whether certain other substances are present which normally are not produced by the stomach. The principal substances which should not be found, except as they or their salts have been introduced as a part of the ingested food, are lactic, acetic, and butyric acids. These acids are formed in large quantities under certain pathological conditions which favour fermentation of the ingested food. The motor power of the stomach—i. e., its churning power and its ability to expel the products of gastric digestion (chyme) through the pyloric orifice—is also to be determined; and some information is to be obtained from a microscopical examination of the material obtained by the stomach tube.

A. TEST MEALS

The gastric juice is usually not secreted in sufficient quantity for analytical purposes unless the stomach contains food. The amount and time of appearance of the various ingredients of the secretion will vary normally within certain limits according to the quantity and quality of the food taken. Consequently for purposes of analysis and comparison it is necessary to give a definite quantity and quality of food, and to withdraw the contents of the stomach at a definite period of digestion. Hence the utility of the various test meals which have been devised. Of these the three following are the most useful. The first mentioned is usually all-sufficient.

Ewald's Test Breakfast.—This consists of one or two baker's rolls or one or two slices of dry bread (35 to 70 grammes, a little over 1 to 2 ounces), and 300 to 400 cubic centimetres (roughly, 9 to 12 ounces) of water or weak tea. The amount of lactic acid contained in this quantity of bread is so small that it does not vitiate the examination in ordinary cases. It is taken in the morning, no food having been ingested since the previous evening. Between an hour and an hour and a half after the meal the contents of the stomach are to be withdrawn.

Boas's Test Breakfast.—This meal is employed in cases in which it is of great importance to eliminate any possibility of the introduction of lactic-acid-forming substances as part of the food—e. g., in suspected gastric cancer. It is necessary to thoroughly wash out the stomach, either on the night previous, or at least one hour prior to giving the meal. The meal consists simply of oatmeal soup prepared by adding a tablespoonful of oatmeal to one quart of water and boiling it down to one pint. Nothing is to be added except a little salt. The gastric contents are to be removed an hour or an hour and a half after.

Von Leube and Riegel Test Dinner.—This comprises 400 cubic centimetres (12 to 14 ounces) of soup, a slice or two—50 grammes (about 2 ounces)—of wheat bread, 100 to 200 grammes (3 to 6 ounces) of chopped or minced beefsteak, and 200 cubic centimetres (6 ounces) of water. The gastric contents are to be withdrawn four hours after. Important information regarding the activity of both the muscular and chemical functions of the stomach may often be obtained by having the patient eat a hearty meal comprising meat, vegetables, and dessert at night, removing the residue for inspection in the morning, and, after lavage, administering Ewald's breakfast.

The fluid obtained from the stomach is either allowed to settle in a tall glass jar, the supernatant portion being taken as required, or, better, is filtered through paper or fine muslin.

B. THE CHEMICAL TESTS AND THEIR TECHNIC

The general practitioner is often deterred from a chemical examination of the stomach contents by an exaggerated idea of the difficulties of the processes and the elaborateness of the required apparatus. While it is true that some of the methods which may be employed require certain resources commonly found only in a laboratory, it is also true that by a choice of procedures the necessary clinical investigations can be made with comparatively simple means.

A simple and satisfactory routine of examination is as follows:

The extracted chyme is placed in a conical glass and allowed to settle for a half hour, preparatory to a gross examination. There is usually a tendency to settle in three layers, which vary in thickness according to the conditions present. The upper layer is seen to be mucus, perhaps in lumps, frequently of a dirty appearance and suggesting a pharyngeal origin. More commonly it is a smooth glairy mucus, the exact origin of which is later to be determined. Underneath this is normally a layer of turbid fluid, whitish or greenish yellow in color. On the bottom is a layer composed of the residues of ingested food. These food particles are coarser when the muscular and secretory power is low and there is an excessive secretion of mucus; they are finer when the converse is true. In looking at these particles of food residue an idea is at once obtained as to the state of the secretions.

The greater the proportion of clear fluid the greater is the secretory power; in hypersecretion there is little else but this clear layer of fluid. Frequently there is no such separation, and on inspection we see that bread particles are held together by mucus. This appearance is evidence of an actual increase of mucus, due either to a secretory neurosis or a gastric catarrh.

Flecks of blood are to be looked for in the mucus. The faintest tinge of blood colour throughout the fluid is to be investigated with care by chemical tests. A yellowish colouration which changes to greenish on exposure to air is due to regurgitation of bile. Slight evidence of this is of no particular importance, but if very pronounced it is suggestive of stenosis of the duodenum below the papilla of Vater.

After such an inspection the stomach contents are filtered through a small moist filter paper of coarse texture and the filtrate is examined as follows:

To Test for Free HCl.—*Qualitative.*—Five cubic centimetres of the filtrate are measured with a pipette and poured into a large beaker or Ehrlenmeyer flask. A few drops of dimethyl-amido-azobenzol (Topfer) in a 0.5 per cent alcoholic solution are then added to this measured filtrate.

If free HCl is present the filtrate is turned a beautiful cherry red. The intensity is proportionate to the strength of free acidity. If there is no actual change of colour, except that imparted to the filtrate by the "dimethyl," it may be assumed that there is no free acidity. If there is a doubtful change of colour to an orange tint there is probably no free acid, but it may be well to make sure by using the more complicated but most delicate and conclusive phloroglucin-vanillin (Günzberg's) test, as follows:

The solution employed is

Phloroglucin	2 grmes. (30 grs.)
Vanillin	1 grme. (15 grs.)
Absolute alcohol	30 c. c. (1 oz.)

This solution is yellow, and, as the colour changes on exposure to light, it should be kept in a dark bottle. The first two ingredients are expensive. Place a few drops of filtrate in a porcelain dish, ad a similar quantity of the reagent, and hold some distance above a alcohol flame. The process of evaporation should be slow and gentl If free hydrochloric acid is present, pink or rose-coloured margina lines will be seen as the last of the fluid dries off. A brownish tin indicates either overheating or the absence of free hydrochloric acid.

Quantitative.—If free acid was found with the "dimethyl," cubic centimetres of the filtrate in the beaker or flask is examine quantitatively for free HCl as follows:

A burette, fitted with a suitable dropping stopcock and havin clear markings of the tenths of cubic centimetres, is filled with sodi um hydrate solution of the strength known as "decinormal." This i added drop by drop to the filtrate; between each drop thorough mix ing is assured by stirring with a glass rod or by thorough agitatio if a flask has been employed. As each drop falls it tends to mak a decolourized zone in the reddish fluid. The dropping is continue until the red disappears, and even the orange tint is not pronounced This "end reaction" shows that there is no more free HCl. Th number of centimetres and fractions thereof required to bring abou this neutralization of the free HCl is then noted.

This number is multiplied by 20 and represents the number o cubic centimetres of decinormal alkali it would have taken to neutral ize the acid in 100 cubic centimetres of filtered chyme. If more o less than 5 cubic centimetres of filtrate was used the proper numer ator must be employed to bring the figures up to 100. If, for ex ample, 2.2 cubic centimetres of alkali were used, 5 cubic centimetre of the filtrate having been treated, 44 cubic centimetres would hav been required for 100 cubic centimetres of filtrate. At the presen time it is customary to designate the free and the total acidity o the stomach contents directly by the number of cubic centimetres o alkali needed to neutralize 100 cubic centimetres of chyme filtrat and not, as formerly, by a percentage figuring. This result is terme the degree of acidity. It is expressed, for example: Free HCl, 40 total acidity, 70.

To Determine the Total Acidity.—Having learned the amount o free HCl, another portion of the filtrate is measured off, or the sam portion which was used for the free HCl determination is employed for ascertaining the total acidity of the filtrate. The total acidity i made up of the free HCl, the HCl which is loosely combined wit albuminous bodies, the acid salts, and any organic acids which may b present.

Test for Total Acidity.—To the measured filtrate add a few drop of a 1-per-cent alcoholic solution of phenolphthalein, drop by dro from a pipette. Each drop causes a reddish colour which quickly disappears on agitation.

Gradually the colour tends to persist, and finally a pinkish colour appears and remains. The end reaction is not reached until the spot where the indicator drops does not turn redder. A reading is then taken on the burette to ascertain just how many cubic centimetres of alkali have been needed. The proper numerator is then used to bring this up to the amount of alkali which would have been used had 100 cubic centimetres of filtrate been taken. Thus, for example, 4 cubic centimetres of alkali having been needed for 5 cubic centimetres of stomach contents, the total acidity is 80.

To Test for Combined Acid.—In case no free acid has been found it is desirable to discover whether in fact there has been some secreted which is held in combination with proteins. This "loosely combined acid" is tested for as follows:

To 5 cubic centimetres of the filtrate several drops of a 1-per-cent aqueous solution of alizarin is added. This gives a brownish colour to the filtrate. Decinormal soda is added drop by drop until a violet colour is reached. The final colour should be previously determined by adding alizarin to a distinctly alkaline fluid. It is important to get the real end colouration. For this purpose hold the beaker over a white paper, and look carefully at the effect of each drop of alkali as it strikes the filtrate. This end reaction is reached when the alkali has neutralized all acidity of the filtrate except that due to loosely combined HCl. If then we multiply by the proper numerator to make the total come up to that which would result had we used 100 cubic centimetres of filtrate, and subtract the figure thus obtained from that of the test for total acidity we have an idea as to the presence and amount of combined hydrochloric acid. We may discover that some acid has been secreted, but not more than enough to supply the affinities of the proteins present. It frequently happens that the figure for combined acid is so low that no conclusions can be drawn.

The amount of free organic acids and the acid salts can be estimated by deducting from the total acidity the amount of free and loosely combined acids; the remainder represents the organic acids and the acid salts. It is more satisfactory to reach conclusions on the subject of the presence and amount of organic acids by the tests yet to be described.

If there is no free acid whatever as shown by the tests, it is a matter of considerable importance to determine the presence of the organic acids. If the dimethyl test is doubtful, or if the free HCl is found to be as low as 20 or less, then, also, it is well to test for organic acids. If free HCl is present in normal, or more than normal, amount, these tests are unnecessary, as it is known that under these conditions they will be negative or so faint as to be of no importance.

To Test for Free Lactic Acid.—(1) *Uffelmann's Test.*—Place 10 cubic centimetres of the filtered gastric fluid in a large test tube or a separation funnel and add 50 cubic centimetres of ether. Shake thoroughly for several minutes and pour off the ether. The separated ether may be divided into two portions, one to be tested for lactic acid, the other (if required) to be subsequently employed for the detection of acetic and butyric acids. The portion to be tested for lactic acid should be put in a test-tube or shallow dish and set in hot water. The other portion, in a similar container, is allowed to evaporate spontaneously.

The residue of the first portion is to be dissolved in about 5 cubic centimetres of water. Test this with Uffelmann's reagent, which should be freshly made by placing in a test-tube 3 drops of liquefied carbolic acid and 3 drops of a saturated aqueous solution of the sesquichloride of iron. This is to be diluted with water until a pale amethyst-blue tint is secured. To the bluish solution add a small portion of the ethereal extract, when, if lactic acid is present, the blue tint changes to a canary yellow.

(2) *Kelling's Test.*—Place in a test-tube 5 cubic centimetres of the gastric fluid and add ten times its bulk of water. Treat the diluted fluid with 1 or 2 drops of a 5-per-cent watery solution of the sesquichloride of iron. If, upon looking at the tube against a white background, the fluid is distinctly green, lactic acid is present.

To Determine the Presence of Acetic and Butyric Acids.—Take the residue left by the slow evaporation of the second portion of the ethereal extract and dissolve it in about 5 cubic centimetres of water. Evaporation without heat is advisable in order to avoid driving off the volatile acetic and butyric acids.

(1) **Test for Acetic Acid.**—It may often be recognised by its odour. To demonstrate its presence, take a portion of the watery solution of the residue and exactly neutralize it with a little dilute sodium-hydrate solution or small pinches of powdered sodium carbonate. The exact point at which neutralization is complete is best ascertained by placing from time to time 1 drop of the fluid upon red and another upon blue litmus paper until no change of colour occurs in either case. Add 2 drops of a very dilute solution of perchloride of iron. If acetic acid is present, a claret or dark-red colour results.

(2) **Test for Butyric Acid.**—This may be often recognised by its rancid odour, but its presence may be demonstrated by adding to

the remaining portion of the dissolved ethereal residue a small fragment of calcium chloride. If butyric acid is present it will separate out in small oily drops, which possess the characteristic strong odour.

If any butter is on the bread which is taken as a test breakfast, or if it is not the first meal of the day, or if the meal be a Riegel dinner with a fatty meat, this test for butyric acid may be made worthless by the presence of butyric acids in the food.

To Test for Rennin and Pepsin.—*If a test breakfast shows no free acid whatever, or a total acidity below 20 degrees,* it is a matter of importance to make quantitative tests for ferments. It is, as a rule, unnecessary to test for the ferments when the acid secretion is at or above the normal. It is, however, a fact, that no acid may be secreted under certain conditions, but the ferments still be discoverable. Their continued presence and strength give diagnostic indications of value. Qualitative tests alone are of greatly restricted value. Pepsin secretion seems the last to be lost.

Rennin Estimation.—Rennet itself is not secreted by the stomach mucosa, but its precursor, or zymogen, which becomes activated if free acid is present. To estimate the strength of rennet zymogen, proceed as follows: Take 1 cubic centimetre of the filtrate and add 9 cubic centimetres of distilled water. This is labelled with blue pencil on the test-tube 1:10. After this has been carefully mixed and measured, one half is taken and made up to 10 cubic centimetres with water; this is labelled 1: 20. Half of this is taken and made up to 10 cubic centimetres, and the test-tube containing it labelled 1: 40. In a similar way we provide also a 1: 80, a 1: 160, and finally a 1: 320 dilution, each tube being carefully marked. To each test-tube, add 5 cubic centimetres of lukewarm, previously boiled milk. Add to each also 2 to 3 cubic centimetres of 1-per-cent calcium chloride solution. All of these test-tubes are now placed in a water bath (readily extemporized) and kept for 15 minutes as nearly as possible at 100° F. It is well to make a control tube containing only the cooked milk and the calcium solution. In hyperacidity a coagulation of casein may be obtained with as high as 1 : 1000 dilution. Normally, the first 3 dilutions show a stiff coagulation, the 1: 160 shows a moderately stiff coagulation, and the 1: 320 a flocculent coagulation.

From these tests we may draw conclusions as follows: In anacid cases if the milk-curdling ferment is normal it is probable that we have to deal with a nervous disturbance of secretion. If such a case be watched the chances are that the acidity will undergo daily variations and possibly soon become normal. If the ferment is positive for no higher dilution than 1: 80, the likelihood is that we have to deal with a catarrhal condition of the mucosa. If such a case be watched and properly treated the ferment will probably be seen to improve. If there is an absence of the ferment, or the reaction is positive for no higher dilution than 1 : 10, there is a strong suspicion of an atrophic condition of the mucosa. If it is believed that combined acids may have anything to do with the coagulation, this is readily proven by boiling the gastric juice, thus destroying the ferment; it then will fail to cause coagulation. If such is the case, neutralized filtered chyme must be used. In carcinoma there is a tendency for the ferment to disappear after the secretion of free acid has ceased. Instead of making some improvement under treatment, as is the case in neuroses and the less severe forms of catarrh, there is a gradual failure up to entire disappearance.

Pepsin Estimation.—In ordinary practice this is unnecessary, as the quantitative rennin estimation is more accurate. A comparatively simple, but satisfactory, method is to cut thin disks of coagulated egg white, divide these disks in half, place one half in normal stomach contents, and the other in the specimen under examination, after making sure that a slight degree of free acidity is present. Then, for example, if the normal contents dissolve the egg white in 1 hour, and the specimen requires 3 hours, the pepsin power of the latter is but $\frac{1}{3}$ of the normal.

Tests for Occult Blood.—These tests are of the utmost importance in diagnosis of organic stomach diseases, and should be made part of the routine procedure regardless of the gross appearance of the chyme.

Benzidin Test (*Adler's, modified by Schlesinger and Holst*).—As much benzidin (Merck) as will go on a knife blade is shaken up in a test-tube with a few cubic centimetres of glacial acetic acid and allowed to settle. A few cubic centimetres of stomach contents are thoroughly boiled in a test-tube and a few drops added to the upper clear portion of the benzidin-acetic-acid mixture. To this is now added about 5 drops of 10-per-cent hydrogen peroxide, and if blood in anything but negligible amounts be present the mixture will turn a greenish colour in a very few minutes.

Guaiac Test.—This test, elsewhere described (page 678) is less delicate than the former and is therefore useful only as a confirmatory test in case the former gives a faint or doubtful reaction.

C. TO TEST THE MOTOR POWER OF THE STOMACH

Many methods have been devised for determining the motor power of the stomach and the ability of the organ to empty itself; practically the method of choice is test lavage after meals of a given size. The Riegel meal is ordered and the stomach washed out seven hours later. If the stomach is found empty, its motility is not reduced. The amount and character of food remaining affords an indication of the degree of insufficiency.

A convenient way to examine the stomach motility is to have the patient take a meal about eight in the evening, and wash out the stomach before anything is eaten the following morning. For such a meal we may use a soup with rice not well cooked, or some raisins in place of the rice; in addition a few pieces of bread and butter with thin slices of meat, such as corned beef. If food remains are macroscopically evident in the wash water it signifies a mechanical obstruction at the pylorus. Microscopic examination of the sediment of this wash water may be necessary in the detection of lesser degrees of insufficiency.

D. THE MICROSCOPIC EXAMINATION OF THE STOMACH CONTENTS

The extracted chyme after a test meal is not suitable for a microscopic examination, as the undigested foods obscure all else. Information of the greatest value may be obtained, however, from such an examination of the contents of the fasting stomach. For this purpose the tube is passed, preferably in the morning before breakfast, and by "expression," or by suction with a bulb, the stomach contents are obtained. If nothing is found in this way the stomach is washed with 1 or 2 quarts of water, and this water allowed to stand until all solids have settled. The clear fluid is then decanted and the sediment examined by spreading a portion upon a slide, with or without a cover-glass.

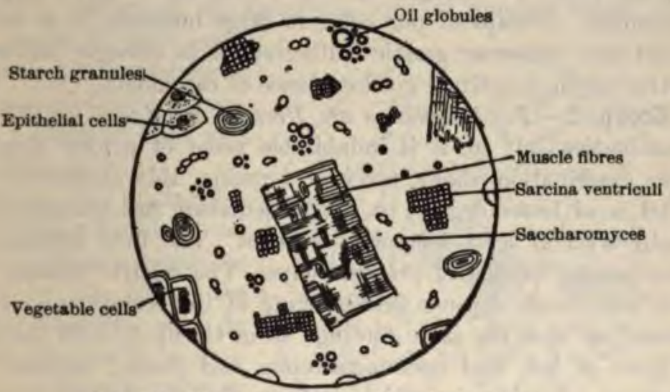


Fig. 258.—Microscopical view of vomited matter.

Bits of tissue are sometimes discovered, and these, after proper hardening and staining, may give clear indications as to the condition of the gastric mucosa. Carcinoma has, not rarely, been thus diagnosed before the appearance of other positive signs.

The sediment differs much in character, depending on the presence or absence of HCl, and on the ability of the stomach to empty itself. Two main groups of cases are thus distinguished by the presence or absence of food remain.

Group 1.—*No Food Remains are Present Even on Microscopic Examination.*—It may therefore be concluded that there is no pyloric stenosis, no actual gastrectasia. If HCl is present, as is the case in the normal stomach, in ulcer, and, commonly, in the neuroses, there will be found free nuclei of leucocytes and epithelial cells, the cell bodies having been digested; bits of mucus showing fine striations, the result of the acid; and myelin spirals, another effect of the normal acid of the gastric juice. If there is no acid, as in achylia, gastritis, and cancer (not involving the pylorus), we find undigested leucocytes and epithelial cells in considerable numbers. Bacteria are present in moderate numbers. Amœba and infusoria are sometimes seen, and the latter are thought by some to be exceedingly suggestive of carcinoma. Clumps of pus cells, in large numbers, if so intimately mixed with columnar gastric epithelium as to strongly indicate their gastric origin, constitute good evidence of carcinoma.

Group 2.—*Food Remains are Present on Macro- or Microscopic Examination.*—If so, it is indubitable proof of pyloric stenosis. If *gross* examination shows no food remains, this obstruction to the outlet is of lesser degree, or the musculature has undergone hypertrophy with at least temporary success. The food substances vary according to the diet of previous days. The relative amount of meat fibre and starch depends on the state of the secretions. If HCl is present we have the same findings as in Group 1, with the addition of more or less food residue, sarcinæ, and yeasts; bacteria are not abundant. Sarcinæ are said to indicate that the stenosis is not acute, as several days are required for their development in the stomach. These findings exclude carcinoma, except the uncommon form which develops on the base of an old ulcer. Ulcer, or cicatrix, is the usual cause for these findings; rarely they may be due to an acid gastritis with hypertrophy.

If no HCl is present and there are food remains, we have the conditions which favour lactic-acid development. Enormous numbers of a lactic acid producing bacillus, the Boas-Oppler, are seen in every field examined. If not obscured by the food remains large numbers of undigested cells may be seen. These findings speak very positively for carcinoma involving the pylorus. The only other condition which occasions stasis, and in which no HCl with its inhibiting effect is found, is distinctly rare—namely, inflammatory hypertrophic stenosis of the pylorus, coincident with a general atrophic gastritis. The Boas-Oppler bacilli require these conditions for their growth; they therefore, if in large numbers, are strong proof of carcinoma. It is clearly established that carcinoma may exist without these bacilli and the consequent formation of lactic acid if the pylorus is not involved in the growth.

1. **Food Particles.**—Fat drops are recognised by their refractility; muscle fibres by their transverse striations; elastic tissue fibres by their curled ends and double contour; starch granules by their concentric striæ, and, if necessary, their blue colour reaction when a weak iodine solution is added; fatty acids by their needle-shaped crystals. Vegetable cells are to be recognised only by a previous acquaintance.

2. **Vegetable Parasites.**—The most important are the *Sarcina ventriculi*, small spherical cells arranged in groups of squares; yeast fungi (*Saccharomyces*), oval or round cells about as large as the average leucocyte, in bunches or chains, staining a deep brown with a dilute iodine solution; and the *Boas-Oppler bacillus* (found quite constantly in cancer of the stomach), rods of unusual length, frequently joined by their ends, and forming characteristic long, angulated threads, stainable by methylene blue and other aniline dyes.

In order to distinguish the Boas-Oppler bacillus from a similar organism, which, as indicated by its name, the *Leptothrix buccalis*, may be found in the mouth, a drop or two of Gram's solution should be added to the specimen. The Boas-Oppler stains brown with the iodine, the Leptothrix blue. Gram's solution consists of one part of iodine, two parts of potassium iodide, and three hundred parts of water.

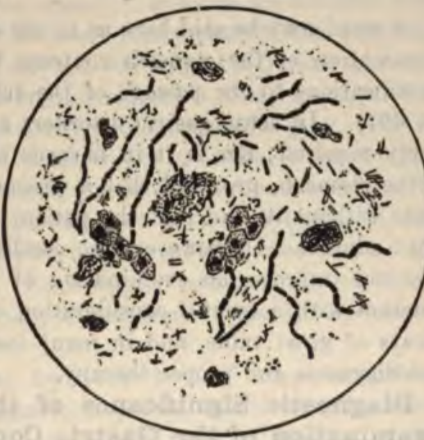


FIG. 259.—Boas-Oppler bacillus. (Reproduced from Simon.)

The Boas-Oppler bacillus is found in from 75 to 85 per cent of the cases of carcinoma of the stomach; very rarely also in non-malignant pyloric obstruction and gastrectasia.

3. **Morphological Elements.**—Leucocytes, red cells (see Hæmatemesis, p. 132), epithelial and mucous-gland cells may also be found. From a diagnostic point of view the most important discovery is that of shreds or fragments of the gastric mucous membrane or of particles of gastric tumours. In order to make a satisfactory examination of such fragments it is necessary to harden, embed, and make sections of them in the usual manner. Unfortunately, however, the information derived from such an examination is not always reliable, as the tumour particles thus obtained are often so disintegrated that it is not possible to find any characteristic cell structure or arrangement. Mucus is recognisable macroscopically, when present in excess, by its tendency to form a sticky layer at the top of the specimen. It adheres to a glass rod dipped below the surface. It is often of importance to determine the presence or absence of *blood*. If present in macroscopic quantities it is readily appreciated as bright or dark red or brown streaks and clots, or coffee grounds. Microscopical amounts, however, must be determined by the benzidin test (p. 666) for occult blood.

III. Diagnostic Value of an Examination of the Stomach Contents

A word may be said here as to the circumstances under which an examination of the stomach contents becomes necessary. The contraindications to the passage of the tube have been stated elsewhere (p. 491). In acute gastric disorders an examination of this kind is rarely required; nor is it in nervous affections and chronic diseases of the stomach, provided that a reasonably certain diagnosis can be made without it, and that the patient is improving under the treatment advised. If, however, the condition is chronic, doubtful, and does not improve, an examination of the stomach contents in conjunction with a careful consideration of the associated symptoms is always of great value, and in many instances indispensable, for correct diagnosis and proper therapy.

Diagnostic Significance of the Various Items of the Examination of the Gastric Contents.—(1) **Quantity of the Gastric Juice.**—The normal amount of *filtrate* to be obtained one hour after Ewald's test breakfast varies from 20 to 50 cubic centimetres. If periodic or chronic hypersecretion (gastrosuccorrhœa) is suspected because of the symptoms, the stomach should be washed out at bedtime and the tube passed before breakfast on the following morning, no fluid or food having been taken in the meantime. If from 100 to 1,000 cubic centimetres of gastric juice are then obtained, we have to deal either with a pure secretory neurosis, or, what is more probable, a gastric ulcer. If the amount of fluid is much above 100 c. c., there is a strong suspicion of pyloric narrowing, and a macroscopic examination should be made of the residue. Total absence of the gastric juice (achylia gastrica) is either a neurosis or, more commonly, an evidence of atrophy of the gastric mucous membrane.

(2) **Acidity of the Gastric Juice.**—The normal total acidity varies from 40 to 60 one hour after Ewald's breakfast. A high degree of acidity (70 to 90) is significant of certain gastric neuroses, and may be associated with hypersecretion. Increased acidity is also found in gastric ulcer. On the other hand, a decreased acidity is a usual concomitant of gastritis, either acute or chronic, and of some neurotic affections of the stomach and carcinoma.

(3) **Presence and Amount of Free Hydrochloric Acid.**—For the production of hydrochloric acid the cells of the gastric glands must be in good condition, the blood supply ample and of good quality, and the innervation competent and regular. A deficiency in one or all of these necessary requirements will produce disturbances in the formation of the acid. Aside from the digestive function of hydrochloric acid, it has been proved that it plays a not unimportant part as a germicide and antiseptic in regard to some of the bacteria which cause either fermentation or disease. Consequently the conditions which diminish the secretion of hydrochloric acid render the individual more vulnerable to all organisms which enter by way of the intestinal tract, either those which are pathogenic (e. g., *Bacillus typhosus*, cholera bacillus), or those which give rise to fermentation and putrefaction (e. g., the bacteria producing lactic or acetic acids). Under normal circumstances the amount of free hydrochloric acid found one hour after the regular test breakfast varies from 0.1 to 0.2 per cent (*euchlorhydria*). Under pathological conditions it may be increased (*hyperchlorhydria*), diminished (*hypochlorhydria*), or absent (*anachlorhydria*).

A *normal secretion* of free hydrochloric acid is not inconsistent with subjective symptoms of gastric disorders, usually of the neurotic kind; but if euchlorhydria is present, it tends to negative chronic gastric catarrh. An *increase* of the free hydrochloric acid (more than 0.2 per cent) is found in gastric ulcer, or in cancer of the stomach originating from an old ulcer. Hyperchlorhydria is most commonly a symptom of a neurosis of the stomach, especially if there is also a continuous hypersecretion. Acid gastritis is a form occasionally discovered, particularly among drinkers. *Hypochlorhydria* (less than 0.1 per cent) indicates in general some condition which has destroyed to a notable extent the secreting glands or cells of the gastric mucous membrane. Consequently, although it may be a neurosis, a diminished amount of free hydrochloric acid is often significant of a more or less chronic gastritis, beginning cancer, dilatation, and, more rarely, of certain cases of gastric ulcer. The *total absence* of free hydrochloric acid is frequently indicative of a gastric neurosis in hysteria and neurasthenia, is usually one of the findings in the later stages of chronic gastritis (atrophic or sclerotic form), and is a quite characteristic but not invariable symptom of gastric cancer.

In the absence of free hydrochloric acid it is of some importance, mainly with reference to treatment, to ascertain whether combined hydrochloric acid is present, and, if so, its amount. If no combined HCl is found, gastric digestion is *nil,* the dietetic management must correspond, and the prognosis is not good. *Per contra,* the presence of combined HCl shows that gastric digestion is partly accomplished, so far as to satisfy, in a measure at least, the chemical affinities of the albuminous foods, although not enough HCl is secreted to afford the normal amount of the free acid.

(4) **Pepsin and Pepsinogen.**—*Absence of pepsin*—i. e., failure of the artificially obtained gastric juice to digest egg albumen without the addition of HCl—is usual when free HCl is lacking, as the presence of the latter is required to transform the pepsinogen into pepsin. The *absence of pepsinogen*—failure to digest after the addition of HCl—is significant of a grave destruction of the glandular elements of the gastric mucous membrane, and therefore warrants a diagnosis of an organic disease—e. g., advanced chronic gastritis, carcinoma—rather than a neurosis. Simple functional derangements (nervous or circulatory) rarely have any marked influence on the secretion of pepsinogen as compared to their notable effect in causing marked variations in the formation of hydrochloric acid.

The presence or absence of *rennin and its zymogen* has practically the same significance as that of pepsin and its zymogen.

(5) **Lactic, Acetic, and Butyric Acids.**—*Lactic acid,* except as derived from the action of certain organized ferments upon the ingested food, or its original presence therein (sarco-lactic acid of meat, lactic acid in bread), is not a normal constituent of the gastric juice. The amount contained in Ewald's test breakfast is not sufficient to give a reaction to the tests usually employed, but if a possible error from this source is to be guarded against, the Boas breakfast should be used. A marked lactic-acid reaction is extremely significant, but not absolutely pathognomonic, of gastric cancer, since it may be found also in the stagnation of gastric dilatation due to non-malignant disease, or when the glandular elements are almost

entirely destroyed. The absence of lactic acid, however, does not forbid the existence of cancer.

The presence of *butyric acid,* except when fats have formed a portion of the meal, as well as of *acetic acid,* except when alcohol, from which acetic acid may be formed by fermentation, has been ingested, has essentially the same diagnostic significance as that of lactic acid.

(6) **Mucus.**—The persistent finding of considerable quantities of mucus in the gastric contents obtained by the tube is good proof of the existence of a form of chronic gastritis which is characterized by an excessive formation of this substance. It is true, however, that this increase of mucus may be a pure neurosis. Vomiting of mucus unmixed with food tends to exclude gastric dilatation, and, if the clinical symptoms of gastritis are absent and it occurs especially in neurasthenic women, is doubtless akin to the neurosis improperly termed membranous enteritis.

The Results of an Examination of the Gastric Contents in Special Diseases or Conditions.—It should be clearly understood that a diagnosis can not be based solely upon an examination of the stomach contents, as in a certain proportion of cases abnormal findings may coexist with an absence of other symptoms. Every evidence of disease, both subjective and objective, is to be taken into consideration before arriving at a definite conclusion.

(1) *Normal Findings.*—The reaction is acid; free hydrochloric acid present in proportion of 0.1 to 0.2 per cent; total acidity, 40 to 60; ferments (pepsin, rennin) present; lactic, acetic, and butyric acids absent; little mucus, and only traces of food.

(2) *Gastric Ulcer.*—Total acidity usually increased; free hydrochloric acid usually increased; ferments present; lactic, acetic, and butyric acids absent; frequently contains blood pigment; stools intermittently contain blood.

(3) *Cancer of the Stomach.*—Total acidity variable; free hydrochloric acid greatly diminished or absent, unless the growth has as a basis an old gastric ulcer; ferments not infrequently absent; lactic acetic, and butyric acids present in quantity after Boas's breakfast; if there is pyloric obstruction, mucus, coffee-ground material, stagnant food, *Boas-Oppler bacillus.*

(4) *Dilatation of the Stomach not caused by Malignant Pyloric Stenosis.*—Total acidity normal or increased; free hydrochloric acid sometimes diminished, more commonly increased; ferments present; lactic, acetic, and butyric acids absent after Boas's, present after Ewald's, breakfast; decomposed and undigested food, yeast fungi, and bacteria.

(5) *Acute Gastritis.*—Total acidity decreased; free hydrochloric acid absent; ferments diminished; lactic, acetic, and butyric acids usually absent; mucus, partly digested food particles, some red blood cells, fluid tinged green (bile pigment).

(6) *Chronic Gastritis.*—Total acidity diminished (in atrophic form reaction neutral or alkaline); free hydrochloric acid diminished or absent, and in the atrophic form absent; lactic, acetic, and butyric acids absent after Boas's breakfast; ferments present (in atrophic form absent); partly digested food present (in the atrophic form no evidence of digestion); much mucus indicates the so-called "mucous gastritis"; shreds of mucous membrane (erosions) present except in atrophic form.

(7) *Hyperchlorhydria.*—Total acidity, 70 to 140; free hydrochloric acid much increased; ferments normal or increased; diagnosis not to be made unless the hyperacidity is persistent.

(8) *Hypersecretion, Periodic (Gastroxynsis) or Chronic.*—Presence of 100 to 1,000 cubic centimetres of gastric juice in the fasting stomach, continuously or periodically.

(9) *Achylia Gastrica.*—Total acidity low, may be neutral; no hydrochloric acid; no ferments; gastric juice practically absent. While the condition usually signifies the presence of one of the diseases causing anachlorhydria, it may be a neurosis.

SECTION XLI

THE MICROSCOPICAL AND CHEMICAL EXAMINATION OF FÆCES

THE gross characters of the fæces have been previously described (p. 142). The microscopical examination involves a search for food particles, morphological elements, crystals, and parasites (animal or vegetable).

Test Diet and Fæces.—Macroscopically, microscopically, and chemically the fæces vary greatly with the diet. In bedside practice it is customary to examine the fæces at once in the hope of elucidating some particular point. In chronic disease much information can be obtained if the patient is put upon a certain fixed diet, the normal fæcal result from which is known to the investigator. Schmidt and Strassburger have suggested such a dietary, and speak of the resulting fæces as test fæces. The dietary suggested is not suited to our customs in minor particulars, and the following modification (ROBERTS) is more available:

For Breakfast

A teacupful of well strained oatmeal	160 C.
A tablespoonful of cream	80 C.
A teaspoonful of sugar	40 C.
Lean meat, finely chopped, slightly cooked in pan—an amount equal to a heaping tablespoonful	60 C.
Three slices of toast (4 by 4 by ½ inch thick)	150 C.
An inch cube of butter	65 C.
A large glass of milk (8 oz.)	160 C.

At Midday

Potato puree (one potato to 8 oz. milk)	225 C.
A slice of toast, size as above	50 C.
A half-inch cube of butter	30 C.
Lean meat, as above, a teacupful in amount	150 C.
Add an inch cube of butter	65 C.
One large, well-baked potato	75 C.
Add 2 teaspoonfuls of cream or ½ inch cube of butter	40 C.
Two slices of bread, size as above	100 C.
An inch cube of butter	65 C.
A large glass of milk	160 C.

For Supper

A teacupful of rice, cooked dry	150 C.
Add an inch cube of butter	65 C.
Two poached eggs	140 C.
Three slices of bread	150 C.
An inch cube of butter	65 C.
A large glass of milk	150 C.

In this dietary there are about 2,220 calories, made up of 100 grams of proteid, 80 grams of fat, and 300 grams of carbohydrate.

On the morning the diet is started give a capsule containing 5 grains of carmine to demarcate the fæces resulting from the diet.

If the stool is homogeneous any portion is satisfactory, but it is well to take specimens from various parts if it present a diversified appearance. This is gently mixed to a semifluid consistence in a small glass mortar.

Glass Plate Examination.—An important adjunct to naked-eye inspection. A few drops of the fæces are placed upon a glass plate about 3 by 5 inches in size; a similar plate is then placed above the specimen. The preparation is held up to the light and inspected for mucus, fat, foreign bodies, and worms. If one plate is moved upon the other mucus is readily distinguished by its peculiar glistening appearance; and fat in excess gives a greasy feel and look.

Microscopic Examination.—These plates are placed upon the stage of the microscope and searched with the lowest power. Suspicious objects can be more closely examined if a cover-glass is placed on the spot selected after the plates have been separated; in this way eggs may be discovered, even if few in number.

A slide of large dimensions is then selected and a large drop of the fæces mixture placed in the middle, and one on each end. A large cover-glass is placed over the first. Two drops of Lugol's solution are mixed with the middle specimen and properly covered. To the one on the other end a few drops of glacial acetic acid are added; the fæces are thoroughly mixed with the acid, and that end of the slide gradually heated in the flame until bubbles just begin to appear; when somewhat cooled a cover-glass is placed over the preparation.

The *first* (or native) preparation is searched for meat fibres, connective tissue, mucus, and eggs. Normally only a few meat fibres having very faint striations are found. An excess indicates a deficiency in pancreatic digestion. Connective tissue, known by its whitish-yellow appearance and faint longitudinal striations, is normally not found. In failing gastric digestion it may be in excess, but only if the meat has not been more than heated through. Mucus, even when recognised microscopically, evidences a pathological condition. The finer the division of the mucus and the more intimately intermixed it is with fæcal matter the higher is its origin. This is a safe general rule. Fine bits of mucus, stained greenish, come from the upper intestine and indicate a catarrhal process. In this specimen also we look for the eggs of parasites.

The *second* preparation (stained with Lugol's solution) normally shows an occasional starch cell, coloured blue. In certain forms of intestinal indigestion, particularly those occasioned by upper intestinal catarrh, entire fields of the microscope may be stained blue. A diet over-rich in starch may give these findings without indicating disease; this is often observed in artificially fed infants.

The *third* (acetic acid) preparation normally shows a few fatty acid crystals, but in hepatic and pancreatic deficiency such crystals may cover the slide so treated. In actual pancreatic disease there is apt to be a number of droplets of neutral fat.

Still another microscopic examination sometimes of value is of a drop of the mixture taken from the sublimate test (see *infra*); an exaggeration of the peristaltic rate in the small intestine is shown by the presence of green-stained meat fibres and bits of mucus.

Chemical Tests.—*Bile Salts.*—The so-called clay-coloured stools are frequently erroneously supposed to be alcoholic. In part the lack of colour in such cases may be due to excess of fat, and when this excess is removed the stool shows some colour; in part, also, it is due to the presence of a colourless bile salt—leukohydrobilirubin.

The mercuric chloride test is made by adding a few cubic centimetres of a saturated solution of corrosive sublimate to a small mass of fæces. The amount of bile present is estimated by the quickness of appearance and depth of a pinkish-red colour; the non-appearance of this colouration at the end of 12 hours demonstrates the absence of bile. A greenish colouration, in whole or part, evidences an exaggeration of peristalsis, a failure of bilirubin conversion.

Occult Blood Tests.—The *benzidin test* (p. 666), being the most delicate, is done first. As small an amount of blood, as 5 ♏ *per orem*, usually shows in the stools. A piece of solid fæces about the size of a hazelnut is rubbed up with a few cubic centimetres of water and the mixture thoroughly boiled. To the acetic-acid solution of benzidin a few drops of the fæcal mixture is added, and then a few drops of a 10-per-cent solution of hydrogen peroxide. The positive reaction, a green

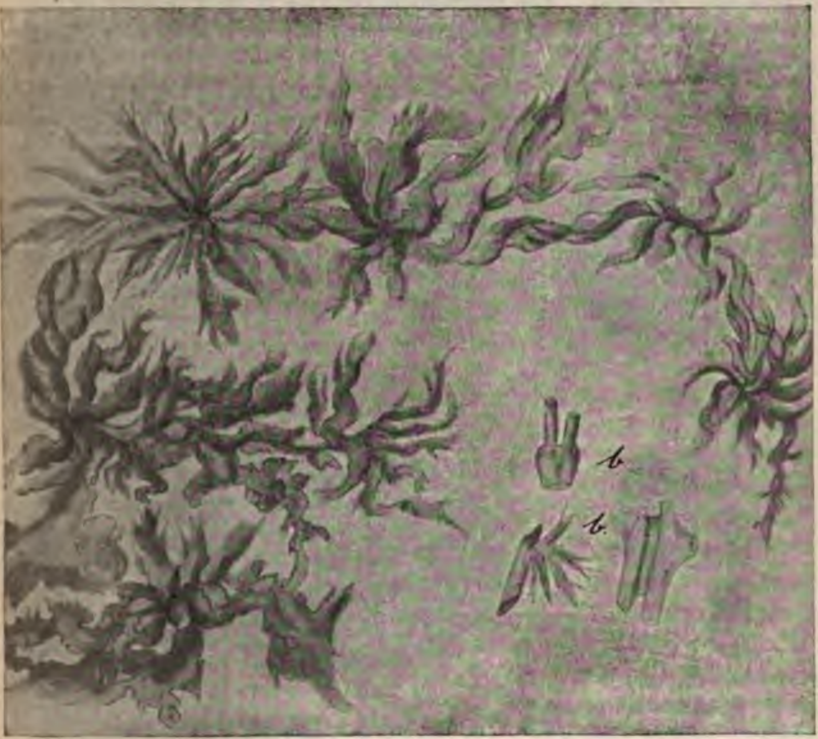


FIG. 260.—Spirals of undigested meat fragments in fæces. (Natural size.) *b*, pieces of bronchi. (Boas.)

or greenish blue, appears in the course of a minute; this fades gradually to a purple and then to a reddish hue. If more than 4 or 5 drops are required the reaction is doubtful. The less delicate (*guaiac*, *aloin*) tests should then be used. The steps for these are as follows:

Rub up with ether a chestnut-sized lump of fæces; let stand a half hour, until fat is extracted; pour off ether. A few drops of glacial acetic acid are then mixed with the fæces, and a few c.c. of ether added; the acid ether extracts the hæmatin, if present, from the fæces. This acid ether is decanted and in it is put a small amount of freshly powdered gum guaiac or Barbadoes aloin. To this is added a c.c. of old, oxygenated, but pure oil of turpentine, and the mixture thoroughly agitated. The reaction is positive if in a minute or two a blue or greenish colour results if guaiac has been used, or a cherry red if aloin has been used.

If no meat or meat products have been ingested by the patient for several days, a positive reaction for blood, if its origin from the rectum or respiratory passages can be excluded, means that, in all probability there is a malignant or ulcerative process somewhere in the gastro-intestinal canal. In cancer the reaction is found comparatively early in a majority of instances; once present it is constant. In ulcer of the stomach or duodenum, blood is found occasionally during the subacute or chronic stages, and often daily in early or fresh cases.

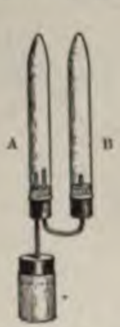


Fig. 260*a*. — Apparatus for Schmidt's fermentation test.

The occult blood tests are of great value in establishing the differential diagnosis between disease affecting the upper abdomen, and should be in general use.

Schmidt's Fermentation Test.—A small spatulaful of fæces is rubbed up with 10 cubic centimetres of distilled water; if the fæces are thin more is taken, no dilution being necessary for watery stools. This mixture is placed in the bottom vessel (Fig. 260*a*) and its cork put in securely. Tube A is then filled with water and its cork inserted. Tube B is corked and placed, and the apparatus is set in the incubator and kept at a temperature of 37° C. for 24 hours. The reaction of the specimens is taken upon starting and afterward.

Normally not more than one third of B is filled with water from A as a result of gas arising from the fæces. The air escapes from B through a hole in the top. If half is filled it evidences a pathologic condition. On the Schmidt diet *starch* may not be seen microscopically, with the Lugol's solution, and yet be shown by fermentation. If the gas is from fermentation of the *carbohydrate* food residue the reaction of the fæces is sharply alkaline, and the gas has a smell of butyric acid. A positive test indicates, according to Schmidt and Strassburger, that there is an abnormality of the small intestine, probably a catarrh of the mucosa. *Putrefaction* usually takes longer to show itself in gas development, and the fermentation tube is left in the oven for 24 hours longer. The alkaline reaction and the putrid smell show that there has been decomposition of albumins which should not be present in the fæces.

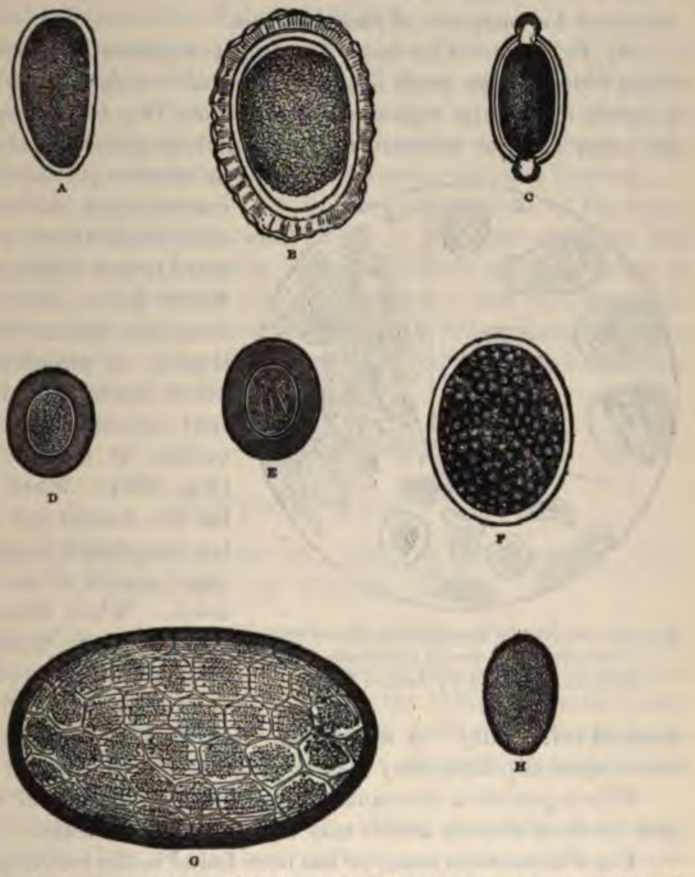


FIG. 261.—Ova of entozoa, × 350 (after Heller). A, Oxyuris vermicularis; B, ascaris lumbricoides; C, trichocephalus dispar; D, tænia solium; E, tænia mediocanellata; F, bothriocephalus latus; G, distoma lanceolatum; H, distoma lanceolatum. See also pp. 149–155.

Parasites, Animal and Vegetable.—Of the many varieties of living organisms found in the intestine the following are pathogenic, and therefore of clinical importance:

(*a*) **Animal Organisms.**—(1) *Vermes or Worms.*—The adult vermes have already been described in connection with the naked-eye inspection of the fæces (page 149). In the event of the non-discovery of the full-grown organisms the search for and finding of their ova in a suspected case is of importance. In Fig. 261 the ova of the worms most likely to be encountered are shown with sufficient distinctness for purposes of identification.

(2) *Protozoa.*—The most important representative of the protozoa found in the fæces is the *Amœba coli* (or *dysenteriæ*). This is a motile unicellular organism varying from 10 μ to 25 μ in diameter. Its outer zone or ectosarc is clear and homogeneous; the endosarc or interior portion is granular, containing a nucleus of variable distinctness, and one or more rather striking vacuoles. When active, the contour is irregular, because of the protrusion of pseudopodia composed mainly of the translucent ectosarc. At rest, the outline is circular or ovoid (Fig. 262). When searching for the *Amœba coli* special attention should be given to the small masses of mucus in the stool. When found in the fæces they are usually at rest, and are to be recognised by their light greenish tint and marked refractility. A warm stage facilitates their recognition. In dried cover-slip films they may be stained with methylene blue.

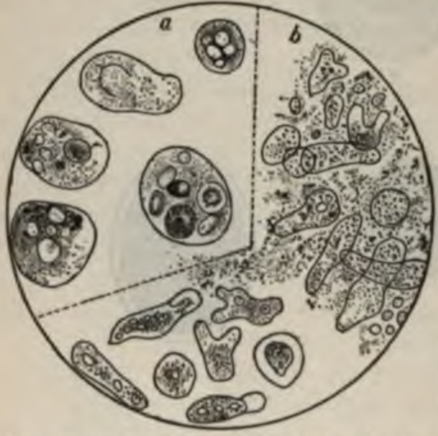


FIG. 262.—*a*, Amœba dysenteriæ fixed and stained (Councilman); *b*, amœba dysenteriæ in stools (after Lösch, Virchow's Archiv, Bd. 65).

This organism is the cause of the amœbic dysentery of the tropics, and the liver abscess which may complicate the disease.

The *Plasmodium malariæ* has been found in the red corpuscles contained in the stools from a case of chronic malarial colitis (SIMON).

(*b*) **Vegetable Organisms.**—Of these, the most important are the *Comma bacillus*, the *Bacillus typhosus*, the *Bacillus coli communis*, *Bacillus dysenteriæ* (SHIGA), and the *Bacillus tuberculosis*. Their sure recognition, with the exception of the last mentioned, requires the resources of the bacteriological expert.

(1) *Comma bacillus.*—This is a curved, rod-shaped, mobile organism, shorter and thicker than the tubercle bacillus. It is the specific cause of Asiatic cholera.

(2) *Typhoid bacillus* (EBERTH).—This is a flagellated motile rod with rounded ends, about half the diameter of a red blood corpuscle.

(3) *Bacillus coli communis.*—This organism, which closely resembles Eberth's bacillus of typhoid fever, is constantly present in normal fæces. It may become pathogenic and cause suppurative inflammation—e. g., cystitis, appendicitis, and perforation peritonitis, pyelitis, and inflammation of the gall bladder.

(4) *Bacillus tuberculosis.*—This may be demonstrated in the fæces by making and staining cover-slip films as directed for the sputum (page 655). When found they are not to be considered indicative of intestinal tuberculosis unless the clinical symptoms of the latter are present, as they may originate from swallowed sputum.

(5) *Bacillus dysenteriæ.*—This, like the other bacilli of the colon group, is a short rod with rounded ends. It has active motion, but not locomotion; responds to the basic stains, and is decolourized by Gram's method. Two forms, the Asiatic (SHIGA) and the American (HARRIS), are recognised by their reaction, the latter turning litmus mannite red (SIMON). It agglutinates with blood from cases of dysentery in the manner of the Widal reaction.

SECTION XLII

DIAGNOSTIC INFERENCES TO BE DRAWN FROM THE RESULTS OF URINALYSIS

IN view of the abundant supply of excellent special treatises, both large and small, which deal with the *technic* of urinalysis, it has seemed a work of supererogation to cumber this work with necessarily abbreviated descriptions of the large number of extant methods. This section therefore deals with the *diagnostic significance* of the various findings of the urinalysis.

The physical examination of the kidney has been previously described (page 512); so also have certain symptoms relating to the genito-urinary system (page 160).

I. EVIDENCE DERIVED FROM A PHYSICAL EXAMINATION OF THE URINE

(1) **Quantity of the Urine.**—The amount of urine excreted in 24 hours varies considerably under normal circumstances. The average total quantity is stated variously by different writers, the discrepancy depending largely upon the country in which their observations were made. For the United States we may accept as

normal 1,200 cubic centimetres (40 oz.) for the adult man, and 1,000 cubic centimetres (33 oz.) for the adult woman. In children between 2 and 12 years of age the number of ounces is approximately equal to double the years of the child's age.

The total daily excretion is physiologically increased in cold weather and by the taking of large amounts of fluid; it is decreased in hot weather, by free perspiration, and by a diminished consumption of fluids. With reference to diurnal variations, it may be remembered that from 2 to 4 times as much urine is passed during the day as during the night.

An amount less than 500 cubic centimetres (17 oz.) or more than 3,000 cubic centimetres (99 oz.), if persistent, may be considered to be pathological; but the occasional finding of such quantities does not necessarily imply an abnormal condition.

(*a*) **Increased Quantity.**—Polyuria may signify diabetes (mellitus or insipidus) in which 25,000 cubic centimetres (825 oz.), or even more, have been passed; and chronic interstitial nephritis (2,000 to 4,000 cubic centimetres, 66 to 132 oz. or more). Amyloid disease of the kidney is attended with an increased excretion of urine. Polyuria also occurs during the resorption of large effusions (pleural, peritoneal, pericardial, anasarcous); as a symptom of certain functional diseases of the nervous system, notably hysteria and neurasthenia, epilepsy, chorea, or migraine; and, usually as a favourable sign, during the defervescence of acute fevers. A persistent polyuria is seen in some organic diseases of the nervous system, as in certain cases of cerebellar, bulbar, and spinal tumours, meningitis, and locomotor ataxia. An excessive flow of urine may in rare instances be a part of the condition termed phosphatic diabetes (see Phosphates).

(*b*) **Diminished Quantity.**—Oliguria is one of the evidences of a low blood pressure, and is consequently met with in cases of broken compensation due to valvular disease, or in weak heart dependent upon degeneration of the cardiac muscle occurring as a result of long-continued fevers or wasting disease. In all fevers the urine is usually scanty. A diminution is also seen in acute and chronic parenchymatous nephritis. Interference with the return venous circulation of the kidney—i. e., an increase of the renal intravenous pressure—produces oliguria, as in pressure from ascites or abdominal tumours, or a damming back of the blood by thrombosis of the inferior cava or renal vein. So also does the loss of a large amount of fluid from the body, as in cases of severe diarrhœa, cholera, persistent vomiting, or large hemorrhages. A calculus lodged in the ureter, or a tumour pressing upon the latter, may account for an oliguria. Scanty urine finally may be a symptom of hysteria, melancholia, and lead-poisoning.

Complete suppression of urine (Anuria, *q. v.*) has been described elsewhere.

(2) **Colour of the Urine.**—Normally the urine varies from a light yellow to a brownish red. The more acid the urine and the greater its specific gravity the more highly coloured it is, with the exception of diabetic urine, in which the colour is light and the specific gravity high.

(*a*) *Pale Urine.*—This usually indicates an excess of water, and is found in the various conditions enumerated as causing polyuria. Its presence contraindicates high fever.

(*b*) *Greenish-yellow or Yellowish-brown Urine.*—Usually indicates the presence of bile (see Choluria), but may be due to the ingestion of santonin, salol, carbolic acid, guaiacol, naphthaline, or resorcin.

(*c*) *Red, Orange-coloured, Reddish-brown, Smoky, or Brownish-black Urine.*—Generally caused by the presence of blood or its derivatives (see Hæmaturia). Black urine (containing melanin) is, in rare instances, due to the existence of melanotic cancer in some part of the body. The administration of senna, rhubarb, and chrysophanic acid may colour the urine orange or brown. A port-wine colour may signify the existence of hæmatoporphyrinuria (*q. v.*).

(*d*) *Milky Urine.*—Is seen as an evidence of chyluria, or lipuria, or is in some cases due to the presence of pus.

(*e*) *Blue Urine.*—May be due to the presence of indigo formed from an excess of urinary indican (*q. v.*), or occur as a result of the administration of methylene blue, or sweets coloured with it.

(*f*) *Urines which Darken on Standing.*—May indicate the presence of alkapton or related oxyacids; or melanin (melanotic cancer); or the ingestion of carbolic and salicylic acids, salol, and pyrocatechin. An iridescent brittle film composed of calcium phosphate may form on the surface of standing urine if the latter is alkaline. It has no clinical significance.

(3) **Odour of the Urine.**—The normal odour of the urine is variously described as aromatic, or resembling that of bouillon. A so-called "urinous" or ammoniacal odour is indicative of decomposition. The taking of asparagus, onions, santonin, or cubebs will impart characteristic odours. Turpentine affords an odour of violets; in diabetes it resembles new-mown hay; in acetonuria, overripe apples; and a fæcal odour may be due to a fistulous communication between the intestinal and urinary tracts.

(4) **Consistence of the Urine.**—Normally it is aqueous and very slightly viscid. The presence of large amounts of bile, albumin,

sugar, pus, blood, or blood plasma (fibrinuria) renders it more or less viscid, so that the froth formed upon shaking may remain for an unusually long time, and the urine is filtered with difficulty.

(5) **Specific Gravity of the Urine.**—Normally the density of the urine varies between 1.015 and 1.025. The specific gravity depends upon the relative amount of the fluid and the solids contained in solution (not in suspension). It may under normal circumstances temporarily rise above or descend below these limits, depending for the most part on the amount of fluid ingested or which passes off as perspiration. Pathologically, as a general rule with several exceptions, an increase in the total amount of urine is accompanied by a lowered specific gravity, while in oliguria the density is increased.

Polyuria of a persistent type, with a *low* specific gravity, is significant of chronic interstitial nephritis or diabetes insipidus; with a *high* specific gravity (1.030 to 1.045), of diabetes mellitus. Oliguria with a low specific gravity may be present in cases where defective elimination of solids is conjoined with a weak heart, as in many chronic diseases or grave acute ailments.

(6) **Total Urinary Solids.**—If the last two figures of the specific gravity of the collected and mixed urine of 24 hours are multiplied by the coefficient 2.33, the result is the number of grammes of solids in 1,000 cubic centimetres of urine. Thus, if the specific gravity is 1.022 and the amount passed is 1,200 cubic centimetres, $22 \times 2.33 = 51.2$ grammes to 1.000 cubic centimetres, or 5.1 per cent. Two hundred cubic centimetres will therefore contain $2 \times 5.1 = 10.2$ grammes, which added to 51.2 gives a total output of 61.4 grammes.

Another method is: Multiply the last two figures of the specific gravity by the number of ounces voided in 24 hours and the product by 1.1; e. g., if the total amount is 40 oz., and the specific gravity is 1.022, then $40 \times 22 \times 1.1 = 968$ grains (about 2 oz.).

The normal average daily amount of solids in the urine varies from 60 to 70 grammes (1.8 to 2.2 oz.). A notable diminution in this amount is significant of a corresponding degree of renal insufficiency. The cause of the insufficiency must be determined by a consideration of the associated signs and symptoms which may indicate that the kidneys themselves are diseased, or that their eliminative power is lessened as a remote or indirect result of disease elsewhere.

The following table (constructed after Etheridge) presents the minimum amounts of total urinary solids which can be considered as normal with reference to the weight of the individual:

Weight.	Minimum urinary solids.
90 pounds	500 grains.
100 "	570 "
110 "	640 "
120 "	710 "
130 "	780 "
140 "	850 "
150 "	920 "
160 "	990 "
170 "	1,060 "
180 "	1,100 "

Dyspepsias, neuralgias, nervous irritability, bronchitis, headaches, insomnia, and backache have been referred to a deficient elimination of urinary solids, *without* existing nephritis.

(7) **Urinary Deposits** (*Macroscopic*).—The formation of a deposit, visible to the unaided eye, in the urine when the latter is allowed to stand, depends more upon the reaction of the fluid than upon the amount of the contained ingredients. Consequently the existence of a more or less abundant sediment composed of a certain substance does not argue the presence of that substance in abnormal quantity—a question to be settled only by chemical analysis.

A deposit of "cayenne pepper" grains, brick-red in colour, consists of uric acid, and may or may not indicate an increased elimination of the acid. Its occurrence is mainly indicative of a very acid or scanty and concentrated urine, such as occurs in fever, gastric disorders, wasting diseases, and lithæmic conditions. If actual concretions (uric acid sand or gravel) are found, their presence may serve to clear up the nature of attacks of pain previously suspected to be of renal origin. The very common brick-dust sediment is composed of amorphous urates. It occurs, especially in cold weather, under the same circumstances as uric-acid deposits. An abundant white deposit of phosphates, resembling pus, may be found in neutral or alkaline urines, and when persistent is usually associated with neurasthenia and debility.

It is perhaps regrettable that there are no accepted terms to express the clinical conditions, sometimes well characterized, in which there is a persistent naked-eye deposit of certain substances (urates, phosphates), while the total output, as ascertained by chemical examination, may or may not be increased or diminished. Even some of the systematic writers upon urinalysis, after giving warning not to mistake a deposit for an excess, fall into the very error against which they have cautioned their readers.

(8) **Pneumaturia.**—In rare instances, in connection with evidences of cystitis, gas may be passed at the end of micturition, with or without an audible sound. If air has not been introduced into the bladder during instrumentation (cystoscopic examination, irrigation of the bladder, etc.), it is due either to a fistulous communication between the bladder and some portion of the intestine, or to the presence of gas-forming organisms which have entered or been carried into the viscus. The yeast fungus, the *Bacillus coli communis*, and the *Bacillus aerogenes capsulatus* have been isolated, and in most cases glycosuria is present. If it is suspected that the bladder contains air, the suspicion may be confirmed by passing a catheter and keeping its end under water, or by desiring the patient to urinate in a bath.

II. EVIDENCE FROM THE CHEMICAL EXAMINATION OF THE URINE

(1) **Reaction of the Urine.**—(*a*) **Acidity.**—Normally the reaction of the collected urine of 24 hours is acid. An excessively acid reaction is found in the majority of scanty urines, particularly the concentrated urine of acute rheumatic fever and acute febrile diseases in general. It is also observed in lithæmia, gout, diabetes, scurvy, leucæmia, and chronic nephritis. A persistent high acidity is suggestive of possible renal lithiasis.

(*b*) **Alkalinity.**—If the diet is purely vegetable the reaction may be alkaline. It is also temporarily alkaline after the ingestion of a large meal of any kind of food. If the urine is alkaline when voided, and the alkalinity is due to the presence of *ammonia*, it may be inferred that cystitis exists. If the urine is alkaline when voided, but the reaction is due to a *fixed alkali*, it may be due to the ingestion of organic acid salts, anæmia, certain neurasthenic or debilitated conditions, and prolonged vomiting.

(2) **Chlorides.**—The normal average daily excretion of chlorides amounts to 12 grammes, but varies physiologically from 10 to 15 grammes, the variations depending mainly on the amount ingested as a part of the food. If quantities between these limits are found it may be safely inferred that the appetite and digestion are not impaired to any notable extent.

(*a*) **Increased Chlorides.**—A persistent increase in the chlorides (15 to 30 grammes) is extremely suggestive of diabetes insipidus. An increase occurs also in the early stage of general paresis; in prurigo; during convalescence from certain acute fevers, especially the third day of convalescence from acute lobar pneumonia; during the post-convulsive stage of epilepsy; and during the rapid absorption of large effusions. In the latter case it is a favourable prognostic sign.

(*b*) **Diminished or Absent Chlorides.**—In cases of inanition or starvation the excretion of chlorides will diminish or almost cease, the sodium chloride, which constitutes the great bulk of the chlorides, being retained in the body fluids so as to preserve very nearly its normal proportion in the latter. Pathologically the chlorides are diminished in all fevers except in intermittent malarial fever, which shows no diminution or only a relatively slight decrease. The most striking instance of a great decrease, or even a total disappearance, of the chlorides is witnessed in acute lobar pneumonia, a symptom which may be of great value in the differential diagnosis of this form of pneumonia from pleurisy and empyema. The acute fevers presenting a decrease, but not to such a marked degree as in pneumonia, are typhus fever, rheumatic fever, scarlet fever, small-pox, recurring fever, and acute yellow atrophy of the liver. The excretion of only 0.05 gramme of chlorides in 24 hours indicates a very severe form of disease. The chlorides are also diminished in varying degrees in excessive diarrhœa, anæmia, rachitis, chronic plumbism, chorea, melancholia (decrease marked), gastric cancer, hyperchlorhydria and hypersecretion associated with gastric ulcer (chlorides may disappear), in most cases of albuminuria, and in large effusions.

(3) **Phosphates.**—Under normal circumstances the daily elimination of phosphoric acid varies from 2 to 3 grammes (30 to 45 grains). The acid is excreted in combination as alkaline and earthy phosphates, the alkaline salts predominating. It is derived mainly from the food, partly from the decomposition of lecithin and nuclein.

(*a*) **Decreased Phosphates.**—A diminution in the excretion of phosphates is customary in typhus, typhoid, and intermittent malarial fevers, in pulmonary tuberculosis with high temperature, and acute febrile diseases in general, although there are some unexplained exceptions to this statement. A decreased elimination has also been noted in the various forms of nephritis, acute and chronic rheumatism, Addison's disease, hysteria, chronic lead-poisoning, and acute yellow atrophy of the liver.

(*b*) **Increased Phosphates.**—An excess of phosphates (phosphaturia) exists in wasting diseases, and in leucæmia and severe anæmia. An increased elimination is said to be particularly marked in wasting diseases of the nervous system. A considerable sediment of the earthy phosphates occurs in alkaline urine when the alkalinity is due to a fixed alkali. It is found in dyspeptic and nervously debilitated individuals. Occasionally the deposition takes place in the bladder, and the few whitish drops passed at the end of urination may be mistaken for semen and cause much unnecessary anxiety on the part of the patient. In such cases there is usually, but not necessarily, an actual increase, as determined by a quantitative analysis. There is a condition, perhaps a nosological entity, in which with polyuria, thirst, loss of flesh, and other symptoms of diabetes, sugar is usually absent and the urine is alkaline, but there is a great increase (7 to 9 grammes) in the phosphates (phosphatic diabetes). In diabetes mellitus the phosphates may increase as the sugar diminishes, and *per contra.*

(4) **Oxalates.**—Oxalic acid, of which from 0.010 to 0.020 gramme ($\frac{1}{6}$ to $\frac{1}{3}$ grain) is excreted daily, exists in combination as calcium oxalate. It is normally held in solution by the acid sodium phosphate of the urine, and when the latter is deficient the oxalates are precipitated. The amount of oxalic acid excreted depends largely upon the diet. The taking of certain vegetables, especially cabbage, rhubarb, and tomatoes, will cause a considerable increase. Asparagus, spinach, carrots, string beans, and celery have a similar action. In estimating the pathological importance of an excess of oxalates allowance must be made for the effect of diet. Oxalic acid may also result from oxidation of uric acid or an incomplete oxidation of carbohydrates. In the latter case the intermediate product is oxaluric acid.

A persistent increase in the oxalates (oxaluria) may be due to disorders of the stomach and intestine. When associated with neurasthenia, hypochondria, debility, dyspepsia, and perhaps various neuralgias, oxaluria constitutes a well-marked clinical condition, considered by certain writers to be a special diathesis. This condition is due to some unknown disturbance of metabolism. Oxaluria is not infrequently associated with transient albuminuria, and occurs in gout, lithæmia, and spermatorrhœa. Oxalates may be deposited in the bladder and form a calculus. Indianuria often coexists.

(5) **Sulphates.**—The sulphuric acid occurring in the urine exists in two forms: first, as the mineral (inorganic or preformed) sulphates in combination with sodium and potassium; second, as organic (conjugate or ethereal) sulphates in combination with indol, skatol, and phenol. The amount of the former is to the latter as 10 to 1, and the daily total excretion of both varies, in health, from 2 to 3 grammes (30 to 45 grains). With the exception of the small amount of the mineral sulphates ingested as a part of the food, the sulphates are derived from the breaking up of albuminous substances in the body.

Consequently an increased excretion of the total sulphates has been observed in acute febrile diseases, especially pneumonia and acute myelitis, as well as in non-febrile wasting maladies such as cancer of the esophagus, diabetes (both mellitus and insipidus), and progressive muscular atrophy. With reference to the organic sulphates, see the following paragraph.

(6) **Indican.**—When albuminous substances are undergoing bacterial putrefaction in the intestine, or are rapidly decomposing in any part of the body, as in the putrid pus of septic peritonitis or in empyema, indol is formed. When the indol is absorbed it is oxidized, forming indoxyl, and the latter unites with the *preformed* potassium sulphate to become the *conjugate* potassium indoxyl-sulphate, or, as it is more commonly termed, indican. If indican, which is itself colourless, is treated with strong acids and oxidizing agents, it is decomposed with the formation of indigo blue.

Indican is present in small quantity in healthy urines. It is *increased* (indicanuria) when an excessive degree of intestinal putrefaction is taking place. According to Simon, an excessive excretion of indican is often indicative of a decreased proportion of hydrochloric acid in the gastric juice, as under such circumstances the normal restraining influence of the acid upon putrefactive processes is abolished. Consequently an excess of indican is found in gastric diseases attended by hypochlorhydria or anachlorhydria (*q. v.*), notably gastric cancer and the various forms of gastritis. It occurs also in diseases of the small intestine which involve diminished or absent peristalsis, as in peritonitis, and ileus or obstruction of this portion of the digestive tract; whereas in conditions affecting the colon alone no increase is found. As a rule, indicanuria does not result from ordinary constipation. As previously stated, an excess of indican is found when putrid pus exists in any part of the body. The amount of indican is increased by the ingestion of considerable quantities of red meat, and diminished by an exclusive milk diet.

(7) **Urea.**—In health, the bulk (85 per cent) of the nitrogen eliminated by the kidneys is in the form of urea. It is derived in part from the unused proteids of the food, and in part from the physiological waste of the body or tissue proteids. It is probable, although perhaps not definitely determined, that the liver is the chief seat of production of urea. The daily excretion of urea in health varies from 20 to 40 grammes (about 300 to 600 grains), with an average of 28 grammes (450 grains). The variations depend partly upon the amount of proteid food ingested, partly upon the body weight. Under normal conditions the more abundant the diet and the greater the weight of the body the more urea is formed. The elimination of urea is absolutely less in women and children than

in men, but relatively greater (compared with the body weight) in children than in adults.

In disease, the total daily excretion of urea may be increased or diminished.

(*a*) **Increased Urea.**—The excretion of urea is in large measure an index of the activity of tissue destruction. Its total daily output is therefore increased in the acute febrile and inflammatory diseases, particularly in those which terminate by crisis—e. g., pneumonia. The largest output is found in diabetes, in which, with a few exceptions, urea is found in excess, due partly to the abnormally great appetite, but partly also to the marked tissue waste which attends this disease.

A *plus* amount of urea may occur in severe leucæmia, pernicious anæmia, scurvy, and paralysis agitans; also after the use of electricity, and poisoning by certain drugs, especially arsenic and phosphorus.

(*b*) **Decreased Urea.**—A lessened elimination of urea occurs in the various forms of nephritis and renal insufficiency. While it can not be said that urea alone is the cause of uræmia, yet its amount furnishes a fair gauge of the eliminative ability of a diseased kidney. A decrease in urea is also observed in certain diseases of the liver, (cancer, cirrhosis) which affect its functionating power and therefore cause a decreased urea formation. Diminished amounts are noted in melancholia, general paralysis, Addison's disease, chronic anæmias, chronic plumbism, osteomalacia, chronic rheumatism, and certain cases of diabetes in which there is a deficient absorption of nitrogenous materials from the small intestine.

(8) **Uric Acid and its Congeners.**—Uric acid is one of a number of grouped substances which, as each one contains the nucleus purin (C_5N_4), are termed synonymously purins or purin bodies or bases, also formerly called alloxurs, or alloxuric bodies. Those of most common occurrence are hypoxanthin, xanthin, uric acid, guanin, and the methyl xanthins (caffeine and theobromine). These bodies exist either as "free" purins, or are combined with albumin in the shape of nucleic acid to form nucleins ("bound" purins). With the exception of uric acid they occur in certain food stuffs, both animal and vegetable.

Of the purins found in the urine, uric acid constitutes nine-tenths, the remaining tenth consisting of the xanthin bases. The total daily excretion of uric acid varies, under normal conditions, from 0.2 to 1.5 grammes (3 to 22 grains).

The purin bodies found in the urine may be *exogenous*, i. e., derived from the metabolism of the nucleins and xanthins contained in the ingested food; or *endogenous*, resulting from the metabolism of the nuclein-containing leucocytes and other cells of the body. Both exogenous and endogenous purins are practically waste products on their way to final elimination. About one-half of the purin bodies are oxidized and cast off as uric acid; the remaining moiety, through the metabolic agency of the liver cells, is excreted as urea, or as bodies intermediate between urea and uric acid.

Among the food stuffs muscle and gland tissue contain approximately equal quantities of free purins. There is no marked difference between red and white meat in this respect. Glands, however, e. g., sweetbreads (thymus gland) afford large amounts of bound purins (nucleins), while muscular tissue (meat) possesses but little.

Among vegetable foods, beans, peas, oatmeal, onions, and asparagus contain purin bodies. Coffee and tea have among their constituents the methyl xanthins (caffeine, theobromine). Most malt liquors (beer, porter) contain purin bodies; wines and spirits do not. If a diet, *without purin content*, is desired it should number as its principal items, milk, cheese, butter, and eggs.

In health both exogenous and endogenous purins are rapidly excreted; in gout, except during an acute attack when there is some retention, the endogenous purins are eliminated as in health, but the exogenous or food purins are cast off so slowly that there is an excess of purins in the blood.

Futcher, in a series of observations in cases of gout, found that with rare exceptions the excretion of uric acid was below 0.4 gme. for the days preceding the onset of acute symptoms. On the second or third day of the attack the output reached 0.8 to 1.9 gme., or more.

Late investigations, using modern methods, have rendered doubtful the theories that gout and its related conditions are due to the retention and presence in excess in the blood (uricacidæmia) of uric acid, *per se*; and that, by reason of decreased alkalinity of the blood, the urates are deposited in the tissues. This view, promulgated by Garrod and advocated with single-minded enthusiasm by Haig, is simple and attractive. But it has been proved that in gout the blood does not necessarily contain an excess of uric acid, nor is its alkalinity decreased; while certain conditions, attended by an enormous formation of uric acid (leucæmia in particular), do not present gouty manifestations.

Regarding the relation of uric acid to disease, Hall says that it "is a necessary result of normal nuclein metabolism. In disease it is symptomatic of conditions which hinder or prevent its solubility and excretion, and does not itself cause the lesions which accompany uricacidæmia." It must be confessed that we are still in the dark as to the real nature of gout and its associated conditions.

The clinical relations of an excess or a diminution of uric acid are as follows:

(*a*) **Increased Uric Acid.**—As previously stated, the uric-acid output may be increased by special articles of diet, including fat and sugar, and by muscular exercise; so also by Turkish baths. It is quite constantly increased in the acute fevers (e. g., pneumonia, typhoid fever, acute rheumatic fever), and less frequently in digestive disorders. In leucæmia, because of the large destruction of the nuclein contained in the greatly increased number of leucocytes, the daily output of uric acid may amount to 4 grammes (60 grains); so also with splenic diseases in general. During and directly after acute gouty attacks uric-acid elimination is increased. In some cases of diabetes sugar is replaced from time to time by an excessive excretion (3 grammes, 45 grains) of uric acid. In the so-called lithæmic or uric-acid diathesis the output of uric acid is increased, but whether the uric acid is the sole cause of the "lithæmic" symptoms is questionable.

(*b*) **Decreased Uric Acid.**—Under a strict milk diet the amount of uric acid decreases. In chronic gout the elimination is low, increasing, as previously stated, during and after the acute outbreaks. It is also low in most diabetics, in anæmia and chlorosis, chronic plumbism, and chronic interstitial nephritis.

It is hardly necessary to remind the reader that a deposit of uric acid or urates, which occurs mainly when the urine is highly acid, does not necessarily imply an increased excretion.

(9) **Albumin.**—Several albumins may be met with in the urine, of which serum albumin and serum globulin are of the most clinical importance. The term "albuminuria" is understood as indicating the presence of one or both, usually both, in the urine. Other proteids which may occur are albumoses ("peptones"), nucleo-albumin, and fibrin, but these have relatively little diagnostic value.

Albumin can hardly be considered under any circumstances as a normal constituent of the urine, although the existence of a "physiological" albuminuria is claimed by some authorities. There is no doubt, however, that an albuminuria occurs very frequently in which the organic changes in the kidney, if there be such, are so slight and evanescent as to be unimportant. This form of albuminuria, compared with that caused by serious organic lesions, may properly be termed functional, although not physiological. Probably the presence of albumin in the urine always implies some interference with the integrity of the epithelial cells of the glomeruli or of the renal tubules, which epithelium when intact prevents the passage of the proteids of the blood into the urine. Whether the disturbance is insignificant and transient (renal anæmia or hyperæmia, irritating substances in the blood) or great and permanent (chronic nephritis), must be determined by the associated signs and symptoms (presence or absence of fever, cardiac hypertrophy, increased arterial tension) and the presence or absence of other urinary abnormalities (blood, pus, casts, etc.). The not uncommon snap judgment that Bright's disease exists because albumin is found in the urine is not justified by the facts.

Albumin, when found in the urine, may vary from a mere trace up to an amount which causes the urine to boil almost solid. The actual amounts eliminated in 24 hours run from 2 grammes (30 grains) up to 12 grammes (180 grains). Very exceptionally even larger quantities are encountered.

The principal causes and varieties of albuminuria are as follows:

(*a*) **Febrile Albuminuria.**—In any disease attended by fever there may be a slight albuminuria, unattended by evidences of acute nephritis. It becomes manifest at the height of the fever and disappears when the temperature returns to the normal. It occurs particularly in typhoid fever, pneumonia, cerebro-spinal meningitis, acute rheumatic fever, scarlet fever, diphtheria, malaria, and erysipelas. It is probably largely dependent upon the intensity of the infective process.

(*b*) **Albuminuria Due to Blood Changes or the Presence of Certain Abnormal Substances in the Circulation.**—A slight amount of albumin may appear in the urine as a consequence of the blood changes in scurvy, purpura, leucæmia, severe or pernicious anæmia; or of abnormal ingredients in the circulation, as in jaundice, diabetes, syphilis, poisoning with lead or mercury, ether or chloroform anæsthesia, and the ingestion of various drugs, such as turpentine, cantharides, carbolic acid, and other similarly irritating substances. In certain cases in which excessive amounts of uric acid, oxalic acid, and urea are eliminated, albumin may also be present in small quantity.

(*c*) **Albuminuria Due to Disturbances of the Circulation.**—A moderate albuminuria may occur in diseases or conditions which cause a passive renal hyperæmia—e. g., cardiac valvular disease or pulmonary disease giving rise to an increased pressure in the right heart and a consequent hindrance to the flow of blood from the kidney by way of the renal veins; or pressure upon the latter by a tumour or a gravid uterus.

(*d*) **Neurotic Albuminuria.**—Albumin may be discovered in small quantity, usually not persistent, in apoplexy, tetanus, migraine, delirium tremens, exophthalmic goitre, and head injuries. It may be present after an epileptic paroxysm.

(*e*) **Functional Albuminuria.**—Albumin may be found, usually but not always in small quantities, in the urine of young persons, particularly in boys. The albumin may appear intermittently at irregular intervals of days or weeks, lasting for similar periods; or it may become manifest at regular intervals and last for a definite time, as in the cases where it is present at the end of the day and absent in the morning (cyclic albuminuria); or appear after a hearty proteid meal (dietetic albuminuria); or following a cold bath or severe exercise (transitory albuminuria). A large proportion of these cases recover in time, but in some the albuminuria persists, the pulse tension is increased, and organic changes develop.

Albumin may appear in children with spinal deformities, especially lordosis. This *orthotic* type disappears with correction of the deformity.

(*f*) **Albuminuria Due to Organic Renal Disease.**—Albuminuria occurs as a regular symptom in acute nephritis, and in the active hyperæmia which constitutes the early stage of the inflammatory changes. It is also constantly present, often in large quantity, as an evidence of chronic parenchymatous nephritis. In chronic interstitial nephritis albumin may be absent, or present in such limited amount that the most delicate methods are required for its demonstration; but in the majority of cases a trace at least can be found by the ordinary tests. In amyloid and fatty degeneration, particularly the former, albumin is rarely absent, and may be discovered in considerable quantity; so also with neoplasms of the kidney. Pyelitis or suppurative nephritis may be responsible for the presence of albumin derived from the pus which the urine contains. With reference to the relative preponderance of serum albumin and serum globulin in disease it is claimed by Senator that in amyloid degeneration of the kidney the serum globulin may equal or exceed the serum albumin in many cases, and that this relation constitutes an important diagnostic symptom. In other ailments when serum albumin is present, globulin is also found, but in very small quantities.

(*g*) **Extra-renal Albuminuria.**—The urine may be free from albumin when secreted, but become mixed with some albuminous material (pus, blood, chyle, lymph) at some point between the renal tubules and the bladder. Pyelitis may perhaps come under this head. Other causes are, in both sexes, inflammation of the ureters, cystitis, urethritis, simple or gonorrhœal; in men, the presence of semen in the urethra;

in women, menstrual blood, vaginal discharges (including fistulous communication with an abscess), or the presence of semen in the vagina. In such cases only a trace of albumin is usually found. In order to determine the origin of the albumin under such circumstances it may be necessary (especially in women) to obtain a specimen of urine by catheterization of the bladder, or perhaps of the ureter.

(*h*) **Albumosuria.**—It was formerly supposed that true peptones occurred in the urine (peptonuria), but later investigations have shown that the substances found are propeptones or albumoses, and the condition is therefore an albumosuria. The diagnostic value of albumosuria has not as yet been definitely decided. Traces of albumoses have been found in many of the specific infectious fevers in which bacterial disintegration of the tissues is in progress, such as lobar pneumonia, typhoid fever, scarlet fever, measles, and the majority of septic inflammations. Albumosuria is also quite constant in cases where considerable pus accumulations exist, as in empyema or large abscesses in the liver or elsewhere ; in suppurative meningitis; and chronic suppurations in general. A large albumosuria is rather significant of multiple myelomata of the bones (Bence-Jones type), and has been found in osteomalacia and some cases of nephritis.

(*i*) **Nucleo-albuminuria.**—The clinical significance of the presence of nucleo-albumin in the urine is probably slight. When present, it is indicative of catarrhal or desquamative processes somewhere in the urinary tract. It may be found in connection with functional albuminuria, febrile albuminuria, acute nephritis, choluria (bile in the urine), and cystitis; also in leucæmia. Considerable amounts are occasionally encountered during the administration of large doses of certain drugs, particularly bichloride of mercury, arsenic, and naphthol.

(*j*) **Fibrinuria.**—The formation of a reddish or almost colourless sticky sediment or coagulum, or, if much fibrin is present, the conversion of the urine into a jellylike mass upon standing, is a very rare occurrence. It is due to the presence of fibrinogen and a ferment capable of forming fibrin. It is seen in certain cases where the plasma of the blood enters some portion of the urinary tract, as in chyluria, croupous inflammation of the tract, villous growths in the bladder, and occasionally after the ingestion of cantharides. The term fibrinuria does not apply to the presence of ordinary blood clots, the latter coming properly under the head of hæmaturia.

10. **Blood and its Compounds in the Urine.**—Under certain circumstances blood itself may appear in the urine (hæmaturia) or be represented by some form of blood pigment (hæmoglobinuria).

The distinction rests mainly upon a microscopic examination. If blood as a whole is present, red corpuscles will be found more or less abundantly; but if the smoky or reddish colour of the urine is due to pigment, red corpuscles are absent, or found in very small numbers.

(*a*) **Hæmaturia.**—In slight hæmaturia the colour of the urine may be unaltered, but red corpuscles will be discovered by the microscope. In the severer grades of hæmaturia the urine is smoky, red, brown, or even black, and deposits a heavy sediment which may contain blood coagula. According to the length of time during which the blood has been in the urine the red cells may retain a nearly normal appearance, or, especially in ammoniacal urine, because of the hæmoglobin having been dissolved out, may be simply colourless, shadowy, or transparent circles. The blood may come from the kidneys or their pelves, the ureters, the bladder, or the urethra.

Renal hæmaturia is most commonly due to acute congestion of the kidney or acute nephritis, and in a moderate degree to chronic nephritis. The number of red cells found in the latter may be taken as a gauge of the severity of the disease. It occurs also in the malignant types of the acute specific infections such as scarlet fever, smallpox, and malaria. In the latter disease it may become epidemic. Bloody urine may be voided in leucæmia, hæmophilia, purpura, and scurvy. It has been observed in filariasis and distomiasis, and in cancer, tuberculosis, and abscess of the kidney. Aneurism of the renal artery, hydronephrosis, renal infarctions, and thrombosis of the renal vein are rare causes; and poisoning by cantharides, turpentine, or carbolic acid is usually attended by hæmaturia. A stone in the kidney frequently causes blood in the urine; so also does traumatism (rupture) of the kidney. *Ureteral* hæmaturia may be due to the passage of a calculus; *vesical* hæmaturia to the presence of a stone, diphtheritic cystitis, ulceration or cancer of the bladder, injury, ruptured veins, or the presence of parasites; *urethral* hæmaturia to impacted calculus, gonorrhœa, or instrumentation. Finally, blood may appear without apparent cause, renal "epistaxis" or "hæmophilia." These cases occur in neurasthenic or hysterical individuals. A rare congenital hereditary form is described (GRAHAM).

The determination of the *source of the blood* is oftentimes difficult. In renal hæmaturia the blood is intimately mixed with the urine, individual red cells are usually decolourized, and blood casts are found. If the blood is from the pelvis of the kidney, or the ureter, coagula corresponding in shape to the pelvis, or the ureter, may be found. It may be necessary to employ ureteral catheterization. In hemorrhage from the bladder, the blood and the urine are not so distinctly blended, the clots may be of such an irregular form and large size as to indicate their origin, and the individual cells retain their normal appearance. The cystoscope may be required to determine this point. If the blood is from the urethra it ordinarily drips away spontaneously, and if the urine is voided the last portion is clear.

(*b*) **Blood Pigment in the Urine.**—The blood-colouring matters found in the urine are hæmoglobin, methæmoglobin, and hæmatoporphyrin.

Hæmoglobinuria embraces not only the presence of hæmoglobin, but also an oxidation product of the latter—methæmoglobin—which may be formed in acid urine after standing for some time, and in many instances is present when the urine is voided. Hæmoglobinuria is indeed usually a methæmoglobinuria. This condition was of old mistakenly called hæmatinuria. Hæmoglobinuria occurs when there is such an extensive disintegration of red cells that the liver is unable to transform the whole of the liberated hæmoglobin into bilirubin, and the excess escapes by way of the kidneys. There are none, or but few, of the red cells to be found in the urine.

In the large majority of instances hæmoglobinuria is due to some toxic material in the circulating blood, such as arseniuretted or sulphuretted hydrogen, potassium chlorate, carbolic acid, naphthol, muscarine, and carbon monoxide; as well as to the specific toxines of typhoid fever, scarlet fever, yellow fever, the grave form of jaundice, syphilis, and malarial fever, especially, if not solely, the æstivo-autumnal form. Quinine may cause it, but apparently only in those who are, or who have recently been, the subjects of malarial fever. It follows transfusion of blood from one mammal into another, and has been observed as a sequence of severe burns and sunstroke. It has been noted in leucæmia associated with cholæmia. There is a rare epidemic hæmoglobinuria of the newborn in which cyanosis, jaundice, and symptoms referable to the nervous system are present.

As a rarity a paroxysmal hæmoglobinuria has been observed. Each attack is preceded by chill and fever, closely resembling a malarial paroxysm, followed by the passage of urine containing blood pigment. The attack lasts from a few hours to two days, and occurs in connection with syphilis, Raynaud's disease complicated with epileptic seizures, exposure to cold, and muscular overexertion.

Hæmatoporphyrinuria—the presence of iron-free hæmatin in the urine—is of little clinical significance. The urine is of a port-wine colour and darkens on standing. Traces of this pigment occur in normal urine, and may be found in large quantities as a result of the continued taking of sulphonal or trional, especially in women. Its appearance is an indication for the prompt stoppage of the drug and the administration of alkalies. It has been found also in exophthalmic goitre, acute rheumatism, pleurisy or pericarditis with effusion, pneumonia, pulmonary tuberculosis, cirrhosis of the liver, and intestinal hemorrhages.

11. **Alkaptonuria.**—The urine is of normal colour when voided but darkens upon standing. It contains a substance termed alkapton (glycosuric acid or related oxyacids), which has no apparent clinical significance aside from the fact that it may give the glucose reaction with Trommer's or Fehling's test.

12. **Sugars in the Urine.**—The only sugars of clinical importance found in the urine are glucose, and, in a few cases, lactose. Rarely maltose, levulose, inosite, and the pentoses may be found, but their presence has little if any significance.

(*a*) **Glycosuria.**—Although the presence of glucose in the urine is a cardinal symptom of diabetes mellitus, glycosuria and diabetes are not synonymous terms. Thus glucose may appear temporarily in the urine of any healthy person after the ingestion of a sufficiently large amount of sugar or starch, the power of assimilation of such substances having been exceeded (alimentary or digestive glycosuria). A temporary glycosuria may occur, especially during convalescence, in acute febrile maladies, such as typhoid fever, scarlet fever, measles, pneumonia, acute rheumatic fever, epidemic influenza, diphtheria, and malaria; and in various injuries and diseases of the nervous system. A transient glycosuria may occur in stout persons.

Before a diagnosis of diabetes mellitus can be made it must be determined that the elimination of glucose is persistent, covering a period of weeks, months, or years, although there may be periods during which no sugar appears in the urine. If glucose is absent in suspected cases the dietetic test may be employed. This consists in giving 100 grammes (3¼ oz.) of pure glucose and examining the urine three or four hours after. If notable quantities are found, a pathological condition unquestionably exists, as the body in health is able to assimilate this amount of carbohydrate.

(*b*) **Lactosuria.**—Lactose is occasionally discovered in the urine in early, somewhat more often in late, pregnancy; or during the period of lactation. Its presence is due to the resorption of lactose from the milk in the breasts, which occurs especially when for any reason the formation of milk is beyond the demand, or when any obstruction exists (mastitis) to its free outflow through the nipple. The finding of lactose in the urine has been considered presumptive evidence of the good qualities of the mother as a wet nurse. Two cases seen by me several years ago gave an impression that the presence of a considerable amount of lactose in the circulating blood may cause a peculiar nervous unrest, muscular weakness, and a tendency to syncopal attacks.

13. **Acetone.**—Traces (8 to 27 milligrammes per day) of this substance are found in normal urine. In all probability it is derived from the fats. If carbohydrates are eliminated from the diet there is a marked increase in the amount of acetone.

Acetonuria is therefore found in states of inanition, and in fevers, especially typhoid fever, pneumonia, scarlet fever, measles, acute rheumatic fever, acute miliary tuberculosis, and septicæmic conditions, but is much less apt to occur if the dietary allows sugar and starch. It is also found in the cancerous cachexia, especially carcinoma of the stomach; in certain mental and nervous diseases, as in melancholia and locomotor ataxia; in autointoxication of intestinal origin; and after chloroform anæsthesia. The acetonuria associated with diabetes is of more clinical importance than in the conditions mentioned. The simultaneous finding of glucose and acetone in the urine is good proof of the existence of diabetes, and the larger the amount of acetone the more severe is the disease. In the treatment of diabetes it has been suggested that sugar or starch should be freely given if, with unpleasant symptoms, the amount of acetone is found to be greatly increased.

14. **Diacetic Acid.**—The presence of this substance in the urine (diaceturia) has exactly the same meaning as that of acetone, as it occurs under the same circumstances.

15. **Oxybutyric Acid.**—It is regarded as probable by competent observers that β-oxybutyric acid is the exciting cause of diabetic coma, but this point is still in dispute. Be that as it may, the finding of β-oxybutyric acid in the urine of a case of diabetes indicates a grave type of the disease, and if considerable amounts of acetone and diacetic acid are also present it is probable that diabetic coma is impending. Oxybutyric acid is a product of the breaking down of albuminous material.

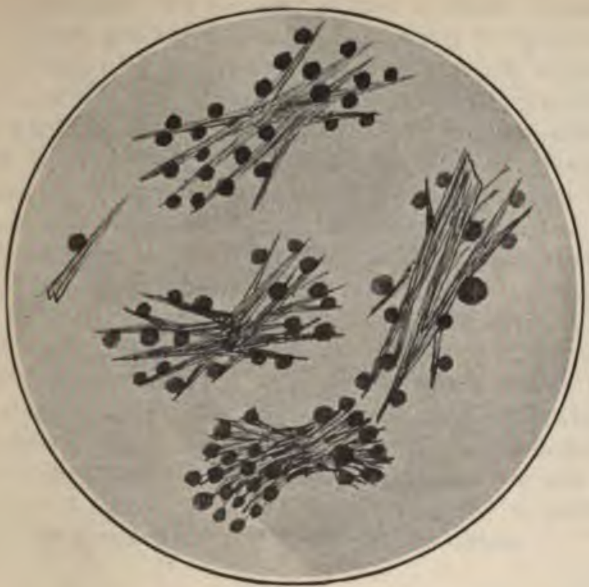
16. **Lipaciduria.**—This is a condition in which certain fatty acids (formic, acetic, butyric, propionic) are found in the urine. It has been observed in diabetes, leucæmia, and certain fevers, as well as in some diseases of the hepatic parenchyma. Lipaciduria has no diagnostic value.

17. **Cystin in the Urine.**—This rare finding has little clinical significance. Cystinuria is associated with the presence of certain diamines, which, with the cystin, appear to be due to a special form of intestinal putrefaction. The condition is in some instances hereditary, and may result in the formation of a cystin calculus.

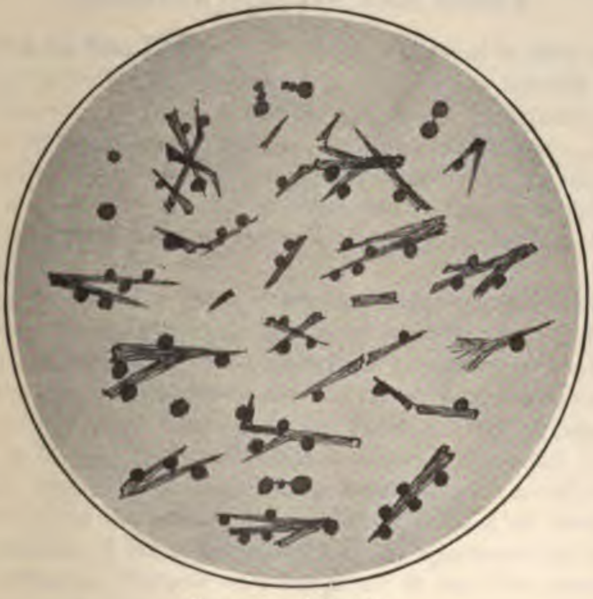
18. **The Diazo-reaction of Ehrlich.**—This reaction may be found at times in pneumonia, scarlet fever, malaria, variola, measles, septic conditions, and advanced malignant disease. The diazo-reaction is of some clinical importance in *typhoid fever*. If in a doubtful case the reaction is found between the 5th and 13th day of the disease and not later than the 22d day, it is probable that the disease is typhoid fever. In acute miliary tuberculosis the reaction generally does not appear until the beginning of the 3d week.

19. **The "Cammidge" Reaction.—Diagnostic Value.**—Pentose has long been known occasionally to be present in the urine, but hitherto no clinical importance has been attached to its presence or its absence. The pancreas is undoubtedly much richer in pentose than is any other organ of the body. It therefore seems reasonable that in pancreatic disease some form of pentose should appear in the urine. Cammidge has succeeded in obtaining a reaction in the urine which he believes is due to a precursor of pentose, and finding this reaction only in the urine of those afflicted with pancreatic disease, he considers this reaction to be a great aid in the diagnosis of these obscure conditions. Some clinicians claim that it is present in only 80 per cent of pancreatic disease; others that it appears when there is no disease of the pancreas. The most that can be said at the present time is that a positive reaction is exceedingly suggestive, and that a negative reaction throws considerable doubt on the diagnosis of a pancreatic lesion.

Technic.—At first Cammidge described two elaborate tests, but has recently worked out a simpler and more definite reaction—the "C," or combined reaction. This is as follows: The mixed morning and evening urine is carefully tested for albumin and sugar, which if present are removed; the former by thorough boiling and filtration, and the latter by fermentation with a pure culture of yeast, such as *saccharomyces apiculatus*, an organism that does not affect the pentoses. When free from these bodies, the urine, always of slightly acid reaction, is filtered, and 40 c.c. treated with 2 c.c. of HCl of a specific gravity of 1.16. This acidified urine is then boiled for 10 minutes in a small flask, the mouth of which is partially closed with a small funnel; this acts as a condenser. The specimen is cooled, made up again to 40 c.c. with *aq. dest.*, and to it is slowly added 8 grams of lead carbonate to neutralize the excess of acid. When effervescence has subsided and the solution cooled, the precipitate is removed by careful filtration. To the clear filtrate 8 grams of tribasic lead acetate is added and time allowed for a thorough reaction. The glycuronates are separated from the pentoses by the boiling with HCl, and are precipitated by the lead acetate. This mixture is repeatedly filtered



Perfect crystals.



Fragments of crystals.

FIG. 263.--A positive "Cammidge" reaction. Crystals bright yellow; spheres bright red. Fragile. With $\frac{1}{12}$ objective and enlarged $\frac{1}{2}$. (M. R. Barker.)

until entirely clear. To the clear filtrate is added 4 grams of dry sodium sulphate, and the mixture brought slowly to the boiling point. The lead is thus precipitated, and is to be removed by filtration.

Ten c.c. of this clear filtrate are taken and made up to 17 c.c. with distilled water; to this is added 1 c.c. of 50 per cent acetic acid, 2 grams of dried sodium acetate, and $\frac{8}{10}$ grams of phenylhydrazin hydrochlorate. This mixture is gently boiled, on a sand bath, in a small flask with funnel condenser, for about ten minutes; or, better still, until reduced to 15 c.c. A filter paper is made hot by boiling water, and through this hot paper the final mixture is poured. If not equal to 15 c.c. it is made up to that amount with water.

If the reaction is positive, in a few minutes to 12 hours fine, yellowish, acicular crystals (Fig. 263) appear, either enough to make a distinct mass, or only a few discovered by microscopic examination. Phenylhydrazin may form such crystals with other than pentose, but the latter body affords crystals which dissolve in 5 per cent sulphuric acid in $\frac{1}{2}$ to 3 minutes. This differentiation test is performed by running a few drops of the acid under the cover-glass.

III. EVIDENCE FROM THE MICROSCOPICAL EXAMINATION OF THE URINE

(1) Fat in the Urine.—When fat is present in such quantities that it is practicable to recognise it as such by the unaided eye, it is termed *lipuria.* Ordinarily the fat is present in minute droplets, which are recognised only by microscopical examination. If outside contamination (oily containers, oil-lubricated catheters, addition of milk by malingerers) can be excluded, fat in the urine may be due to the ingestion of excessive amounts of fat (e. g., fat meat, cod-liver oil), or oil inunctions.

It may be found in cases of fracture involving the bone marrow and causing fat embolism; in the fatty degeneration of phosphorus poisoning; in long-continued suppurative processes (pyæmia, phthisis); in the lipæmia of diabetes mellitus; in obesity, leucæmia, chronic alcoholism, and some diseases of the heart and the pancreas. The fat may be derived from fatty degeneration of the renal epithelium in chronic nephritis, of pus cells in pyonephrosis, or of neoplasms somewhere along the urinary tract. If the urine is so crowded with minute particles of fat as to present a milky appearance to the naked eye, it constitutes *chyluria* or galacturia. It may clot more or less extensively, and contain leucocytes, red corpuscles in sufficient numbers to colour the coagulum, and albumin. Chyluria may be, and usually is, of parasitic origin, due to the presence of the *Filaria san-*

guinis hominis; but in rare instances the parasites are not found and the etiology is obscure.

(2) **Pus in the Urine.**—*Pyuria* is found as a very important symptom in a number of affections of the urinary tract, from the kidney to the end of the urethra inclusive. It occurs also in some instances as an evidence of disease in neighbouring organs. The amount of pus varies from a moderate increase in the number of leucocytes normally found in the urine and to be perceived only by microscopical examination, to a quantity which, when allowed to settle, will form a deposit an inch or more in depth. In neutral and acid urines the form and character of the pus cells may be well preserved, but in strongly alkaline urine they are swollen and perhaps completely disintegrated. If an old abscess has ruptured into the urinary passage nothing but granular or fatty detritus may be found.

When pus is discovered in the urine the question immediately arises as to its source. Not infrequently the determination of the condition giving rise to the pyuria is extremely difficult, but in the majority of cases attention to the following points, together with a consideration of the history and associated symptoms, will enable a satisfactory answer. It may be stated in general that the largest amounts of pus come from an inflamed bladder, or from the rupture of an abscess into the urinary tract, although very considerable quantities may be discharged from the kidney. Urine containing pus from the kidney, or from an outside source, is usually acid; from the bladder, more or less strongly alkaline. It is of course alkaline if pyelitis and cystitis coexist. If the pus appears and disappears rather suddenly, or varies notably in quantity at different times, either its renal or its outside origin may be suspected.

(*a*) **Urethritis.**—If the pyuria is due to urethritis (simple or gonorrhœal), a drop of pus may be squeezed from the urethra, and if the two-glass test is employed, the first portion of urine voided contains pus, while the second is clear. If the second portion also contains pus in even larger quantity and is alkaline, the presence of a coexisting cystitis may be inferred. A gonorrhœal ulcer, or a suburethral abscess (especially in women), may also be responsible for pyuria. These conditions are to be discovered by the urethroscope.

(*b*) **Cystitis.**—If the urine is alkaline (frequently offensive) and the pus, which is often gelatinous and ropy, issues with the last portion of the urine, the pyuria is due to cystitis. The cystoscope may be employed to confirm the diagnosis, and the sound to discover a possible calculus.

(*c*) **Ureteritis.**—The nature of a pyuria due to a tuberculous or gonorrhœal stricture at the lower, middle, or upper part of the ureter, above which the pus may collect, is to be determined only by ureteral catheterization.

(*d*) **Pyelitis or Pyelonephritis.**—The continuous but remittent presence of pus in an acid urine suggests tuberculous, calculous, or obstructive pyelitis. If the pus flows intermittently it is more likely to be caused by suppurative or surgical kidney with abscesses of considerable size. A coexisting cystitis causes the urine to assume the cystitic type, (*b*) preceding, but also suggests the possibility of an ascending renal infection. Ureteral catheterization may determine beyond doubt the presence or absence of pyelitis.

(*e*) **Outside Sources of Pyuria.**—Certain suppurative foci may rupture into the urinary tract, almost invariably into the bladder, and utilize it for drainage. Pyuria of this kind is most frequently due to salpingitis, simple or tuberculous, but may also arise from an abscess of the ovary or extra-uterine pregnancy, suppurating ovarian or dermoid cyst, and psoas or acetabular abscess connected with disease of the vertebra or hip joint. A vesico-intestinal fistula or malignant disease involving the bladder by contiguity may also be classed under this head.

A bacteriological examination of the pus may afford valuable evidence by revealing the gonococcus, the tubercle bacillus, colon bacillus, or the bacillus of typhoid fever, as well as the ordinary pyogenic organisms.

(3) **Red Blood Corpuscles.**—See Hæmaturia, page 696.

(4) **Epithelial Cells.**—Under normal circumstances only a small number of epithelial cells are found in the urine, representing the slight physiological desquamation constantly occurring in the whole length of the urinary tract. Large quantities of epithelium when found in urinary sediments are indicative of some pathological condition (disturbances of circulation or inflammatory changes) affecting the kidney or the urinary passages. Unhappily, the place of origin of the cells, and in consequence the seat of the disease, can not be inferred from the character of the cells, except in rare instances, although in conjunction with the history and the associated signs and symptoms the cell findings may afford valuable evidence as to the nature and location of the morbid process. The uncertainty as to the place of origin of the epithelial elements arises largely from the fact that similar varieties of epithelium are found in widely separated parts of the urinary tract. Thus a certain variety of epithelium when derived from the superficial surface of the mucous membrane lining a definite part of the urinary tract may be quite characteristic of that particular area, while the cells of the deeper layers of the membrane at the same point may exactly resemble the

superficial cells of other portions of the tract. If, therefore, as happens in all but slight pathological processes, there is a more or less extensive desquamation, one is apt to find a deposit of the mixed varieties. Moreover, by their sojourn in the urine if it be alkaline, the exfoliated cellular elements tend to become swollen, large, and round, thus interfering with the characters by which they might otherwise have been recognised.

There are three varieties of epithelium the recognition of which is serviceable, i. e., *round*, *caudate*, and *flat*.

(*a*) **Round Cells.**—These cells are usually a little larger than leucocytes, but may be of the same size. They are distinguished by a large and distinct single nucleus which is visible without the aid of acetic acid. They originate mainly from the uriniferous tubules, the deep layers of the mucous membrane of the pelvis of the kidney, the bladder, and the male urethra. If albumin is present, or if casts are found to which some of the round cells are clinging, the renal origin of the latter may be inferred. An unusually large number of well-preserved and typical round cells occurring under such circumstances is indicative of an acute diffuse nephritis; while if the cells are fatty, markedly granular, or partly disintegrated, the existence of a chronic diffuse process is more probable. If, with a large number of round cells, pus is present, without casts or more than a trace of albumin, a pyelitis may be strongly suspected.

(*b*) **Caudate Cells.**—Conical, columnar, spindle-shaped, cylindrical, or "tailed" epithelial cells originate from the superficial layer of the mucous membrane of the pelvis of the kidney and the deep layers of the vesical mucous membrane. The cells from the latter location are said to have longer "tails" or processes by which it may be possible to identify them. Although by no means characteristic, tailed cells are usually abundant in cases of pyelitis.

(*c*) **Flat Cells.**—Large squamous or pavement epithelial cells are usually derived from the bladder or the vagina. The vaginal cells are usually larger than those from the bladder, and are very commonly grouped. An unusual abundance of flat vesical epithelial cells is indicative of cystitis, and if, in addition, many columnar cells are present, the cystitis is probably of a severe grade, involving the deeper layers of the vesical mucosa. If in a woman there is doubt as to whether a vaginitis and not a cystitis is the source of the squamous epithelium, the vulva should be cleansed and a catheter specimen of urine obtained.

(5) **Tube Casts in the Urine.**—Urinary tube casts are perhaps the most important of all findings during an examination of the urine. In at least the large majority of cases when casts are dis-

covered albumin is present, although it may be in such small amount as only to be discovered by the more delicate tests. There are three main divisions of tube casts: *First*, those composed of cellular elements or micro-organisms, viz., red corpuscles, leucocytes, epithelial cells, or bacteria; *second*, casts of degenerated cellular elements and unorganized protoplasm—granular casts; *third*, casts composed of a homogeneous, hyaline material—hyaline and waxy casts.

Judging from personal experience, it seems probable that casts are found more frequently in sediments obtained by centrifugation than in those which result from standing. Certainly, in my own practice of late years, the centrifugal apparatus having been systematically employed, casts, especially of the hyaline variety, have been reported as occasionally present in a much larger proportion of urines than was formerly the case. This fact emphasizes the truism that the presence of two or three hyaline casts, like that of a trace of albumin, unless found in repeated examinations, does not, *per se*, necessarily imply permanent renal disease.

(*a*) **Epithelial Tube Casts.**—These occur as hollow or solid cylinders composed entirely of epithelial cells; or the cells are adherent to the surface of a cylindrical hyaline matrix. The cells may be granular or fatty. When found, such casts always signify a desquamative nephritis, the epithelial lining of the renal tubules having been partly or entirely exfoliated. Granular or fatty degeneration of the cells argues in favour of a chronic process.

(*b*) **Blood Casts.**—Casts composed of red corpuscles bound together by a fibrin network are not often seen, as they are apt to be obscured by the considerable number of free corpuscles by which they are usually accompanied. Such casts are encountered in acute renal hyperæmia, acute nephritis, hemorrhagic infarction, and renal hæmaturia. When found in a case of hæmaturia their presence furnishes indubitable proof that the blood originates from the kidney.

(*c*) **Pus Casts.**—Very seldom casts are seen which are formed of leucocytes alone. When found they are indicative of a multiple suppurative nephritis. Small numbers of leucocytes adhering to other varieties of casts, particularly the hyaline form, are not infrequently seen in the non-suppurative nephritides. Plugs or balls of pus are significant of pyelitis.

(*d*) **Bacterial Casts.**—Casts proved to consist of bacterial masses (micrococci) moulded into cylinders in the urinary tubules are sometimes discovered. Their presence is significant of pyelonephritis in which the infection has travelled upward through the ureters to the renal pelvis, or suppurative nephritis due to the lodgment of an infective embolus from some distant septic focus.

(*e*) **Granular Casts.**—Finely or coarsely granular casts, often fragmentary, resulting from degenerative changes affecting the epithelium, red corpuscles, leucocytes, or hyaline material of which they were originally composed, are frequently found. If such casts are present it is safe to infer the existence of a chronic or degenerative process in the kidneys.

(*f*) **Fatty Casts.**—Casts whose surfaces are completely covered with fat droplets or globules are usually significant of subacute or chronic nephritis with fatty degeneration, especially the large white kidney.

(*g*) **Hyaline Casts.**—These are transparent, homogeneous casts, sometimes showing fine pale granules or striations, and not infrequently presenting a few adherent epithelial cells, red cells, or leucocytes. They are the most common of all casts, and are found not only in all inflammations, acute or chronic, of the renal tubules, but when purely hyaline and temporarily present may result from circulatory disturbances without organic changes.

(*h*) **Waxy Casts.**—These casts, regarded by some writers as merely a variety of hyaline cast, are not at all indicative of amyloid (waxy) degeneration of the kidneys. They are of infrequent occurrence, and when present may signify any one of the subacute or chronic diseases of the renal substance.

(*i*) **Cylindroids.**—These structures are probably of renal origin and closely affiliated with hyaline casts. Like the latter, they are indicative of renal irritation and circulatory disturbances, as in oxaluria and excessive elimination of uric acid, but may be present without albumin and unaccompanied by indubitable casts. While their exact significance is a matter of dispute, some writers regarding them with suspicion, it is very likely that their pathological importance as indicating organic changes is minimal. They are found especially in the urine of children.

(6) **Prostatic Threads.**—Threads of mucus, sufficiently large to be easily seen without the aid of the microscope, are significant of prostatic irritation.

(7) **Spermatozoa.**—These are found in the first urine passed by men after emission (coitus, pollutions, following an epileptic convulsion), and in women when the external genitals have been soiled during coitus. They occur in some cases of injury or disease of the spinal cord. Occasionally a small amount of semen is expressed from the vesiculæ seminales by hard fæcal masses, during the severe expulsive efforts accompanying obstinate constipation. The persistent presence of spermatozoa in the urine is the cardinal symptom of true spermatorrhœa.

(8) **Elastic Fibres and Tumour Fragments.**—Elastic fibres when present indicate a severe ulcerative process in the bladder. Very rarely particles of papillomatous or villous growths of the bladder are detached, and appear in the urine as vascular connective tissue formations covered with epithelial cells. In other tumours nothing but disintegrated and unrecognisable material is usually found.

(9) **Parasites.**—The following organisms have been encountered in the urine, either by centrifugation and the preparation of dried and stained cover-glass films, or by cultures.

(*a*) **Pathogenic Bacteria.**—The *typhoid bacillus* is present in the urine at some period in the course of almost all cases of typhoid fever, and its finding constitutes one of the diagnostic tests of the disease. The discovery of the *tubercle bacillus* in the urine is good evidence of tuberculous disease of the urinary organs. The finding of the *gonococcus* may explain the true nature of a supposed rheumatism. While the presence of bacteria in the urine does not necessarily imply renal inflammation, if considerable numbers of the diplococcus of pneumonia, the typhoid bacillus, the *Bacillus coli communis*, or the pyogenic staphylococci and streptococci are discovered in the urine, and the clinical symptoms of a nephritis or pyelonephritis coexist, it may safely be inferred that the inflammation is due to infection by the particular organism found.

(*b*) **Other Parasitic Organisms.**—Of other parasitic forms of life, yeast cells may occur abundantly in urine containing sugar; rarely the ova of distoma hæmatobium and the *Filaria sanguinis hominis* in hæmaturia and chyluria; trichomonas in hæmaturia; also, rarely, echinococcus hooklets and fragments of membrane if a cyst has ruptured into the urinary passages; and very infrequently ascarides, in cases of vesico-rectal fistula.

Before deciding that the urine contains bacteria or other microorganisms, a specimen should be examined directly after voiding, having taken the necessary precautions, especially for cultural tests, of carefully disinfecting the external genitals, and sterilizing containers, and catheters, if the latter are employed.

IV. COLLECTIVE RESULTS OF URINALYSIS IN SPECIAL DISEASES AND CONDITIONS

(1) *Movable Kidney.*—In the majority of movable kidneys the urine presents no abnormality. Occasionally, when the ureter becomes badly strangulated or kinked (Dietl's crises) and its lumen temporarily constricted, the urine is scanty, loaded with uric acid or oxalates, and contains blood, albumin, and epithelial cells. This condition is usually followed by a copious discharge of pale urine.

(2) *Acute or Active Renal Hyperæmia.*—The urine is acid, somewhat decreased in quantity, and contains more or less blood, small amounts of albumin, and a few, usually hyaline, casts.

(3) *Passive or Mechanical Renal Congestion.*—The urine is scanty, acid, of high specific gravity, usually cloudy with urates, and contains at least traces of albumin, with a few small hyaline casts and occasional red corpuscles. It is the typical urine of chronic valvular disease of the heart.

(4) *Acute Diffuse Nephritis.*—The urine is greatly diminished in amount (4 or 5 oz. in 24 hours), or is even totally suppressed (anuria). It is smoky, blackish, or of a chocolate color. The specific gravity is high. Albumin is found in large amount, and the heavy deposit contains abundant red corpuscles, blood, hyaline, and epithelial tube casts. The total urea is lessened.

(5) *Chronic Diffuse Nephritis.*—The quantity of urine is diminished, it is cloudy from urates, the specific gravity may be high in the early, but is low in the later, stages. Albumin is abundant, sometimes more so than in any other disease. The heavy sediment contains large numbers of nearly all the varieties of tube casts, hyaline, epithelial, granular, and fatty. The latter are especially characteristic. Occasional red corpuscles, many leucocytes, and numbers of degenerated epithelial cells are also found. The amount of urea is decreased.

(6) *Chronic Interstitial Nephritis.*—The urine is increased in quantity, light yellow, clear, with a persistently low specific gravity. Albumin is scanty, occurring in traces, and is sometimes absent. A few narrow hyaline casts are almost constantly found in the very small deposit. Cellular elements are as a rule no more abundant than in normal urine. Polyuria, persistent low specific gravity, and the presence of a few hyaline casts constitute the urinary signs of this disease. Albumin may or may not be present in small quantity.

(7) *Amyloid Disease of the Kidney.*—There is polyuria, the urine is clear, pale, and of low specific gravity, with a scanty sediment. There is almost always a characteristically abundant amount of albumin, perhaps with an unusually large proportion of globulin. Moderate numbers of large hyaline, and occasional waxy, casts are found. If casts are numerous a certain proportion are fatty and granular.

(8) *Pyelitis.*—(*a*) In the simpler forms of pyelitis, such as occur in the specific fevers, the urine is cloudy and acid, containing pus cells, young epithelial cells (mucous corpuscles), and sometimes red blood corpuscles. Albumin is present in traces.

(*b*) In varieties involving both pelvis and kidney, the urine is generally acid, at least in the earlier stages and when not complicated by cystitis. The quantity of pus is considerable, and may be formed into plugs or rolled into balls by its passage through the ureter. The pyuria may be intermittent because of a varying degree of obstruction to its exit from the renal pelvis (calculus, kinks, pressure). If the obstruction is long continued, the renal pelvis becomes distended with pus (pyonephrosis). The epithelium may or may not be abundant, and a few casts and red corpuscles may be present. Albumin is found, generally in proportion to the amount of blood and pus.

(*c*) In acute suppurative nephritis the urine is cloudy, containing pus, blood, epithelium, bacteria, and casts. The latter are occasionally composed of pus or bacteria.

(9) *Hydronephrosis.*—When the ureter is partially obstructed and the urine is secreted under pressure, it is of a low specific gravity, and traces of albumin are occasionally present, together with a few red corpuscles and pus cells. Tube casts are not common, but epithelium is abundant.

(10) *Renal Calculus.*—The urine is strongly acid, and contains blood, usually enough to give a smoky tint. Small concretions may be found. Later a calculous pyelitis may be present, and the characters of the urine become those previously described in (*b*), paragraph (8) preceding.

(11) *Renal Cancer.*—Hæmaturia is the most important, and sometimes the first, evidence of the disease. An occasional but characteristic finding is that of blood casts of the renal pelvis and the ureter. Tumour particles are practically never found.

(12) *Renal Tuberculosis.*—The urine presents the features of ordinary pyelitis, pus, blood, albumin, epithelium, and, occasionally, caseous masses. Microscopic examination will show tubercle bacilli, or, if not found by the microscope, they may be demonstrated by culture and inoculation.

(13) *Cystic Kidney.*—The urine resembles that of interstitial nephritis, as previously described in paragraph (6), except that there may be a larger amount of albumin, a decided hæmaturia, and the tube casts found are usually large and granular.

(14) *Renal Embolism.*—The urine becomes suddenly and decidedly albuminous, with moderate hæmaturia, and a little pus. It contains varying numbers of hyaline and epithelial tube casts. During the 3 or 4 weeks (or less) subsequent to the attack the urine steadily regains its normal characters.

(15) *Diabetes Insipidus.*—The quantity of urine is enormously increased (20 to 40 pints), it is pale, and the specific gravity is low (1.002 to 1.007). The total urea output is increased. It does not contain albumin (except in occasional traces), sugar, or casts. Inosite (muscle sugar) is infrequently present.

(16) *Diabetes Mellitus.*—The quantity of urine is greatly in excess of the normal (6 to 40 pints), the specific gravity is usually high (1.030 to 1.045), but exceptionally is low. The urine is pale, acid, has a sweetish odour, and contains from 1 to 10 per cent of glucose. The urea is increased. Acetone, diacetic acid, and β-oxybutyric acid may be present, and traces of albumin are not infrequent.

(17) *Uræmia.*—Although this condition may be due to various diseases of the kidney, two urinary findings are practically constant, without reference to the particular variety of the causative lesion, viz., a considerable reduction in the amount of urea (200 to 100 to 50 grains in 24 hours), and the presence of tube casts.

(18) *Cystitis.*—In acute cystitis the urine is acid and contains blood, pus, albumin, and epithelium. In chronic cystitis the urine is turbid, alkaline, and ammoniacal. It contains a considerable amount of viscid pus, albumin, much epithelium, crystals of triple phosphates, and numerous bacteria (coli communis, staphylococcus, streptococcus). Occasionally blood is present.

(19) *Vesical Calculus.*—Prior to the development of cystitis the urine persistently contains uratic or phosphatic deposits, with a few pus cells and red corpuscles. After the bladder is inflamed, the characters of the urine are as stated in paragraph (18), except that blood may be present in larger amounts.

(20) *Vesical Cancer.*—Marked and increasing hæmaturia, the presence of blood clots, abundant epithelium, and, in rare instances, fragments of the growth in an acid urine, characterize malignant disease of the bladder. When cystitis is initiated the special characters of vesical inflammation (18) supervene.

(21) *Vesical Tuberculosis.*—Blood in small amount, and increasing quantities of pus in an acid urine, followed by the evidences of a chronic cystitis (18), constitute the urinary findings in tuberculosis of the bladder.

SECTION XLIII

DIAGNOSTIC PUNCTURE AND THE EVIDENCE DERIVED THEREFROM

In order to make or verify a diagnosis it is sometimes desirable to ascertain the character of the fluids which may be present in the cavities of the body or in cystic or abscess cavities.

Technic of the Puncture.—A diagnostic or exploratory puncture may be made, in case of necessity, by an ordinary hypodermic syringe, but is better done by using the ordinary aspirator or an exploring syringe. The latter is simply a larger hypodermic, with a longer and stouter needle. The puncture should be made with strict antiseptic precautions. The needle should be boiled (conveniently in a test tube) for 5 to 10 minutes, or put in a steam sterilizer. The skin at the point to be punctured must be scrubbed first with soap and water, then with bichloride solution, finally with alcohol. The hands of the operator should be similarly treated. To minimize the pain of the needle thrust, a few minims of a 2- to 4-per-cent solution of cocaine may be injected subcutaneously; or local anæsthesia secured by pressing for a few moments a bit of ice sprinkled with salt against the selected point, or using an ethyl chloride spray for the same purpose. The needle should then be thrust rapidly but steadily to the desired depth. If a vacuum bottle is in use the stopcock should then be turned, or the piston of the syringe slowly pulled out. If the attempt is unsuccessful, the needle should be slowly entered a little deeper, or gradually withdrawn, while suction is continued. The puncture may be covered with sterilized gauze, or, better, sealed with collodion in which a little bismuth subiodide has been mixed.

The following points of puncture are those commonly employed:

(1) *Pericardial Cavity.*—The puncture may be made either in the fourth interspace, ½ to 1 inch from the left sternal margin; or in the fifth interspace, 1½ inch from the left sternal margin; or if the effusion is large, by entering the needle in the left costoxiphoid

angle, close to the costal margin, and passing it upward and backward.

(2) *Pleural Cavity.*—The puncture may be made either in the seventh interspace in the midaxillary line, or, perhaps better, in the eighth interspace, just outside of the lower angle of the scapula.

(3) *Peritoneal Cavity.*—Ordinarily the puncture is made in the linea alba, midway between umbilicus and symphysis, first making sure that the bladder is empty.

(4) *Lumbar Puncture* (Quincke).—As this is most frequently required to be done in children, general anæsthesia is often necessary. The patient should be placed in the right or left lateral position, bent somewhat forward, back toward the operator. Identify the 12th dorsal spine by means of the last rib, and count downward to the 3d (or 4th) lumbar spinous process. An exploring needle is then entered, about ⅓ inch to right or left of the median line, on a level with the tip of the spinous process, in a direction somewhat upward and inward. It should be introduced slowly to a depth of 2 to 4 centimetres (¾ to 1½ inch) in children, 4 to 8 centimetres (1½ to 3 inches) in adults, before it pierces the membranes and enters the vertebral canal. Unless the pressure is great the cerebro-spinal fluid escapes drop by drop, and not continuously. Aspiration is not required. The fluid should be collected in a test tube. If a bacteriological examination is to be made, the tube should have been sterilized, and is to be plugged with cotton when filled. The procedure is usually free from danger. It is wise to avoid lateral movement of the needle while obtaining the fluid, in order to prevent unnecessary laceration and consequent hemorrhage.

(5) *Puncture of the Liver.*—Exploratory aspiration of the liver may be done by entering the needle either in the seventh interspace in the midaxillary line, or in the lowest interspace in the anterior axillary line, or posteriorly at the central point of the area of hepatic dulness. General anæsthesia is required. This procedure may be essential for diagnosis in abscess or hydatid cyst of the liver, and is unattended by danger. Puncture of the gall bladder is rarely done, as there is danger of leakage from the opening into the peritoneal cavity.

(6) *Cysts and Abscesses.*—These are to be punctured at the most prominent point (e. g., hydronephrosis), or in the center of the dulness caused by the fluid (e. g., localized empyema), having due regard to the presence of important structures which may run a risk of injury.

Examination of the Fluid Obtained.—The fluid having been secured, it is necessary to note its colour, transparency, degree of fluidity, and odour. After taking its specific gravity with an accurate urinometer, a portion may be set aside in a conical glass and allowed to settle, observing subsequently the appearance of a deposit or the formation of a coagulum. Another portion should be filtered, and the filtrate tested with regard to its reaction with litmus paper and the presence of albumin, sugar, mucin, and sometimes of urea.

The presence of albumin is ascertained by the same tests as those used for its detection in urine. Esbach's albuminometer may be employed for its approximate quantitative estimation. The presence of mucin is affirmed by the appearance of a precipitate upon adding acetic acid in excess to a portion of the fluid. The presence and amount of urea (rarely found except in hydronephrosis) may be determined by boiling and filtering the fluid, and, after evaporating the filtrate to a small bulk, testing it as for urea in urine.

A drop of the sediment obtained by standing (or, better, by centrifugation) should be submitted to microscopical examination, with reference to the presence of red corpuscles, leucocytes, epithelial or endothelial cells, hooklets, scolices or fragments of membrane (echinococcus), actinomyces, and the *Amœba coli*. Pathogenic bacteria may be sought for in cover-glass films appropriately stained, or by bacteriological methods.

Cytodiagnosis.—In transudates (see (1) following) there are ordinarily only a few endothelial cells and scattered leucocytes. Cholesterin and Charcot-Leyden crystals have been observed.

In exudates (see (2) following) there will be found erythrocytes if blood is present, endothelium, and more or less abundant leucocytes. On the predominance of one or another of the forms of the leucocytes depends the method of *cytodiagnosis*. Stated in general terms, it is believed that exudates due to chronic tubercular disease, especially chronic exudative pleurisy, present a marked increase of the lymphocytes; while in non-tubercular exudates the polymorphonuclear elements preponderate. Simon puts the percentage of lymphocytes in tubercular pleurisy as ranging from 50 to 98, increasing with the duration of the disease. The other forms of leucocytes are infrequent and apparently do not possess diagnostic importance. Attempts have also been made to apply this method to the diagnosis of malignant neoplasms (see (3) following). While cytodiagnosis has been employed mainly with reference to the pleura, it is equally applicable to effusions into the abdomen and the joints. In arriving at conclusions one must take into consideration the relation of the specific gravity, fibrin, and albumin content, and the variety of leucocytes preponderating.

The Characters of the Fluid According to its Source.—(1) *Dropsical Fluids.*—Fluid effusions obtained in pathological quantities from the closed (serous) cavities of the body may be due on the one hand to passive congestion, as in cardiac valvular disease, or to defective drainage by way of the kidneys, as in nephritis. On the other hand, the accumulation may be the result of inflammation. The former are dropsical fluids or *transudates*, the latter, inflammatory fluids or *exudates*. It is not always easy to distinguish one from the other. Both may be clear, serous fluids of a light yellow or yellowish-green colour, and both contain albumin. If the specific gravity of the fluid is under 1.015, and the percentage of albumin is less than 2.5, it may be definitely inferred that it is a transudate. Transudates are sometimes reddish (sanguineous), very rarely milky (chylous). If a transudate contains blood, clotting on standing may occur, usually to a moderate, sometimes to a considerable, extent.

(2) *Inflammatory Fluids.*—If, on the contrary, the specific gravity is above 1.018, and there is more than 4 per cent of albumin, usually (but not necessarily) with the formation of a considerable coagulum, the fluid is an exudate. As a general rule fluids, whether of inflammatory or non-inflammatory origin, derived from the pleural cavity, are richer in albumin than those obtained from the peritoneal sac.

(3) *Serous, Sero-fibrinous, and Hemorrhagic Fluids.*—It is mainly with these forms of effusion that difficulties may arise in determining whether a given specimen is a transudate or an exudate. The points of distinction have just been described. Under the microscope serous and sero-fibrinous exudates will show small numbers of fattily degenerating endothelial cells, polymorphonuclear leucocytes, and red corpuscles, the latter usually from the puncture. Bloody pleural exudates are, in the majority of cases, due either to tuberculous or carcinomatous disease of the lungs or pleura. The microscope shows, in addition to leucocytes and endothelial cells, large numbers of red corpuscles and, occasionally, crystals of cholesterin. A search for bacilli may be made in suspected tuberculous pleurisy, but they are rarely found. In supposed malignant disease of the pleura a diagnosis often may be made possible by the finding of an unusually large number of epithelial cells exhibiting mitotic figures, many of them atypical (Dock). To recognise these figures, prepare a cover-glass film of the sediment, dry in the air, fix for one hour in equal parts of absolute alcohol and ether, and stain with dilute hæmatoxylin. The discovery of very large epithelial cells, especially if grouped, or of solid particles presenting an alveolar structure, constitutes good evidence of malignancy. The presence of globules of fat (rendering the fluid milky), or grouped crystals of the fatty acids, speaks in favour of carcinoma.

(4) *Sero purulent, Purulent, or Putrid Fluids.*—Fluids possessing the characters indicated by these names are unquestionably exudates or inflammatory effusions. Sero-pus and pus are usually readily recognised. The microscope shows a vast number of leucocytes, mainly of the polymorphonuclear variety, which, if the pus is old, present fatty granules and vacuolations, or are shrunken and crenated. A certain number of red corpuscles are also found. A brown or brownish-green fluid, having an extremely offensive and characteristic odour, is a putrid exudate containing degenerated pus cells and crystals of fatty acids, cholesterin, and hæmatoidin. The finding of a putrid fluid in the pleural sac may indicate the perforation of a subphrenic abscess or a gangrenous spot of lung into the cavity; in the peritoneal cavity, a similar perforation of a gastric or intestinal ulcer; although in either case it may occur as the result of a malignant growth or, more rarely, without an assignable cause.

Pus may be examined for the possible presence of actinomyces, the *Amœba coli*, and various pathogenic bacteria, especially the *Diplococcus pneumoniæ* and the streptococcus.

(5) *Chylous Fluids.*—A turbid milky fluid may be found, most frequently in the peritoneal cavity, less commonly in the pleural cavity, and, as a rare occurrence, in the pericardial sac. The milkiness is almost always due to the presence of fat, sometimes in distinct droplets (chyloid exudate), in other cases in a state of fine molecular division (chylous exudate). A few instances have been reported (LION) in which the turbidity was caused by the presence of numerous albuminous granules. Chylous and chyloid fluids are found in connection with filariasis, cancer of the mesenteric glands, peritoneum, or pleura, and in conditions which lead to the presence of large numbers of fattily degenerating endothelial cells. When found in the peritoneum (chylous ascites) the fluid may have come from a perforation of the thoracic duct. It has been suggested (OSLER) that mild grades of chylous ascites occurring in patients upon a strict milk diet may be due to the excess of fat which is in the blood (lipæmia) under such circumstances.

(6) *Cerebro-spinal Fluid.*—Normally this is a clear, colourless, or slightly yellow, watery fluid, with a specific gravity varying from 1.005 to 1.007. It contains albumin (0.1 per cent), and a substance which reduces Fehling's solution. A coagulum seldom forms. Microscopic examination shows only a few leucocytes and endothelial cells.

If the fluid is cloudy, forms a considerable coagulum, and under the microscope presents large numbers of leucocytes, the existence of a purulent meningitis is assured. The fluid may have a specific gravity as high as 1.012, and may be sero-purulent or even consist of pure pus.

The fluid obtained in cases of brain tumour, as well as in serous and tuberculous meningitis, is normal in appearance and composition, but owing to increased intracranial pressure it flows in much larger quantities, varying from a few to 100 cubic centimetres. If only a few drops can be obtained it is usually safe to exclude the diseases just mentioned.

Microscopical examination of the centrifugalized fluid (by stained cover-glass films) and bacteriological examination by cultures may afford most valuable, and sometimes indispensable, evidence of the nature of a meningitis. Thus may be found the *Diplococcus intracellularis meningitidis* (meningococcus), the organism causing epidemic cerebro-spinal meningitis, the bacillus of tuberculosis, the diplococcus of pneumonia, and the streptococcus of septic meningitis.

(7) *Pancreatic Cyst.*—The fluid obtained from a cyst of the pancreas is watery, clear, colourless, or slightly yellow, of a low specific gravity, and may or may not form a slight coagulum. If its origin from the pancreas is suspected it may be tested with regard to its power of digesting egg albumen. A few small particles of the latter are placed in the fluid, which is usually alkaline, but if not, is rendered so by adding 10 per cent sodium-hydrate solution. After two or three hours a drop of very dilute solution of sulphate of copper is added, and if a reddish violet colour appears (biuret reaction) the presence of albumose, and therefore of trypsin, is assured. A negative result, however, does not mean that the fluid is not from the pancreas, as the trypsin disappears unless the fluid is recent.

(8) *Hydatid Cyst.*—The fluid is watery, colourless, clear, or slightly cloudy, with a specific gravity of 1.006 to 1.010. If this rare condition is suspected, a careful microscopic examination should be made for shreds of the cyst wall, hooklets, and scolices.

(9) *Distended Gall Bladder.*—As previously stated, puncture of the gall bladder is rarely done for diagnostic purposes. If occasion arises for the identification of its contents the fluid will be found to be viscid, colourless, or stained with bile, and of a low specific gravity. Mucin can generally be found by the usual tests.

(10) *Hydronephrosis.*—The fluid is watery, colourless, or slightly yellow, with a specific gravity of 1.008 to 1.020. A chemical examination may reveal urea and uric acid in considerable quantities, which, together with the finding of renal epithelium by the microscope, constitute characteristic features.

(11) *Ovarian Cysts.*—The fluid from an ovarian cyst varies considerably in colour, consistence, and composition. It may be of a

greenish, yellowish, or brownish tint; watery, viscous, or colloid in consistence; and, according to the amount of albumin contained, the specific gravity runs from 1.002 to 1.050.

If it is suspected that the fluid under examination is from an ovarian cyst, one should test for metalbumin (paralbumin). Mix a portion of the fluid with 3 times its volume of alcohol, let it stand for 24 hours, filter, shake the precipitate with water, filter again, and apply the following tests to the watery filtrate: Boil a portion, and if the fluid becomes more or less opaque without the formation of a precipitate, metalbumin is present. Acidify another portion with acetic acid, and add a few drops of a 10 per cent solution of potassium ferrocyanide. In the presence of metalbumin the fluid thickens and becomes of a yellowish colour.

SECTION XLIV

THE USES OF THE RÖNTGEN LIGHT IN MEDICAL DIAGNOSIS

WHILE the mechanical procedures of a Röntgen ray examination are learned with comparative ease, it is distinctly otherwise with the interpretation of the fluoroscopic picture or the skiagraph. Further experience has been convincing that special aptitude, and much study and experience, are requisite correctly to determine the meaning of X-ray observations. A reliable Röntgenologist is, and should be so considered, an expert specialist, to be consulted, as such, by the internist desiring this kind of diagnostic evidence.

This section, therefore, without taking up methods, states the diseases and conditions (EASTMOND) in which more or less diagnostic aid may be expected from a skilled X-ray examination—as follows:

Thorax.—Osteitis, osteo-myelitis, necrosis, or new growth of the bony framework are readily recognised as such, and differentiated. The exact nature of a new growth, however, may not be apparent.

The diaphragm may be studied at rest, taking into account its form and position, and the relative height of the two sides. Changes due to the presence of abnormal growths and increased pressure from below; or disease within the thorax; or variation from the normal excursion during inspiration and expiration, may be seen.

Lungs.—The ready penetration of the lungs by the X-ray makes any deviation from the normal apparent, and even the slightest infiltration or thickening may be distinguished.

Tuberculosis.—Is diagnosticated in all stages (Fig. 264). Slight catarrhal inflammation, however, may escape notice. Cavities are easily identified. This method of examination is most valuable for diagnosis, and for determining the exact extent of the lesions. It is superior to percussion, and, frequently, to auscultation. It affords the earliest definite diagnosis, and supplies a permanent record of the condition. It is of especial value when the physical signs are doubtful, or tubercular lesions exist elsewhere in the body; to watch the progress of the disease; to determine its existence in the presence of other thoracic diseases.

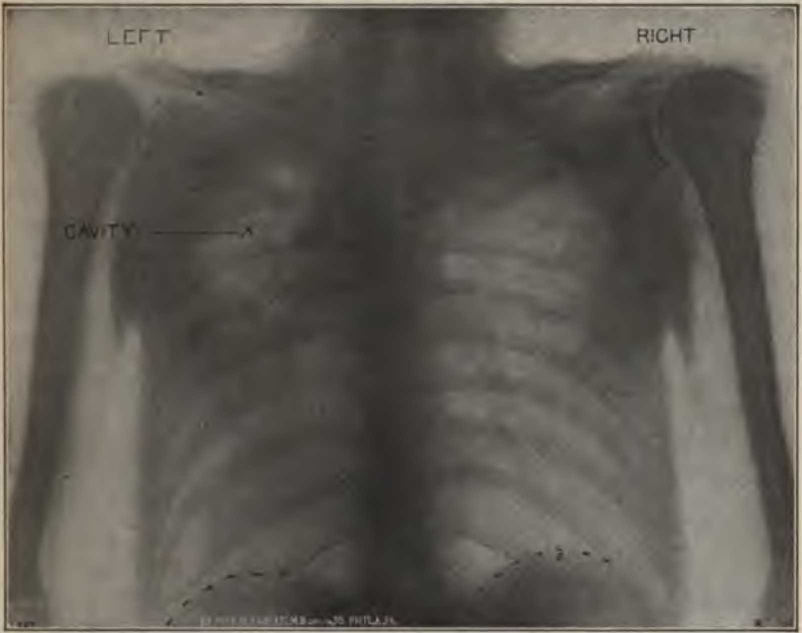


FIG. 264.—Tuberculous consolidation of both apices. Cavity in left apex. Dotted lines show the outlines of the diaphragm. Skiagraph by Kassabian.

Pneumonia.—Of value in confirming the clinical diagnosis; in detecting the presence of unresolved or deep-seated areas; and in the differentiation from tuberculosis or abscess.

Pleurisy.—The extent of fluid may be ascertained; also the presence or thickening or adhesions of the pleura.

Empyema and Abscess.—These conditions are easily recognised, and their exact location determined.

Pneumothorax and Hydropneumothorax.—These are usually apparent.

Œdema and Congestion.—Distinct lesions may be made out, but not always the exact nature of the condition, nor the causative factor.

Emphysema.—Areas of disease easily mapped out.

New Growths.—Appear very distinctly and may be located exactly. Often difficult to differentiate from abscess or an area of local consolidation.

Heart and Pericardium.—Reveals the size, shape, and position, of the heart, and shows abnormalities with greater exactness than percussion. The heart-action and character of the pulsations may be studied. The great vessels can be outlined, and changes in contour observed. Adhesions or new growths of the pericardium, or effusions into it, can be diagnosed.

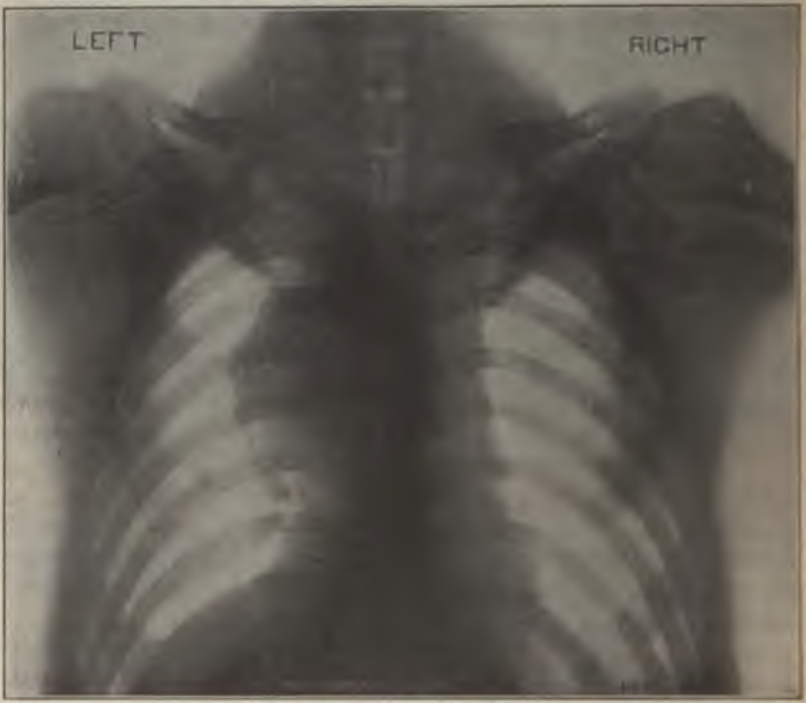


FIG. 265.—Aneurism of the descending arch of the aorta. Posterior view. Skiagraph by Kassabian.

Aorta.—Tortuosities and dilatations can be determined, and the presence of aneurism (Fig. 265) before the existence of physical signs. Aneurisms, when small, cannot always be differentiated from mediastinal tumours.

Mediastinal Tumours.—New growths and abscesses of the anterior or posterior mediastina may be recognised; also disease, calcification, and tumours of the bronchial glands.

Trachea.—Deviation and compression of the trachea from external pressure is easily determined.

Œsophagus.—The position and course may be seen, and dilatations, strictures (Fig. 266) and diverticula noted. Stenosis from spasm, stricture, or pressure from without may be differentiated.

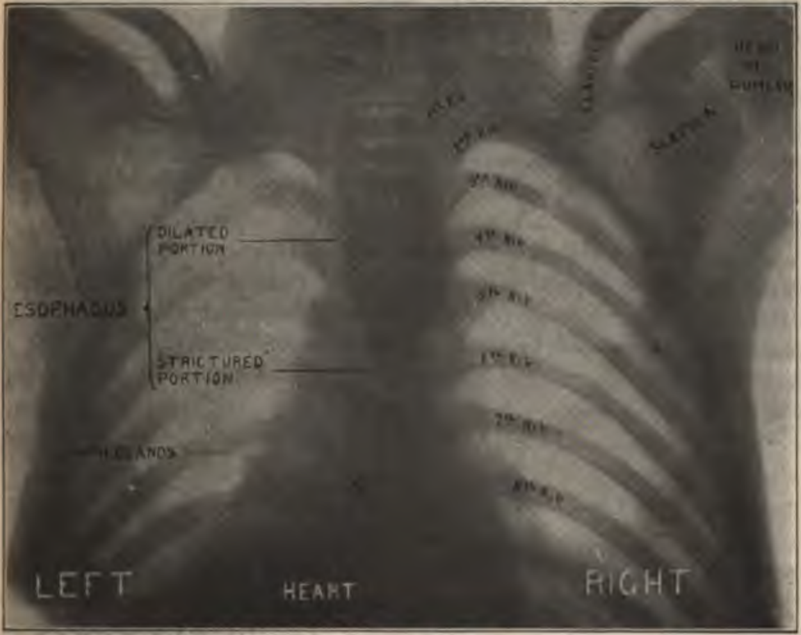


Fig. 266.—Showing stricture of œsophagus. Immediately before exposure, the patient took three drams of bismuth subnitrate, moistened to make a semi-fluid paste. Skiagraph has not been altered or retouched. Lungs show normal translucency. Posterior view. Taken by Dr. C. L. Stone.

Stomach.—The size, shape, and position may be determined, and the degree of motility, together with the character of the peristaltic waves. Displacements are readily seen. The presence of atony, hypermotility, obstruction at the pylorus, infiltration or tumour of the stomach wall, and ulcer may be learned with accuracy.

Intestines.—Prolapse of the colon, strictures, dilatations, and diverticula are readily recognised. Insufficiency of muscular action may be estimated.

Gall Bladder.—Examination of the gall bladder is not very satisfactory. Calculi composed wholly or largely of cholesterin are not visible. When containing a considerable percentage of lime salts, however, they are distinctly seen.

Pancreas.—Pancreatic calculi have been demonstrated.

Liver.—Displacements and enlargements may be discovered. Abscess is rarely apparent.

Urinary Tract.—The size and position of the kidneys can be outlined. Calculi, regardless of their composition, may be found in any part of the tract. The combined error, both positive and negative, is less than two per cent (COLE). The course of the ureters may be learned.

Head.—Disease of the cranial bones may be accurately determined. Brain tumour and hæmorrhage have been demonstrated. This is the most valuable method of diagnosing the existence and extent of disease of the frontal and maxillary sinuses and of the ethmoid cells.

Arteries.—Calcification of the arteries may be shown in the extremities.

Extremities.—The diagnosis of all varieties of disease of the bones and joints is positively ascertained. Simple, tuberculous, syphilitic, or infectious bone involvement may be differentiated, as may also osteomyelitis and tumours. Joint diseases have their characteristic appearances, and the exact nature of the condition presenting can be determined.

SECTION XLV

THE TECHNIC OF LIFE INSURANCE EXAMINATION

By THOMAS C. CRAIG, M.D.

AN insurance company regards a medical examiner as a representative of its Medical Department, and relies on him to safeguard it against all undesirable risks; hence it must repose confidence in his honesty. The medical examiner should regard as confidential any matters relating to the company for which he examines. He should be accommodating to the soliciting agent, and prompt in keeping appointments. He should treat the applicant with the utmost courtesy, but, at the same time, be thorough in discharging his duty to the company. Whenever the examiner discovers anything that he does not think proper to record on the papers, and yet the company should know, he should write a confidential letter, stating the facts, to the "Home Office." The Medical Department appreciates such acts.

Examinations should be made in privacy, and under favourable conditions. No examiner should accept any compensation from an agent or from the applicant, nor should he withhold any information from the company. An examiner should be able to draw some conclusion from the build, general bearing, and behaviour of an applicant; he should also be able to judge whether questions are answered in a frank and honest way. All questions should be plainly asked and, if necessary, explained. All doubtful points and answers should be discussed under the head of "Remarks." The occupation should be correctly given, and all deleterious influences, especially the hazard, of that occupation should be considered and stated.

Does the applicant live in an unhealthy region? Does over-crowding of occupants or contagious disease exist to a degree which is likely to lessen the longevity of the applicant.

Family History.—In the family history of the applicant, the "Home Office" particularly desires to know of the existence of consumption, insanity, apoplexy, cancer, nephritis, or any disease a tendency to which may be transmitted. If possible, avoid such terms as "don't know," "debility," "congestion," "childbirth," "exposure," "complication of diseases," etc., as a cause of death. Inquire as to the symptoms that were present, and if feasible, interview the attending physician. Was there cough with expectoration, emaciation, or night sweats; or, in obscure mental disease, were there symptoms pointing to insanity. The duration of the last illness may throw some light on the cause of death. If the examiner cannot determine the cause of death of the deceased members of the family, an explanation to that effect should be made under the head of "Remarks."

If any relative has died of consumption within three years, and the applicant has resided in the same house in which the death occurred, ascertain what precautions were taken to prevent infection.

Personal History.—The next step is to obtain the personal medical history of the applicant. None of the questions framed to aid the examiner in so doing should be disregarded or lightly considered. Carefully differentiate between trivial ills and those which may in future reduce the expectancy of life. An important point concerning past illness, is to state the number of attacks, their severity, their duration, their complications; has the applicant fully recovered, and, if so, how long since; are there any remaining effects; has the normal weight been regained. An examination of the blood may be necessary for a definite judgment. A sputum examination may determine whether a former expectoration was, or was not, tuberculous. Previous spitting of blood, no matter how trivial, is an important point. Endeavor to ascertain whether it came from the nasal passages, pharynx, alveolar processes, stomach, or respiratory tract. It may be necessary to interview the attending physician. Inquire carefully into a history of abdominal pain, and try to decide whether the trouble was due to hepatic or renal calculus, or appendicitis, or to some less important ailment. A close inquiry as to the symptoms present, the locality and character of the pain, its severity and duration, and whether the attack was accompanied by fever, and nausea and vomiting; whether the stools were sifted for concretions; or whether a full urine analysis was made.

It is very important to ascertain whether the applicant has ever suffered from gout or rheumatism. La grippe, and malarial and other fevers, may lessen longevity, and an examiner should be very certain that the applicant has fully and completely recovered.

A history of renal or liver trouble calls for a searching inquiry. Disease of the nervous system, sun-stroke, or insomnia should be fully gone into. With recurring headaches inquire as to the use of "headache powders." A past or present purulent or bloody discharge from the auditory canal demands a speculum examination of the ear.

Habits.—The habits of an applicant regarding the use of drugs should be a matter of diplomatic and careful inquiry. Has he ever taken a "cure" for this, or for the liquor habit. Accurately ascertain the amount and kind of alcoholic drink used. Is it taken daily or intermittently; to the extent of intoxication or conviviality. Does the applicant indulge in protracted "sprees," and if so, how often and how long. Does he take, habitually, any form of alcoholic drink before breakfast. Be extremely careful to report all such facts.

General Inquiries.—Has the applicant's weight recently increased or diminished, and to what extent. Is his present weight usual. Has the applicant a scar of successful vaccination, or has he ever had small-pox. The venereal diseases should be carefully looked for. In case of gonorrhœa, is there a resulting urethral stricture. Of much more importance is it to ascertain if a venereal ulcer was a local sore, or the initial lesion of syphilis. If the latter, how long specific medication was continued, and whether there remain any glandular enlargements, pigmentary skin discolorations, losses of tissue by ulceration, gummatous enlargements, bone manifestations, or nerve or arterial degenerations. Such matters should be reported confidentially, by letter, to the "Home Office."

Ascertain whether the applicant has ever changed his residence to benefit his health, or has ever been on a special or a restricted diet. Has the applicant suffered any recent or remote injury or accident, and if so, what was its nature, severity, complications, and results. Has recovery been complete. If the applicant has had appendicitis, state the gravity of the attack, the number of attacks, how recent the last one, whether an operation was done, if so, what kind. Did any

TABLE OF STANDARD HEIGHT AND WEIGHT AT VARYING AGES

Based upon an analysis of 74,162 accepted male applicants for life insurance, as reported to the Association of Life Insurance Medical Directors, 1897.

(The figures above and below are 20 per cent. over and under the average.)

Ages.....	15–24	25–29	30–34	35–39	40–44	45–49	50–54	55–59	60–64	65–69
5 feet 0 inches	96	100	102	105	106	107	107	107	105	
	120	125	128	131	133	134	134	134	131	
	144	150	154	157	160	161	161	161	157	
1	98	101	103	105	107	109	109	109	107	
	122	126	129	131	134	136	136	136	134	
	146	151	155	157	161	163	163	163	161	
2	99	102	105	106	109	110	110	110	110	
	124	128	131	133	136	138	138	138	137	
	149	154	157	160	163	166	166	166	164	
3	102	105	107	109	111	113	113	113	112	112
	127	131	134	136	139	141	141	141	140	140
	152	157	161	163	167	169	169	169	168	168
4	105	108	110	112	114	115	116	116	115	114
	131	135	138	140	143	144	145	145	144	143
	157	162	166	168	172	173	174	174	173	172
5	107	110	113	114	117	118	119	119	118	118
	134	138	141	143	146	147	149	149	148	147
	161	166	169	172	175	176	179	179	178	176
6	110	114	116	118	120	121	122	122	122	121
	138	142	145	147	150	151	153	153	153	151
	166	170	174	176	180	181	184	184	184	181
7	114	118	120	122	124	125	126	126	126	125
	142	147	150	152	155	156	158	158	158	156
	170	176	180	182	186	187	190	190	190	187
8	117	121	123	126	128	129	130	130	130	130
	146	151	154	157	160	161	163	163	163	162
	175	181	185	188	192	193	196	196	196	194
9	120	124	127	130	132	133	134	134	134	134
	150	155	159	162	165	166	167	168	168	168
	180	186	191	194	198	199	200	202	202	202
10	123	127	131	134	136	137	138	138	139	139
	154	159	164	167	170	171	172	173	174	174
	185	191	197	200	204	205	206	208	209	209
11	127	131	135	138	140	142	142	142	144	144
	159	164	169	173	175	177	177	178	180	180
	191	197	203	208	210	212	212	214	216	216
0	132	136	140	143	144	146	146	146	148	148
	165	170	175	179	180	183	182	183	185	185
	198	204	210	215	216	220	218	220	222	222
1	136	142	145	148	149	151	150	151	151	151
	170	177	181	185	186	189	188	189	189	189
	204	212	217	222	223	227	226	227	227	227
2	141	147	150	154	155	157	155	155	154	154
	176	184	188	192	194	196	194	194	192	192
	211	221	226	230	233	235	233	233	230	230
3	145	152	156	160	162	163	161	158		
	181	190	195	200	203	204	201	198		
	217	228	234	240	244	245	241	238		

complications exist or follow. Was it a clean or a septic, operation. Is the resulting scar firm, not bulging.

Identity.—The applicant is asked to sign his name to the answers made. If the examiner is not sufficiently acquainted with the applicant to identify him beyond all doubt, he may compare the signature made in his presence with that made by the applicant to the warranty.

Physical Examination.—First weigh the applicant and accurately measure his height. Compare weight and height with the normal standard. (See Table, p. 725.) If *over weight*, has it been taken on recently, or has it been gradual. Feel his muscles, are they hard or soft; feel his bones, are they large or small. Is the excessive weight due to myxœdema, elephantiasis, or a dystrophy. Is the overgrowth symmetrical or localized. What were the height and weight, at the same age, of his parents, and brothers or sisters. State whether the excessive weight is due to fat, bone, or muscle. The following measurements should be recorded:

A. Circumference of the neck.
B. Distance from first cervical vertebra to last lumbar vertebra.
C. Circumference of upper part of trunk; outside of shoulders.
D. Circumference of arm; midway between elbow and shoulder.
E. Circumference of body; around the hips.
F. Circumference of thigh; midway between hip and knee.
G. Circumference of calf; midway between knee and ankle.
H. Length of leg; from hip to ankle.
I. Does he take plenty of outdoor exercise, or is he of sedentary habit.
K. What was his weight at twenty, twenty-five, and thirty years of age.
L. Has his weight materially increased during last ten years.
M. Is this excessive weight a family characteristic.

If the applicant is an *underweight*, the following should be ascertained: Has there been any recent loss of weight? Inquire as to height and weight, at same age, of parents, brothers, or sisters. Is the light weight due to symmetrical smallness. Does he appear to be well nourished. Does he suffer, or has he had any disease affecting his digestion that may account for his light weight. If a recent illness has he regained his normal weight. Has the applicant been on a restricted diet. Has he a good appetite, and does he masticate his food well. How long has he weighed the same as now.

Examine the teeth to see if they are in good condition; and the tongue to see whether it presents a normal appearance, and is free from marks and scars of the teeth. Examine the fauces and note the size and appearance of the tonsils; the colour and condition of the pharynx, and whether it presents any abnormalities. Examine also for nasal disease.

General Ocular Inspection.—Remove all clothing down to the skin, stripping him to the hips. Measure the girth of the abdomen at the umbilicus, and then the circumference of the thorax at full inspiration and at extreme expiration. The identification of a deceased person sometimes becomes very important. The examiner should record sufficient data to enable the "Home Office" accurately to identify the body as that of the person insured. For this purpose note the following points: Colour of the hair and eyes, and whether one eye differ from the other in colour; the site and character of permanent marks and scars; loss of any portion of the limbs, ears, nose, etc.; any deformities; and finally, to state the race and nationality.

Note the colour of his skin whether healthy, muddy, blotched; soft or rough; does the body appear well- or ill-nourished; are there discolorations, signs of hypodermic punctures, seton marks, scars of burns, blisters, old ulcers, or recent eruptions, or surgical cicatrices. Look for spinal curvature. Are the excursions of the chest and abdomen free and full. Tumours or enlargement of parts, if present, should be accurately recorded.

The Pulse and Respiration.—The general ocular inspection concluded, the pulse should be counted in both the standing and sitting posture, placing three finger-tips on the radial. Note the rate, volume, and quality of the pulse. Is there thickening or atheroma of the arteries. Does the pulse intermit or become irregular. Is it full, soft, and compressible. Is the arterial pressure low, medium, or high; if in doubt, use the sphygmomanometer, to determine the systolic and diastolic pressures. Tortuosity of the temporal arteries should be noted.

If any deviation from the normal is observed, inquire as to indigestion, overfatigue, mental worry, undue excitement, the immoderate use of alcohol, tobacco, tea, coffee, or drugs, in order to ascertain a cause for the abnormal rate and character of the pulse. Oftentimes the consciousness of the pulse-counting materially increases its rate. In such cases one should attempt to divert the applicant's attention, in order to overcome this mental acceleration. Next count the respirations. If the rate is above twenty, or less than fourteen, bear this in mind during the physical examination of the thorax, and try to ascertain the cause.

Temperature. — Place a standardized thermometer under the tongue, properly cleansing the instrument before and after using. If the temperature is above or below normal, try to ascertain the cause. If due to some temporary indisposition, postpone the completion of the examination for a few days. If after several attempts there is still a deviation from the normal, finish the examination, report the facts to the "Home Office," and await instructions.

Heart and Lungs.—The next step is to examine the thorax and its contents in a painstaking manner. Is the thorax well formed. Are the respiratory movements full, free, and symmetrical. Examine the different valvular areas with the stethoscope applied to the bare skin. Note the valve-sounds; are they regular, soft, and clear; is a murmur heard; if so, does it occur before, during, or after the valve-sound. Ascertain the seat of its greatest intensity; is it transmitted into the aorta or vessels, or into the axilla and around to the angle of the left scapula. Auscultate the cardiac area and the commencement of the aorta for any roughness or pericardial friction sounds. If a murmur is present, try to decide whether it is organic, or functional and temporary. Differentiate between organic and cardio-respiratory murmurs. Examine the heart with the applicant sitting and lying, and after vigorous exercise. Pay attention to the muscular tone of the first sound. Are all valve-sounds confined to their normal areas or are they diffusely audible. Percuss the cardiac area. Determine the area size of the heart, and locate the apex beat. Finally, draw your conclusion as to whether hypertrophy or dilatation are present.

Next examine the lungs from apex to base in the mammillary, axillary, and scapular lines. Examine with particular care the apices, and along the inner border of each scapula, the latter with the arms folded over the chest. To cough, and then breathe deeply, will sometimes betray a râle that would otherwise be overlooked. Is there evidence of pleural friction, or pleural effusion.

Abdomen.—With the applicant recumbent, examine the abdomen. Endeavour to ascertain the size, and the position of the lower border of the stomach. Palpate the liver and determine whether it is enlarged or diminished in size. Look for the presence of tumours or enlargements of any one or all of the abdominal organs. With a history of abdominal pain, inquire where the pain was most intense. Is the appendix tender. If an operation has been done for appendicitis, or for hernia, test the scar for firmness, or for evidence of hernial protrusion. If a hernia exists, determine its kind and size, and if a truss is worn, whether it supports the hernial protrusion firmly, without friction or discomfort.

Nervous System.—During the preceding examination the examiner can usually judge as to the presence of any disease, either acute or chronic, of the nervous system or the mind.

Urine Examination.—When there is a history of recent trouble in any part of the urinary tract, or when the amount of insurance applied for exceeds a certain amount, a microscopical examination is to

COMBINED EXPERIENCE MORTALITY TABLE

AGE.	Number Living.	Deaths per 10.000.	Expectation. Years.	AGE.	Number Living.	Deaths per 10.000.	Expectation. Years.
10	100,000	68	48.36	**55**	63,469	217	16.86
11	99,324	68	47.68	**56**	62,094	231	16.22
12	98,650	68	47.01	**57**	60,658	247	15.59
13	97,978	68	46.33	**58**	59,161	264	14.97
14	97,307	69	45.64	**59**	57,600	282	14.37
15	96,636	69	44.96	**60**	55,973	303	13.77
16	95,965	70	44.27	**61**	54,275	326	13.18
17	95,293	71	43.58	**62**	52,505	351	12.61
18	94,620	71	42.88	**63**	50,661	378	12.05
19	93,945	72	42.19	**64**	48,744	408	11.51
20	93,268	73	41.49	**65**	46,754	441	10.97
21	92,588	74	40.79	**66**	44,693	476	10.46
22	91,905	75	40.09	**67**	42,565	515	9.96
23	91,219	76	39.39	**68**	40,374	556	9.47
24	90,529	77	38.68	**69**	38,128	601	9.00
25	89,835	78	37.98	**70**	35,837	649	8.54
26	89,137	79	37.27	**71**	33,510	702	8.10
27	88,434	80	36.56	**72**	31,159	758	7.67
28	87,726	81	35.86	**73**	28,797	819	7.26
29	87,012	83	35.15	**74**	26,439	885	6.86
30	86,292	84	34.43	**75**	24,100	956	6.48
31	85,565	86	33.72	**76**	21,797	1,032	6.11
32	84,831	87	33.01	**77**	19,548	1,115	5.76
33	84,089	89	32.30	**78**	17,369	1,204	5.42
34	83,339	91	31.58	**79**	15,277	1,301	5.09
35	82,581	93	30.87	**80**	13,290	1,404	4.78
36	81,814	95	30.15	**81**	11,424	1,514	4.48
37	81,038	97	29.44	**82**	9,694	1,632	4.18
38	80,253	99	28.72	**83**	8,112	1,759	3.90
39	79,458	101	28.00	**84**	6,685	1,897	3.63
40	78,653	104	27.28	**85**	5,417	2,051	3.36
41	77,838	106	26.56	**86**	4,306	2,225	3.10
42	77,012	109	25.84	**87**	3,348	2,422	2.84
43	76,173	113	25.12	**88**	2,537	2,653	2.59
44	75,316	117	24.40	**89**	1,864	2,924	2.35
45	74,435	122	23.69	**90**	1,319	3,237	2.11
46	73,526	128	22.97	**91**	892	3,610	1.89
47	72,582	135	22.27	**92**	570	4,053	1.67
48	71,601	143	21.56	**93**	339	4,572	1.47
49	70,580	151	20.87	**94**	184	5,163	1.28
50	69,517	159	20.18	**95**	89	5,843	1.12
51	68,409	169	19.50	**96**	37	6,486	.99
52	67,253	179	18.82	**97**	13	6,923	.89
53	66,046	191	18.16	**98**	4	7,500	.75
54	64,785	203	17.50	**99**	1	10,000	.50

The above table shows the number that survive at different ages out of 100,000 persons living at 10 years of age; also the expectation of life (or average after life-time) and the yearly death rate at different ages.

From the table we notice that out of 82,581 persons living at age 35, 63,469, or 77 per cent, live to age 55; also of 10,000 persons at age 40, 104 die within one year.

be made. If possible, the centrifuge should be used to obtain the sediment for examination. When available, use a microscope fitted with a mechanical stage. The Welsbach light gives a better illumination than either gas or daylight. Examine with a ½-inch objective for casts, crystals, and epithelium, using the ⅕ or ⅙-inch objective to search for blood, pus, and bacteria.

Female Applicants.—The examination of female applicants is attended with difficulty, and it is sometimes almost impossible to secure a satisfactory representation of their exact condition. It is on account of this fact, among others, that companies hesitate about accepting them. Ascertain the condition, so far as possible, of the reproductive system, in addition to the general facts pertaining to both sexes. Does she menstruate regularly, and in sufficient quantity? If not, is the difficulty due to some local cause, or to the general condition. Is she pregnant? If yes, she should be postponed until a sufficient time has elapsed after labor to establish her usual good health. Have her labors usually been natural, difficult, or instrumental. Has she passed her climacteric? If yes, was that period accompanied by any unusual symptoms. Has her health been re-established? Has she been, or is she, married, or is she a widow?

The urine should be examined in all cases, and the examiner must be satisfied that the urine was passed by the applicant, and no other. Corsets must be removed before examination of the chest.

Final Judgment.—In summing up the information gained from the personal and family history and physical examination, construct a mental picture of the applicant. Weigh all the facts, and from these deduce an opinion as to the outlook, either favorable or unfavorable, for the longevity of the applicant. Does his expectancy (see Table, p. 729) warrant an *average*, or an *extra good*, or a *poor* risk, to recommend for acceptance by the company.

PART II

DIAGNOSIS, DIRECT AND DIFFERENTIAL

HAVING enumerated, in Part I of this volume, the symptoms, signs, and other evidences of disease, mainly with reference to their diagnostic indications—i. e., to what morbid processes each sign or symptom may point—it remains to consider such evidence from an entirely different aspect. Instead of inquiring, "What disease may the symptoms indicate?" one queries, "Do the symptoms found form a combination which is characteristic of a nosological entity—i. e., a recognised and named disease?" Furthermore, another question arises, "Are there any other diseases with which the one in question may be confounded, and in what manner is the distinction to be made?" In other words, the physician having been led by the result of his examination to suspect the existence of a certain disease or diseases, it is evident that he must know what assemblage of symptoms is diagnostic of that particular ailment, as well as the differences between it and the symptom groups of other diseases which may closely resemble it. This knowledge can be obtained only from a study of diseases and their characteristics as distinguished from a study of more or less isolated symptoms and their indications. Consequently, Part II deals in detail with symptomatology and with diagnosis, direct and differential. Pathology, etiology, and prognosis are considered in a cursory manner, not from choice, but from considerations of space and consistency.

SECTION I

INFECTIOUS DISEASES

I. TYPHOID FEVER

AN infection by the *Bacillus typhosus*, most common between 15 and 25 years of age. Prevails especially during the autumn. Period of incubation varies from 8 to 23 days. During this period there may be weakness, lassitude, and general ill-feeling.

Symptoms.—Prodromata are headache, pains in back and legs, chill (rare), chilliness, cough, nosebleed, anorexia, nausea, or slight diarrhœa. The patient finally takes to his bed, from which time the onset of the disease may be definitely dated, although of necessity this point of departure is variable. In the majority of cases the disease lasts 28 days, in the severer cases 42 days, or even longer.

(1) **Onset.**—Usually gradual, may be sudden, especially in children. May begin exceptionally as

Nephro-typhoid, with all the symptoms of an acute nephritis.

Pneumo-typhoid, with a severe bronchitis, a lobar pneumonia, or an acute pleurisy.

Gastro-enteric typhoid, with vomiting and diarrhœa.

Cerebro-spinal typhoid, with nausea, delirium, severe headache, or facial neuralgia, photophobia, cervical retraction, twitching of muscles, convulsions, drowsiness or stupor.

Typhoid septicæmia, with delirium, high fever, and symptoms indicating a severe infection, without localizing lesions.

(2) **Fever.**—*Typical.*—In the typical case the temperature rises gradually during the 1st week, reaching 104° to 105°, with morning remissions of 1 to 1½ degrees. During the 2d week it remains high, with slight remissions; so also during the 3d week, except that the remittent character is more marked. During the 4th week, or at the end of the 3d week, the morning record approaches or becomes normal, while the evening temperatures, which are from 1 to 4 degrees higher than those of the morning, persist, but with gradually decreasing height, until by the end of the 4th week the record is 98.6° (Chart X).

Irregularities in the Temperature.—Very commonly variations from the type just described are found. A low evening and high morning temperature (inverse type) is of no importance. The fever may terminate at the end of the second week, the temperature reaching normal in from 12 to 48 hours. In children, or in cases beginning with a chill, the fever may rise abruptly. A sudden fall of 3 to 10 degrees suggests intestinal hemorrhage or perforation. During convalescence, usually as a result of emotional excitement, dietetic imprudences, or constipation, the temperature may rise quickly to 102° or 103° (recrudescence), returning to normal in 2 or 3 days. Aside from the fever, the condition of the patient is satisfactory. A persistent slight fever (99.5° to 100.5°) may be due to anæmia, to insufficient food (DA COSTA), to latent pleurisy, to beginning disease of the bones, or, in the absence of other symptoms, have no assignable cause. A *relapse* is a replica of the original attack, with characteristic symp-

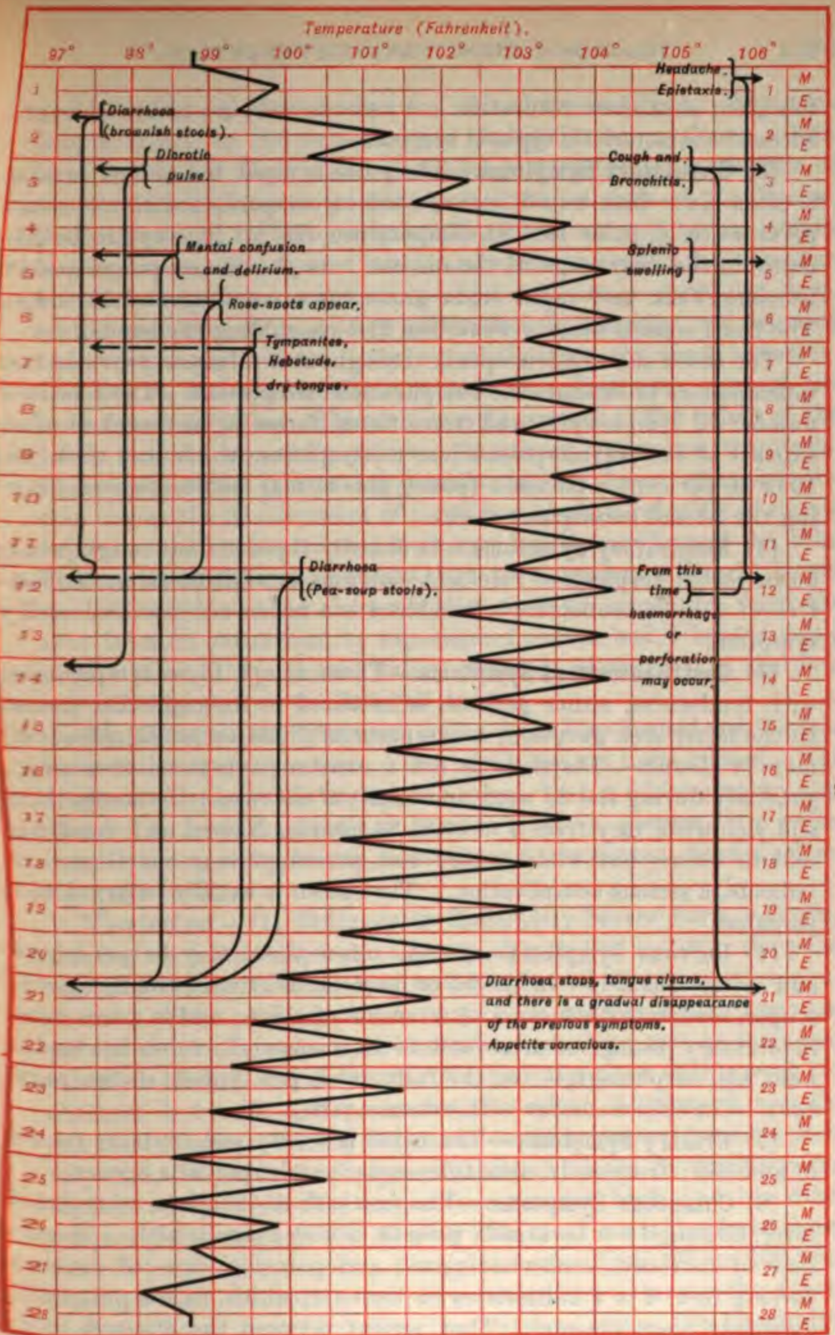


CHART X.—Showing the typical temperature curve of typhoid fever. Also the average times of appearance and the duration of prominent signs or symptoms (shown by black pointing lines), e. g., the dicrotic pulse is manifest on the 3d day, and continues to the 14th day; rose spots appear on the 6th day, and continue to appear until the 12th day.

toms, but is of shorter duration. A *recrudescence* is a return of fever, without characteristic typhoid symptoms.

(3) **Circulatory Symptoms.**—The pulse is rapid, but the pulse rate is often lower than would be expected in comparison with the temperature (e. g., pulse rate 90, temperature 104°). It is apt to be dicrotic in the early stage of the disease. As the disease progresses it becomes weak and rapid or, in grave cases, running and thready. The heart sounds become weak, the first resembling the second.

The blood shows a leucopenia throughout the disease, provided no inflammatory or suppurative complications are present. The relative number of lymphocytes and transitional forms is increased at the expense of the polymorphonuclear neutrophiles, which may sink to 50 or 60 per cent. Anæmia (post-typhoid) may be pronounced during the 3d and subsequent weeks.

(4) **Respiratory Symptoms.**—Ordinarily there is some cough, with an increased respiration rate and sonorous or sibilant râles, indicative of a mild bronchitis. Nosebleed is a rather common and early symptom.

(5) **Gastro-intestinal Symptoms.**—There is apt to be tympanites, with tenderness, either general, or localized in the right iliac fossa. In the latter area gurgling, a symptom of little or no significance, may be found. Diarrhœa is not a constant symptom, occurring, generally during the 2d week, in about ¼ of the cases; the stools, thin and yellowish, vary from 3 to 10 in 24 hours. Nausea and vomiting seldom occur, but when severe and persistent may constitute, or indicate, a serious complication. The spleen is usually enlarged and palpable.

(6) **Nervous Symptoms.**—At the onset there may be persistent and severe headache; in the 2d or 3d week delirium (usually quiet), stupor, moderate deafness, and retention of urine; and in grave cases involuntary passage of urine and fæces, coma-vigil, subsultus tendinum, and carphologia—i. e., the fully developed typhoid status (page 169). Convulsions occur with extreme rarity.

(7) **Urinary Symptoms.**—The urine is scanty and contains traces of albumin; frequently, also, tube casts; rarely, polyuria occurs.

(8) **Cutaneous Symptoms.**—The rose rash or exanthem of typhoid fever, although not invariably present, is very characteristic. It consists of flattened, somewhat raised and palpable, rose-red papules varying from 2 to 4 millimetres in diameter, which, unless petechial, disappear upon pressure. They appear between the 6th and 12th day of the disease, each spot lasting 2 or 3 days, and leaving a slight brownish stain. Coming in successive groups, perhaps up to the middle of the 3d week, they are found most commonly upon the

abdomen, but very frequently upon the chest and back. This rash may be absent in the very young or the aged. An erythema, a brilliant redness of the skin of the chest and abdomen, sometimes of the extremities, may be present during the 1st week of the disease. Sudamina (page 93) are common. The infrequency of herpes labialis in this disease, as compared with pneumonia or malaria, has considerable diagnostic value. Peliomata—*taches bleuâtres*—irregular, deep-seated, pale-blue spots, 4 to 10 millimetres in diameter, and situated upon various parts of the body, especially upon the chest, abdomen, and thighs, are occasionally met with as a diagnostic sign of the presence of body lice. Urticaria may be present. The palms of the hands may be unusually yellow. Cutaneous vasomotor manifestations are common in, but not peculiar to, this fever. Stroking the skin causes the development of a red line (*tache cerebrale*); or simple exposure to the air produces a pink-and-white mottling of the skin. Profuse sweating rarely occurs, but to a moderate degree is not uncommon. A rather characteristic odour or exhalation from the skin is often mentioned by experienced nurses, and, I believe, has a certain limited diagnostic value. During convalescence the skin may desquamate and the hair fall out.

(9) **Miscellaneous Symptoms.**—The cheeks are flushed and the face has a dull, heavy expression. The pupils are apt to be dilated. The tongue at first is red at its tip and edges, with a white coating elsewhere. A deep longitudinal furrow is often present. In severe cases it becomes dry, brown, fissured, and tremulous. Sordes collect upon the teeth.

Varieties.—(1) **Ordinary or Moderately Severe Form.**—During the *first week* the temperature slowly rises to 103° to 104°; the pulse runs to 100 or 110, often less, and is soft and dicrotic; the tongue shows a white coating; there may be a moderate diarrhœa; the patient coughs, complains of headache, and there may be some nocturnal confusion and delirium. Toward the close of this week the abdomen is tumid, perhaps tender, the spleen is enlarged, and the rose rash appears. During the *second week* the patient becomes mentally dull, the headache disappears, the pulse loses its dicrotism and becomes more rapid, the temperature remains high, and the other symptoms are aggravated. During the *third week* the fever shows marked morning remissions, the pulse varies from 110 to 130, and weakness and emaciation are very noticeable. During the *fourth week* the fever slowly subsides, the diarrhœa ceases, the tympanites disappears, the tongue clears, the mental condition improves, and a fierce hunger begins to possess the patient. Chart X represents the course of the average case, with the times of appearance and duration of the principal symptoms, and some of the possible complications.

(2) **Grave or Severe Forms.**—Bad cases present high fever (105° or over), a dry, brown tongue, twitching of the tendons, picking at the bedclothes, excessive meteorism, constant and marked delirium, rapid and feeble pulse, weak heart, pulmonary, renal, gastro-intestinal, or nervous complications, involuntary voiding of urine and fæces; and, if death does not occur, the fever persists into the 5th, 6th, or 7th week, or even longer.

(3) **Mild Form.**—The disease does not last over two weeks, the temperature does not exceed 103°, the rose spots are present in scanty numbers, the spleen is enlarged—in short, while the symptoms of the ordinarily severe form are present, they are of much less intensity.

(4) **Abortive Form.**—The onset is sudden and definite, with shivering and a temperature of 103°. Splenic swelling, meteorism, and rose spots appear promptly (2d to 5th day). The fever lasts from 7 to 12 days, and terminates rather abruptly, often with free sweating. Relapses may occur, and are sometimes more severe than the original attack.

(5) **Latent or Walking Typhoid.**—The symptoms are so slight (languor, diarrhœa, anorexia) that they are disregarded until the disease is well advanced. On examination, characteristic symptoms, especially rose spots, are found; or delirium, intestinal hemorrhage, or perforation leads to an imperative demand for medical aid.

(6) **Afebrile Typhoid.**—While the existence of these cases is affirmed by good authority, they must be of excessively rare occurrence or often pass unrecognised. Headache, weakness, coated tongue, anorexia, slow pulse, splenic swelling, the rose rash, and perhaps an occasional evening rise of temperature to 100.5°, are said to characterize such cases.

(7) **Typhoid Fever in Children.**—The onset is less gradual, nosebleed is not frequent, delirium and insomnia, as well as bronchitis, are more common, diarrhœa is not infrequently absent, the rose rash may be absent, and the duration is usually shorter (2 to 3 weeks).

In infants, inflammation of the bladder, occurring without cystitic symptoms, and due to infection by the coli communis, may be mistaken for typhoid fever. There is continuous high fever and rapid pulse, perhaps with symptoms of bronchitis or broncho-pneumonia. Aside from the rarity of typhoid fever in infants, deep pressure over the pubis is painful, bacteriological examination of the urine (if it can be obtained) will show the presence of the *Bacillus coli communis*, and the Widal reaction is negative.

(8) **Typhoid Fever in the Aged.**—In patients over 40 years of age the temperature curve is lower and less characteristic; cardiac weakness, pneumonia, and renal inflammations are more common, and splenic swelling and rose rash more frequently absent.

Complications and Sequelæ.—There is in typhoid fever a large variety of complicating conditions or lesions, as follows:

(1) *Exaggeration of an Ordinary Symptom as a Complication.*—Hyperpyrexia is more an indication of the severity of the infection than a danger in itself. Excessive tympanites embarrasses the action of the heart and lungs, and favours intestinal perforation by overstretching. Severe diarrhœa and excessive vomiting seriously decrease the strength of the patient and may cause fatal exhaustion. Epistaxis may be profuse and serious. Severe recurring sweats, with a rapid fall of temperature, characterize some rare but striking cases.

(2) *Respiratory Complications.*—Lobar pneumonia rarely initiates the disease, but occurs more commonly during the 2d or 3d week. Hypostatic congestion, announced by a somewhat increased respiration rate, dulness, weakened voice sounds, and moderately fine, moist râles during inspiration at both bases, is found not infrequently in the later stages. Bronchitis (usually present) may extend to the air cells (broncho-pneumonia) with associated atelectasis. Rarely infarcts, abscess, or gangrene of the lung or hæmoptysis may occur. Pleuritis (during convalescence apt to be an empyema) is not very common; still less so is pneumothorax, due to the rupture of a small abscess or to overstraining of the lung. Laryngitis and ulceration of the larynx with sequent perichondritis may occur; and laryngeal paralysis (neuritis) may show itself during convalescence.

(3) *Circulatory Complications.*—Endocarditis or pericarditis may occur. Thrombosis of veins is not uncommon, particularly of the left femoral vein, sometimes of both veins, announced by increased fever, pain, swelling, and a hard œdema of the leg, generally beginning in the foot. The affected vein may be felt as a hard cord, and the superficial veins are plainly visible as bluish lines. One or both arms may also be involved. Less frequently there may be embolism or thrombosis of the femoral artery. The limb becomes cold, numb, anæsthetic, and partly paralyzed. Gangrene of the foot and leg may follow.

(4) *Gastro-intestinal and Abdominal Complications.*—Typhoid ulcers may form in the pharynx and esophagus. Membranous pharyngitis, developing in the 3d week, with dysphagia, is a rare and usually fatal complication. Suppurative parotitis, generally of one side, is not common, but when present is a grave and usually fatal sequel.

Intestinal hemorrhage occurs in from 3 to 5 per cent of all cases, mainly during the 3d or 4th week, and terminates fatally in from 30 to 40 per cent. The symptoms are a sudden fall of temperature (Chart XI), with signs of collapse, and in a few hours the voiding of bloody or tarry stools. If the hemorrhage is large and rapid, death may occur before the blood issues from the rectum.

Intestinal perforation, most common at the end of the 2d or during the 3d week, occurs in practically 3 per cent of all cases, more frequently in men than in women. In the majority of instances (75 per cent) it is announced by sudden acute abdominal pain, with a rapid development of rigidity and tenderness. Vomiting, a weak and rapid pulse, and a marked condition of collapse accompany the abdominal symptoms. If the pre-existing tympanites is great and the general symptoms already severe, the occurrence of perforation may be difficult to determine. The distention increases, and tenderness may be detected by deep and firm pressure; or, in the absence of any local inflammatory (especially suppurative) process, the question may be decided in the affirmative by finding a leucocytosis. A rapid increase in blood pressure is an early symptom.

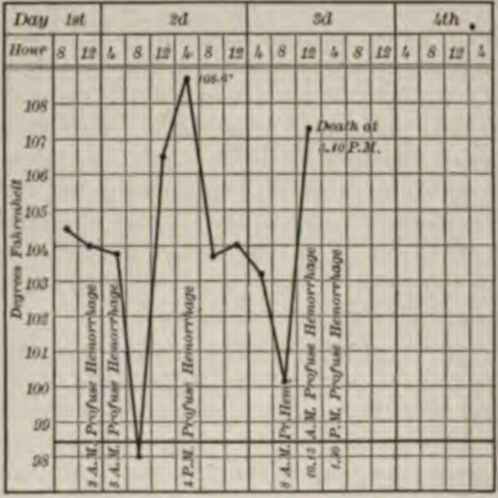
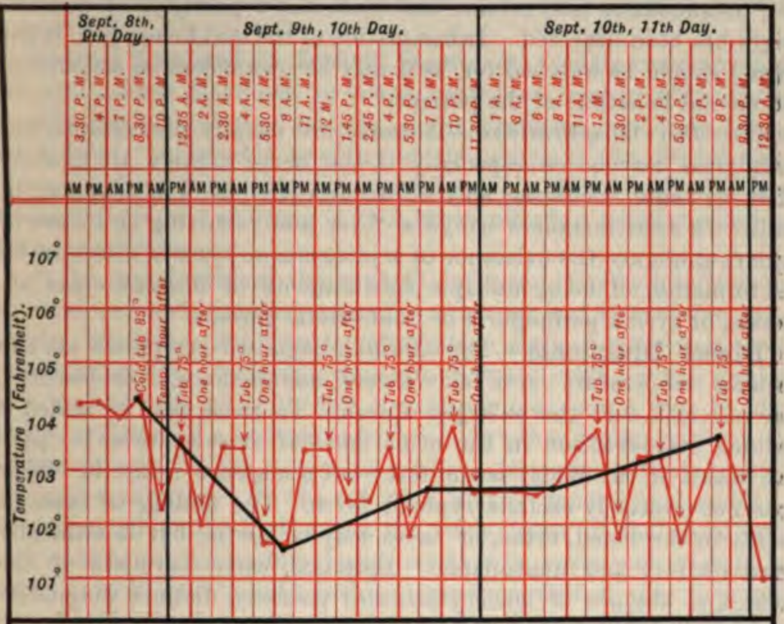


CHART XI.—Fatal hemorrhage in typhoid fever.

Cholecystitis, cholangitis, and jaundice (obstructive or toxæmic) are not uncommon; hepatic abscess is very rare. Gallstones form after typhoid fever with notable frequency. The softened, pulpy spleen may in rare instances rupture. Peritonitis may be a consequence and symptom of perforation or, less commonly, arise, by extension through the intestinal walls, from an ulcer, or a ruptured suppurating mesenteric gland.

(5) *Nervous Complications.*—Neuritis, which may be multiple, is not infrequent. It may be localized, involving one arm or leg. The affected nerves are excessively painful and hyperæsthetic, and the muscles supplied by them lose power. The extensors of the arm or leg may be involved, causing wrist-drop or foot-drop. Especially

after cold bathing "tender toes" (probably a neuritis) may develop, usually disappearing within 10 days. The "typhoid spine" occurs during convalescence, with severe pain in the back and legs upon movement. It is non-febrile, and probably a neurosis.



BATHING CHART OF TYPHOID FEVER RECOVERY.

Black line—Temperatures at 8 A. M. and 8 P. M.

Red line—Intermediate temperatures.

CHART XII.

Among the rare nervous complications are meningitis; hemiplegia, with or without aphasia, generally due to arterial thrombosis, less often to a meningo-encephalitis; poliomyelitis, which may be confused with multiple neuritis; and tetany. Convulsions seldom occur, except perhaps at the onset of the disease in children; in adults, if present, they are due to arterial or venous thrombosis, or encephalitis. Post-typhoid insanity is a possible event during convalescence.

(6) *Complicating Diseases of Bone.*—Inflammation of the joints (arthritis, single or multiple) is rare. Periostitis, caries, or necrosis of bone, usually of the tibia, less often of the ribs, is not uncommon. These processes are essentially chronic and recurring, and may constitute a possible cause of protracted convalescence.

(7) *Genito-urinary Complications.*—An acute nephritis may usher in the disease, more commonly it occurs in a mild form during convalescence. Hæmoglobinuria is a rare, pyelitis an occasional, event. Ovaritis and orchitis have been noted.

(8) *Eye and Ear Complications.*—Conjunctivitis, iritis, and corneal inflammation may occur; and oculo-motor paralysis of neuritic origin has been observed. Inflammation of the middle ear (not infrequent) should be watched for, as it may be overlooked on account of the mental hebetude of the patient.

(9) *Other Complications.*—Bedsores are usually preventable, but with great emaciation, especially if the nerve centers are gravely affected, acute decubitus may occur over the sacrum, scapulæ, or heels. In some instances irregular fever and recurring chilliness or rigours announce the existence of septicæmia or pyæmia, attended by the formation of boils, multiple subcutaneous or intramuscular abscesses, or even a perinephric or ischio-rectal abscess.

Direct Diagnosis.—The cardinal diagnostic symptoms are the peculiar temperature curve (slow ascent, maintenance, slow descent), the rose rash, and the enlarged spleen. To these may be added a decided diazo-reaction in the urine and—of extreme value—a positive result of the Widal serum test; but a negative result in either does not positively exclude typhoid fever. The finding of typhoid bacilli in the blood, urine, or fæces may be useful, but is clinically unsatisfactory and unavailable. Epistaxis, early dicrotism of the pulse, and absence of leucocytosis also possess a distinct diagnostic value.

It should be clearly understood that as different organs may bear the brunt of the infection, so may the manifestations of the disease be extremely variable. Thus, while in many cases the usual abdominal symptoms (meteorism, diarrhœa) are present, in others they may be entirely lacking during the whole course of the disease; or certain symptoms may be replaced, or masked, by symptoms referable to the kidneys (nephritis), lungs (pneumonia), or other organs. In view of the inconstancy of any one or two symptoms, the diagnosis of typhoid fever should not be made unless based upon a study of the entire clinical picture. It is best not to make a positive affirmation of its existence until the disease has progressed far enough to present a sufficient assemblage of symptoms to warrant such a diagnosis. On the other hand, any continued fever should be considered as a possible typhoid until the assumption can be disproved by its course and the evolution of its symptoms. As a matter of fact, the great majority of cases of typhoid fever are correctly diagnosed as such, by competent observers, within the first week or ten days; in a certain small proportion the diagnosis remains uncertain for a longer period; in a still smaller number no positive diagnosis is made, even after recovery; and in a few instances the autopsy reveals the presence of the disease, when it had not been suspected during life.

Differential Diagnosis.—As Herrick cleverly puts it, typhoid fever is not only an imitator of diseases, but many diseases imitate typhoid fever.

Diseases Simulated by Typhoid Fever.—(1) Certain cases are met with and usually diagnosed as *simple continued fever* in which there are no symptoms except malaise, slight headache, moderate fever, and a barely perceptible swelling of the spleen. The illness continues for from 5 to 8 days only, and yet the Widal test (the sole available diagnostic evidence in such cases) affords a positive reaction during convalescence.

(2) In rare instances the typhoid infection may be localized in the meninges and simulate sporadic *cerebro-spinal meningitis*, presenting headache, delirium, photophobia, cervical retraction, and muscular rigidity. Similarly, an *acute nephritis*, with scanty, smoky urine, loaded with albumin, and containing many casts, may be the earliest evidence of a typhoid infection. In other instances the poison first attacks the lungs, causing a *lobar pneumonia*, with its typical signs and symptoms. Or the infection may be general and sudden, with repeated chills, high fever, the early onset of delirium, and other symptoms of a profound *septicæmia*. In every such case it is well to have in one's mind a lurking suspicion that it may turn out to be of typhoid origin, and to watch for diarrhœa, meteorism, splenic swelling, and especially the rose spots; not omitting the Widal test in suspicious cases.

(3) *Malaria.*—Intermittent fever (tertian organism) can not be confounded with typhoid fever. Aside from the finding of the plasmodium, the paroxysms are promptly stopped by quinine.

Infection by the æstivo-autumnal variety, however, may simulate typhoid fever very closely for a time. But, if the work of Osler is to be trusted, it may be confidently stated that in the Northern and Middle States this form of infection is very rare, and a continued fever due to malaria very exceptional. Furthermore, the studies of Dock and Vaughn prove conclusively that many cases diagnosed as malaria are in reality typhoid fever. In view of the vast importance of prophylactic disinfection, even omitting the imperative need for rest and careful diet in the individual case, it is best to accept the dictum that typhoid fever, and not malaria, is to be suspected "in every case of fever of 6 or 7 days' duration, particularly if it resists the action of quinine."

The distinction between a malarial remittent and typhoid fever depends upon the following points:

In malarial fever the remittent character of the fever is marked from the outset: anæmia with a subicteric complexion occurs early in the disease, herpes labialis is more frequent, the pulse is rarely dicrotic, abdominal symptoms are not marked, and the rose spots do not appear. On the other hand, there may be splenic enlargement, and the general condition may so closely resemble typhoid fever that it is impossible to make a differential diagnosis until the end of the first week. This limit is set, because the crucial test—the finding of the plasmodium in the blood—can not in many cases be successfully made during this period for the reason that the hyaline bodies of the æstivo-autumnal parasite are seldom to be found in the blood, and one must wait until the pigmented crescents and ovoids appear, at least 7 days as a rule, after the onset of the disease.

Two other distinctive points are a failure to obtain the Widal reaction during the course of the disease, and the therapeutic test—the subsidence of the fever in 4 or 5 days as a result of the administration of quinine. Recurring chills have little diagnostic value, as they may occur in both diseases: in malaria as a frequent but not necessary symptom, in typhoid fever at its beginning, or at the onset of a relapse or a complication, or when the temperature rises after having been thrust down by antipyretics.

In rare instances there may be a double infection with the *Bacillus typhosus* and the *Plasmodium malariæ*, but the weight of evidence is decidedly against the existence of the so-called typho-malarial fever as a separate disease.

Diseases Simulating Typhoid Fever.—(1) *Acute Miliary Tuberculosis.*—In this the fever is much more irregular, the pulse and respiration are more rapid, there is usually noticeable cyanosis, constipation is the rule, and the rose spots do not appear. On the other hand, the simulation of typhoid fever may be so close that the acutest diagnostician is at fault. A positive Widal test, the finding of tubercles in the choroid, or more rarely of tubercle bacilli in the blood (or in the sputum if signs of apparent pulmonary tuberculosis become manifest), constitute the only reliable differential evidences. Time alone may settle the diagnosis. The diazo-reaction may possibly be of service (page 700).

(2) *Pyæmia.*—The marked prostration, irregular fever, delirium, diarrhœa, and splenic enlargement attending some pyæmias may simulate typhoid fever, but a negative Widal test, or a marked leucocytosis, with perhaps the finding of foci of suppuration, will eliminate typhoid fever.

(3) *Tuberculous Peritonitis.*—This, when coming on slowly, with a continuous low fever, tympanites, and abdominal tenderness, may imitate typhoid fever. The Widal and the tuberculin tests may aid in separating the two, but continued observation may be required.

(4) *Appendicitis.*—This disease may simulate, or be simulated by, typhoid fever. On account of right iliac tenderness and tumefaction due to the swollen ileum and mesenteric glands of typhoid fever, a surgeon has more than once been called to operate for a supposed appendical inflammation. But in the majority of cases of appendicitis the onset is so abrupt and the symptoms and physical signs so distinctive, as compared with typhoid fever, that this mistake is seldom made. Conversely, appendicitis may be taken for typhoid fever, but the history, together with a careful physical examination, and, in addition, the blood examination and serum test, will usually settle the differential diagnosis in such rare cases.

(5) *Ulcerative Endocarditis.*—The more chronic forms of this disease are commonly diagnosed as typhoid fever. The presence or absence of a leucocytosis, and the result of the Widal test may decide the question. Recurring chills, irregular fever, substernal pain, the development of endocardial murmurs, and the absence of abdominal symptoms and the rose spots, point toward ulcerative endocarditis rather than typhoid fever.

(6) *Salpingitis* (*right*).—I have known one case in which the occurrence of continued fever and the typhoid status with right iliac tenderness in this disease led to a diagnosis of typhoid fever, but a vaginal examination, not previously made, settled the question at once by disclosing a fixed uterus and a tender mass in the right pelvis.

(7) *Catarrhal Enteritis.*—This, especially in children, may be taken for typhoid fever, but enteritis lacks the epistaxis, bronchitis, high fever, splenic enlargement, rose spots, and positive Widal reaction.

(8) *Pneumonia.*—When lobar pneumonia is the initial symptom of typhoid fever, or when pneumonia presents the typhoid status (especially if the patient is seen for the first time after this condition has developed), a differential diagnosis may be quite impracticable until the rose spots appear, or a positive Widal reaction is obtained. Ordinarily no confusion need arise.

(9) *Epidemic Influenza.*—Rarely this disease may cause an elevation of temperature, lasting for 3 or 4 weeks, and closely resembling typhoid fever. The non-appearance of the rose rash, and the absence of a positive Widal reaction, with perhaps the discovery of the Pfeiffer bacillus in the nasal or bronchial secretions, will serve for

differentiation. Furthermore, the prevalence of an epidemic of influenza is suggestive.

(10) *Uræmia.*—The more chronic forms of uræmia may closely simulate typhoid fever because of the presence of stupor, rapid and feeble pulse, dry, brown tongue, subsultus tendinum, and continuous slight fever, lasting for weeks. The absence of the rose rash, the negative Widal test, the presence of the urinary characters of uræmia (page 711), and the condition of the heart and arteries, must be relied upon, perhaps with an ascertained previous history of chronic renal disease.

(11) *Trichiniasis.*—The severe cases of this disease have been diagnosed as typhoid, because of the prolonged fever, delirium, dry, brown tongue, abdominal pain, and diarrhœa which may be present. Œdema of the eyelids, swelling and tension of the muscles, and dyspnœa should suggest trichiniasis. In suspected cases the blood should be examined for a marked eosinophilia, and the stools and an excised bit of muscle for the trichina.

(12) *Typhus Fever.*—In modern times and civilized countries an occasion for differentiating between typhus and typhoid fever rarely arises. The onset of the former is sudden, delirium is early and often active, the stupor becomes profound, the pupils are contracted, the conjunctivæ are brilliantly injected, and about the 4th day of the disease the macular and petechial rash appears. The fever lasts 12 to 14 days and terminates by crisis. When an epidemic of typhus is present the diagnosis is usually easy, but it may be almost or quite impossible to distinguish a sporadic case of this disease from typhoid fever.

(13) *Relapsing Fever.*—The early cases of an epidemic of this disease may be diagnosed as irregular typhoid, but in relapsing fever the onset is sudden, with chill and intense general aching, and in 6 or 7 days the temperature falls suddenly to or below the normal. In a week the attack is repeated, and a 3d or 4th similar relapse may occur. If this disease is suspected its nature can be positively determined by a blood examination, which reveals the presence of the spirilla.

Paratyphoid Fever.

A disease in most cases clinically inseparable from typhoid fever, except by bacteriological methods. The etiological factor is a group of bacilli (divided into 2 classes, alpha and beta), closely resembling the typhoid bacillus, but culturally placed between this bacillus and the bacillus coli communis.

Paratyphoid fever, like typhoid fever, has its chief incidence in the autumn months, and, also like typhoid, occurs mainly in adolescents and young adults. As to the manner in which the bacilli enter the body little can be learned from sporadic cases; but in certain small epidemics it was proved by the manner of distribution of the cases, and by the finding of the bacilli in the drinking water, that the infection took place by means of the latter.

The cases have occurred in the United States, Philippines, and Europe.

Symptoms.—While the general appearance of this disease is that of typhoid fever of varying degrees of severity, there are certain features which in many cases may point to paratyphoid infection. The prodromata are generally shorter, epistaxis is less common, the onset is more abrupt, with repeated vomiting and diarrhœa (generally continued throughout the course of the disease), and often intense early prostration. Angina is frequently present. The course of the disease is commonly shorter, with milder general symptoms and less derangement of the sensorium. Spots are present in about one-half the cases; the spleen is usually not palpable but is enlarged to percussion; there is no iliac tenderness (probably due to absence of intestinal ulceration, though hemorrhage may take place); and the pulse is rarely dicrotic. The temperature often falls by crisis. Complications are more frequent than in typhoid fever.

Diagnosis.—It may be safely said that, up to the present time, there is no symptom, or group of symptoms, whereby it is possible to differentiate between typhoid fever and paratyphoid fever. This discrimination can be made positively only by finding the specific bacteria by bacteriological methods, or by the agglutination tests. If the customary Widal is negative, the tests should be made with both groups of paratyphoids; a positive reaction making the diagnosis.

The prognosis, in the majority of cases, is distinctly favourable, the mortality being about 3.5 per cent.

II. TYPHUS FEVER

Symptoms.—Begins suddenly with chills (perhaps recurrent), fever, pain in head, back, and extremities, soon followed by extreme prostration. Cough and bronchitis are common. The pulse is rapid, full, and often dicrotic. The tongue is white, dry, and tremulous, in bad cases becoming brown or black. The expression is dull and heavy, the face duskily flushed, the eyes congested, and the pupils contracted. In the severer cases, delirium, which may become active or maniacal, together with subsultus tendinum, coma-vigil, and carphologia, constitute prominent symptoms. Vomiting may be troublesome, and constipation is the rule.

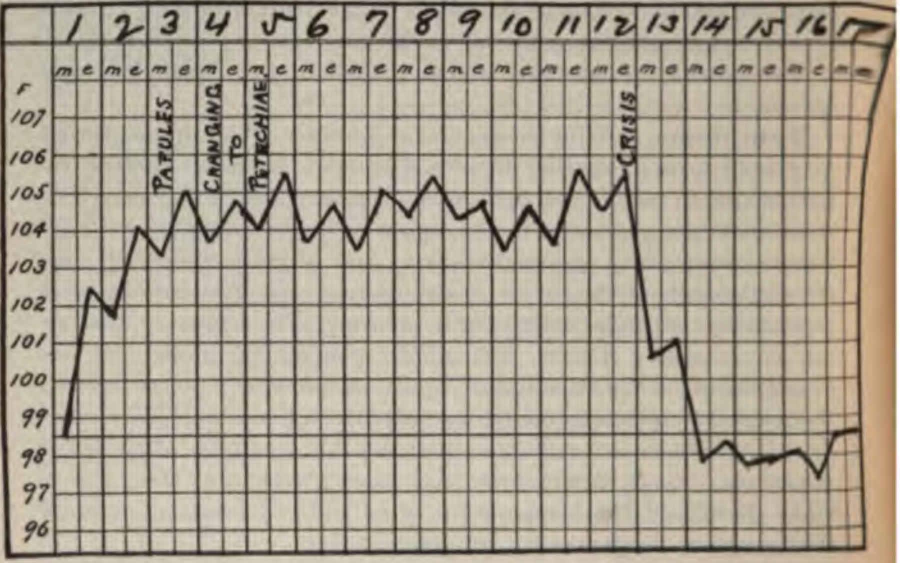


CHART XIII.—Typhus fever.

The fever rises, with moderate morning remissions, during the first 3 or 4 days, reaching a maximum of from 103° to 107°. This maximum is maintained for about 10 days, and then falls by crisis to or below the normal within 12 to 24 hours (Chart XIII). Between the 3d and the 5th day the eruption appears, partly as a dusky subcuticular mottling, partly as papular rose spots, which rapidly become petechial. The rash appears first upon the abdomen, whence it spreads over the body. The urine is diminished in amount, high-coloured, and usually albuminous. Retention of urine is common. Like other infectious diseases, exceptional cases occur which may be either mild, or malignant and fatal within 2 or 3 days.

Complications.—The most common of these is broncho-pneumonia, which may eventuate in gangrene of the lung. Suppurative arthritis, parotid abscess, subcutaneous abscesses, gangrene of the smaller extremities, neuritic paralysis, hypostatic congestion of the lungs, and hyperpyrexia are occasional events. Nephritis, meningitis, and hæmatemesis are rare complicating conditions.

Differential Diagnosis.—(1) *Typhoid fever* (page 748).

(2) *Cerebro-spinal Meningitis.*—In this, one finds a greater irregularity of the fever, greater pain in the back, a more frequent occurrence of convulsions, facial or ocular paralyses, photophobia, intolerance of sounds, and cervical retraction. The eruption is less constant, and does not appear at any definite period in the disease. Finally, lumbar puncture and the finding of the meningococcus may settle the question.

(3) *Smallpox.*—Malignant smallpox may resemble severe typhus, but the hemorrhages (subcutaneous, hæmaturia, hæmatemesis, intestinal, conjunctival) will speak for the former disease.

Prognosis.—The mortality varies from 12 to 20 per cent; in children slight, and after middle age high (perhaps 50 per cent). Death results from toxæmia or a complicating pneumonia.

III. RELAPSING FEVER

Symptoms.—The period of incubation of this disease, caused by the *spirochæte* or *spirillum* of Obermeier, varies from 5 to 7 days. The invasion is sudden, with chill, rapid onset of fever (104° to 106°), intense headache, backache, and aching in the extremities. The pulse runs from 110 to 130. Sweats, jaundice, and delirium (if fever

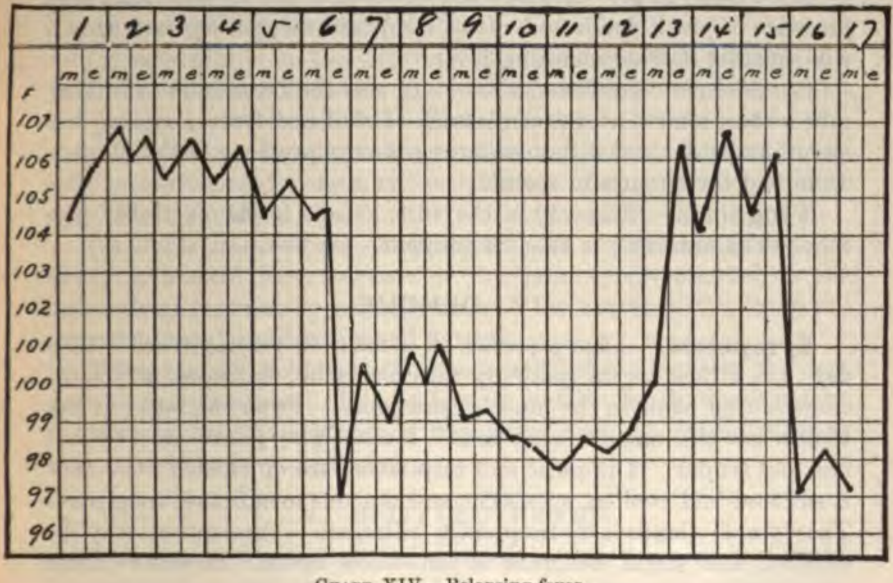


CHART XIV.—Relapsing fever.

is high) are common, and the spleen swells at an early period. In the young there may be nausea, vomiting, and convulsions.

The fever remains high, usually for 7 days (rarely 3 to 10), and falls by crisis (Chart XIV), with profuse sweating, sometimes diarrhœa, and (in elderly persons) with symptoms of collapse. In another week (14th day of disease) the attack may be repeated. The relapse is usually shorter than the original attack. A second relapse may occur about the 21st day, and a 3d, 4th, or 5th relapse may protract the disease. During the apyretic periods the patient may feel quite well and be about. The finding of the spirilla in the blood decides the diagnosis.

Complications.—Uncommon, but pneumonia is not infrequent, and nephritis, hæmaturia, jaundice, hæmatemesis, rupture of the spleen, ophthalmia, abortion, and post-febrile paralysis have been noted.

Differential Diagnosis.—(1) *Typhoid Fever.*—In the early cases of an epidemic the diagnosis of an irregular typhoid may be made, but the course of the temperature declares quite certainly the nature of the disease, and the blood examination with the finding of spirilla makes a positive diagnosis of relapsing fever.

(2) *Yellow Fever.*—Absence of spirilla and splenic swelling, with presence of black vomit and other symptoms of yellow fever (*q. v.*), will separate it from relapsing fever.

(3) *Malarial Remittent Fever.*—In this the fever is intermittent rather than remittent, the paroxysms of chill and fever are more frequent, and the blood examination reveals the presence of the plasmodium and the absence of spirilla.

Prognosis.—Recovery is the rule, except in old or feeble persons. The mortality is about 4 per cent.

IV. DENGUE

Symptoms.—After a period of incubation varying from 3 to 5 days, the attack begins suddenly with headache, chilliness, and atrocious aching pains in the muscles and joints. From the latter symptom arises the name "break-bone" fever. The joints are swollen, red, and tender. The pulse and respiration are quickened; the face is suffused and swollen, eyes, skin, and mucous membranes congested. The lymph glands are frequently enlarged. Prostration may be decided, and slight delirium is sometimes noted. In severe cases hemorrhages from the mucous membranes (epistaxis, hæmoptysis, hæmatemesis, etc.) may occur. Extreme hyperæsthesia of the skin has been observed. In the majority of cases an exanthem occurs, sometimes macular and resembling measles, or diffuse like that of scarlet fever, or papular or urticarial. It has no definite time of appearance and is not distinctive in its characters. In many instances it first appears upon the hands and feet, thence spreading over the greater part of the surface of the body.

The fever steadily rises during the first 3 or 4 days, reaching a maximum of 103° to 107°, at which time it rapidly remits, with free sweating and an almost total disappearance of the painful symptoms. The period of apyrexia lasts from 2 to 4 days, and the seizure is then repeated in a milder form. The average duration of the entire attack is from 7 to 10 days.

Complications.—These are uncommon. Convulsions may attend the onset of the disease (in children); and insomnia, active delirium, hyperpyrexia, pericarditis, and, in rare instances, meningitis may occur.

Differential Diagnosis.—(1) *Yellow Fever.*—As dengue and yellow fever are frequently prevalent at the same time, the two diseases are apt to be confounded. The symptoms which in the majority of cases determine the disease to be yellow fever are the facies, red and injected eyes, flushed face *plus* an icteric hue, slow pulse with high fever, albuminous urine, black vomit, and early jaundice (2d or 3d day). Nevertheless, good authorities may disagree, as in Texas during the epidemic of 1897.

(2) *Influenza.*—In this there is usually no eruption (except herpes), the fever does not show the striking remissions of dengue, and the joints are not implicated. Examination of the nasal and bronchial secretions will discover the bacillus of Pfeiffer. A question can only arise in sporadic cases, as the existence of an epidemic of either will be recognisable.

(3) *Acute Rheumatism.*—A sporadic case might be mistaken for acute rheumatic fever because of the joint symptoms, but in the latter there is generally no eruption, and the course of the fever will prove to be quite unlike that of dengue.

Prognosis.—Seldom fatal, death resulting only from the rare serious complications. The patient is usually much weakened by the disease, and the stiffness of the joints and the muscular pains may be extremely persistent.

V. SPOTTED FEVER

The causative agent of the spotted or tick fever of the Rocky Mountains is an animal parasite (*Piroplasma hominis*), amœboid but not pigmented, found both within and without the red cells. This statement is based upon the studies of Wilson, Chowing, and Anderson. It is best stained with Wright's stain, followed by methylene blue. Inoculation is through the bite of the wood tick. The disease occurs exclusively in the spring and early summer (from the middle of March to the middle of July).

The special habitat of this fever is in the Bitter Root Mountains of Western Montana, and it is localized in a comparatively small area. It is found also, in a milder form and with much less frequency, in Nevada, Oregon, Wyoming, and Idaho. It affects mainly the ranchmen and herders of these regions.

It is proper to state here that some doubt has been cast upon the existence of the *Piroplasma hominis*, by the comparatively recent investigations of Craig and Stiles, these observers failing to verify the previous work done upon this subject.

Symptoms.—Incubation, 3 to 10 days. Onset with chilliness, malaise, nausea, and finally a distinct chill. Pain in the back and head, with general muscular soreness, is present. Constipation exists; the conjunctivæ are congested, becoming yellowish; nosebleed is always present, sometimes severe and continued. Maximum of fever is reached on the 8th to the 12th day, after which there is a gradual decline to normal in favorable cases. The pulse is out of proportion to temperature, 110 to 140. The eruption appears on the 3d day, first on wrists and arms, then spreading, but appearing last and least on the abdomen. It is bright and macular, varying from pin point to split pea in size, becomes petechial, and disappears with the fever. The spleen is early enlarged, and to a considerable degree. The liver is usually slightly enlarged. Nausea may be extreme in the fatal cases. There is no delirium and the mind generally remains clear throughout.

Diagnosis.—Etiology (tick bite), chill, pain in back and head, muscle soreness, constipation, macular eruption appearing first on the wrists on the 3d day and becoming petechial and disappearing with the decline of the fever, will serve to make the diagnosis from meningitis, typhoid fever, peliosis rheumatica, and typhus, which latter it most closely resembles. An absolute diagnosis can only be made by finding the parasite in the blood. The disease is not contagious.

Prognosis.—The mortality is very irregular, 2 to 70 per cent (different localities). The greatest mortality is in Montana; the least in Nevada, Idaho, and Wyoming.

VI. TRYPANOSOMIASIS (SLEEPING SICKNESS)

Etiology.—A trypanosome, conveyed by the tsetse fly (*Glossina palpalis*) and inoculated through its bite. The organism is found in the blood and cerebro-spinal fluid, and in large numbers in the lymph glands.

Occurs only in West Africa (especially Uganda); endemic and epidemic. Negroes alone (with very rare exceptions) are attacked; children under three rarely show the disease.

Symptoms.—The incubation period is very long, several months to 7 years (?). The onset is very insidious. Three stages are described. (1) Early stage. There is a gradual decrease in activity, with increasing lassitude; headache, vague ephemeral pains are present; speech becomes slow and hesitating; pyrexia of 101° to 102° is present; pulse, of low tension, 90 to 130. Especially characteristic is an arrythmical, jerky tremor of the tongue, which may be an early symptom. The lymphatic glands begin to enlarge. (2) Trembling stage. This may be ushered in by nervous crises, such as epileptiform fits, or attacks of mania or melancholia. In other cases it comes on gradually. There is a marked alteration in the expression and general appearance, accompanied by a change in manner and disposition. The twitching of the tongue becomes more marked, and the tremor spreads to the cheeks and lips and to the body generally. The muscles become flabby, the face loses its expression, and the limbs their strength. The speech often becomes mumbling and blurred. The eyes take on a vacant, tired look. There is a disinclination to move or speak, with an increasing tendency to sleep, and the patient passes finally into (3) the lethargic stage. The condition becomes one of stupor and finally coma. The temperature is subnormal, the pulse slow and of very low tension. The sphincters relax and death supervenes, sometimes suddenly, but usually very slowly, the vital functions seeming to dwindle away.

The duration of the disease averages 4 to 8 months. Some cases show a more acute course, while others are much more prolonged, periods occurring in which the symptoms remit and the disease appears to be checked.

The prognosis is hopeless.

VII. CEREBRO-SPINAL MENINGITIS

This disease, most common in children and young adults, and caused by the *Diplococcus intracellularis meningitidis*, presents remarkably wide clinical variations. It is both epidemic and sporadic, and is moderately contagious. The entrance-route of the organism is probably by way of the nasal passages.

Symptoms.—(1) *Ordinary or Common Form.*—After an unknown period of incubation the disease begins abruptly with a violent headache, severe chill, fever (101° to 102°), vomiting, and pain in the back and limbs. In children a convulsion may occur. Very soon the muscles of the neck and back become painful and stiff, with cervical retraction or even opisthotonus. There is photophobia, hyperacusis, delirium (which may be violent), followed in bad cases by stupor or coma. Strabismus (frequent), nystagmus, ptosis, irregularity of the pupils, and facial spasm may be noted. Impairment of sight or hearing may develop. There is often general cutaneous hyperæsthesia, and tenderness along the spine, with tremor, and occasional tonic or clonic spasms of the extremities. The joints may become red and swollen. Kernig's sign (page 300) is usually present.

The fever is irregular and not characteristic. In severe or fatal cases the temperature may rise to 105° to 108°. The pulse is full and rapid, in rare instances abnormally slow. The respiration is not increased, unless pneumonia coexists, but may be of a sighing or even a Cheyne-Stokes character. The bowels are generally constipated, the abdomen is not tender, and vomiting is present, as a rule, only at the onset. The spleen is usually swollen. The urine may be albuminous, and contain sugar and, in the severer cases, blood. There is a leucocytosis, the increase affecting the polymorphonuclear cells. The eruption is variable and not always present. Herpes is frequently seen; in many cases blotchy purple spots and petechiæ are found over the entire surface of the body (hence the name "spotted fever"). Urticarial, erythematous, or roseolous rashes have been observed.

In many cases a certain diagnosis may be made by lumbar puncture and examination of the fluid for the *Diplococcus intracellularis*. The organism may frequently be found in the nasal secretions.

The duration is variable. An acute case may last but a few hours, a chronic case for several weeks, or even months. The majority of deaths occur within the first 5 days. In favourable cases, at the end of this time improvement becomes manifest.

(2) *Malignant, Fulminant, or Apoplectic Form.*—There is a sudden onset, severe chill, perhaps a convulsion, headache, moderate fever, extreme prostration, and purpuric spots. Muscular spasm, slow, weak pulse, and stupor usually appear, and death takes place from collapse in from 5 to 24 hours.

(3) *Abortive Form.*—The attack begins with great intensity as in the ordinary form, but in 2 or 3 days there is a prompt subsidence of the symptoms, with a rapid convalescence.

(4) *Mild Form.*—During an epidemic of the disease there are walking cases with headache, nausea, vomiting (occasional), vertigo, languor, and slight pain and stiffness in the muscles of the neck and back. Fever is slight or absent, and only the presence of the epidemic enables a diagnosis.

(5) *Intermittent Form.*—The fever and the symptoms remit or

intermit every day or every 2d day, or present an irregular remittent temperature like that of pyæmia.

(6) *Chronic Form.*—There is a series of recurrences of the fever with a complex symptomatology, dependent possibly upon chronic hydrocephalus or abscesses of the brain (OSLER, HEUBNER).

Complications and Sequelæ.—Pleurisy, pneumonia (frequent), bronchitis, pericarditis, arthritis (frequent), and parotitis may occur. Chronic headaches (meningitis), chronic hydrocephalus, aphasia, mental impairment; defective vision from optic atrophy, retinitis, or keratitis; defective hearing from inflammation of the middle or internal ear, or neuritis of the auditory nerve; and paralysis of other cranial nerves, may result from an attack of this disease. Nephritis with hæmaturia has been observed as a complication.

Differential Diagnosis.—In sporadic or doubtful cases a diagnostic lumbar puncture should be made (page 711) and the fluid examined for the characteristic organism.

(1) *Typhoid Fever.*—In the majority of cases of this disease the slow development of the symptoms, the temperature curve, the abdominal symptoms, and the Widal reaction will ultimately separate it from cerebro-spinal meningitis. There is, as a rule, no cervical pain and retraction, no herpes, and no leucocytosis. In exceptional cases the cerebral form of typhoid fever begins abruptly, with symptoms indistinguishable from those of cerebro-spinal meningitis.

(2) *Typhus Fever.*—In this the fever is more regular than in cerebro-spinal meningitis; facial paralysis, muscular rigidity, and the severe cervico-dorsal pains are absent.

(3) *Pneumonia.*—When this is complicated by meningitis, the pneumonia precedes the meningitis, there is less pain, and the cervical retraction is not so marked as in cerebro-spinal fever. But if the case is not seen from the beginning, one can not be certain that the meningitis is secondary to the pneumonia, and it may be difficult or impossible to separate it from a case of cerebro-spinal fever complicated with pneumonia.

(4) *Acute Rheumatic Fever.*—On account of the redness and swelling of the joints which may occur in cerebro-spinal meningitis it may be mistaken for acute rheumatic fever, but in the latter the joint symptoms occur at the onset, and there is an absence of cervical retraction, muscular rigidity, facial paralysis, and cutaneous eruptions.

(5) *Tuberculous Meningitis.*—Compared with cerebro-spinal fever, the onset of tuberculous meningitis is not so sudden, the aching, hyperæsthesia, and cervical retraction are less, and there are usually no cutaneous eruptions; while retraction of the abdomen, irregular

pulse, and Cheyne-Stokes respiration are much more common. Furthermore, a pre-existing tuberculous focus may be found, and tubercles discovered in the choroid by ophthalmoscopic examination.

Prognosis.—The mortality varies from 20 to 75 per cent, and is greatest in children under 5 years. High fever, recurring convulsions, and profound coma usually presage a fatal termination.

VIII. INFLUENZA

Symptoms.—After an incubation of from 1 to 4 days the disease (caused by Pfeiffer's bacillus) sets in abruptly with chilliness, or even a severe rigour. The fever is extremely variable, running from 100° to 105°, and lasts with remissions from 1 to 10 days. There is in almost all cases severe headache and general aching, with a degree of prostration out of all proportion to the apparent cause. Delirium is not infrequent. The symptomatology of the disease is variegated, and one symptom group may quickly merge into another. The following forms of the disease are recognised:

(1) *Respiratory Form.*—The early symptoms are those of a severe coryza. Usually pharyngitis, laryngo-tracheitis, and bronchitis follow. The general aching and extreme weakness may be the only distinguishing characteristics. Very frequently a slight patchy broncho-pneumonia, with few physical signs, coexists. The cough is apt to be paroxysmal, violent, and extraordinarily persistent.

(2) *Nervous Form.*—In many cases slight fever, with atrocious headache, pain in the back and limbs, and marked weakness may be the only symptoms.

(3) *Gastro-intestinal Form.*—In a certain proportion of cases, nausea, vomiting, abdominal pain, and profuse watery or serous diarrhœa, with prostration amounting at times to collapse, may constitute the evidence of the disease.

(4) *Typhoid or Febrile Form.*—In some instances, fortunately infrequent, there may be a continued fever, with delirium, dry, brown tongue, and other symptoms of the typhoid status. In certain cases the fever remits or intermits, with recurring chills, and simulates malaria.

Complications, Sequelæ, and Occasional Symptoms.—(1) *Respiratory Organs.*—Broncho-pneumonia (common), lobar pneumonia, pleurisy terminating in empyema (rare), pulmonary gangrene and abscess (rare), pulmonary œdema (sequel of pneumonia), and enlargement of the bronchial glands.

(2) *Circulatory Organs.*—Tachycardia, bradycardia, or persistent irregularity of the pulse; angina pectoris (influenzal), usually without discoverable organic changes, temporary and recoverable; cardiac

weakness; endocarditis, pericarditis, phlebitis and thrombosis of different vessels. Recurring syncope is not infrequent at the onset of the disease, especially in women.

(3) *Nervous System.*—True meningitis, or the symptoms of meningitis which disappear in a day or two; encephalitis, with resulting hemiplegia or monoplegia; and abscess of the brain, are uncommon complications. Various forms of neuritis are not infrequent. Active delirium, depression of spirits, melancholia, dementia, persistent insomnia, neuralgias, and migraine have been noted. Neurasthenia, sometimes severe and prolonged, is a not uncommon sequel.

(4) The spleen may become enlarged, and catarrhal jaundice has occurred with some frequency; (5) hæmaturia, acute congestion, and acute nephritis are not extremely rare; (6) conjunctivitis is frequent, iritis uncommon, and optic neuritis is rare; (7) acute otitis media is common, terminating occasionally in mastoiditis, and vertigo, as a result of labyrinthine disease, has been observed; (8) herpes is frequently seen; occasionally diffuse erythema and purpuric spots are witnessed.

Differential Diagnosis.—In doubtful, anomalous, or suspected sporadic cases a bacteriological examination of the bronchial or nasal secretions should be made in order to find Pfeiffer's bacillus, which, if present, will declare the disease to be influenza. In the midst of an epidemic there is usually no difficulty in making a diagnosis, nor in a sporadic case if the symptoms come under the head of one of the recognised types. The cardinal symptom is the excessive and disproportionate weakness.

(1) *Typhoid Fever.*—Influenza begins suddenly, and lacks the regular temperature curve, rose spots, and positive Widal reaction of typhoid fever.

(2) *Cerebro-spinal Meningitis.*—In certain cases of influenza the sudden onset, headache, backache, delirium, and muscular stiffness may afford a clinical picture exactly like that of cerebro-spinal meningitis, and the differential diagnosis must depend upon the bacteriological examination; in the one case for the Pfeiffer bacillus, in the other for the meningococcus.

(3) *Broncho-pneumonia.*—If the question arises as to whether a given case of broncho-pneumonia is of influenzal origin, a rather peculiar and anomalous combination of physical signs may answer the question in the affirmative, viz., varying degrees of dulness over both chests posteriorly, with weak respiratory murmur, impaired transmission of voice sounds, and a shower of fine and subcrepitant râles at the end of a deep inspiration, heard in scattered areas or patches, especially at the bases. The combination is probably

indicative of a mixture of broncho-pneumonic spots and collapsed lobules.

Prognosis.—In the large majority of cases recovery occurs. The mortality varies from ½ to 2 per cent, and is generally due to a complicating severe pneumonia, especially when affecting the very young or the aged.

IX. WHOOPING COUGH

Symptoms.—Period of incubation varies from 7 to 10 days. Two stages are recognised. (1) *Catarrhal Stage.*—Symptoms those of an ordinary cold: coryza, injected conjunctivæ, and a bronchial (perhaps slightly spasmodic) cough, with slight fever. In a week or 10 days, instead of improving, the disease enters upon the

(2) *Paroxysmal Stage.*—This stage begins with the first "whoop" or "kink." The cough becomes paroxysmal, and the child is usually conscious of the approach of a seizure for a moment or two prior to its occurrence and endeavours to restrain it. The paroxysm, frequently precipitated by emotional causes, begins with a series of short coughs, followed by a deep inspiration during which the characteristic whoop is heard. A 2d, 3d, or 4th series of coughs, each ending with a whoop, may follow, the paroxysm terminating with the ejection of thick mucus, and frequently with vomiting. Physical examination of the lungs is negative, except for the evidence of a slight bronchitis. The number of paroxysms varies from 4 or 5 to 40 or 50 in 24 hours. During the seizure the face is swollen, congested, and cyanotic; the veins are full and the eyeballs project. Urine and fæces may be passed involuntarily. After the disease is well developed the facies is very characteristic, with its swollen, dusky appearance, reddened eyes, and puffy, pinkish lids. Subconjunctival extravasations, and an ulcer under the tongue, are not uncommon. The paroxysmal stage lasts usually 3 or 4 weeks, after which the attacks grow milder and gradually cease. The average duration of a case of moderate severity is 6 weeks; in aggravated cases it may be 4 months.

Complications and Sequelæ.—Epistaxis, hæmoptysis (infrequent), intestinal hemorrhage (rare), subconjunctival extravasations, and petechial spots on the forehead and face may occur as a result of the extreme venous congestion. The pulmonary complications are important, of which broncho-pneumonia (sometimes tuberculous) and pulmonary collapse are most common; lobar pneumonia, pneumothorax or interstitial emphysema (from strain), pleurisy, and enlargement of bronchial glands (very common). Convulsions are not uncommon, sometimes succeeded by a profound coma; hemiplegia, monoplegia, and subdural hemorrhage are rare events. Irregularity of the pulse, dilatation of the right ventricle, and perhaps permanent valvular lesions may result from the great paroxysmal strain upon the heart. Acute nephritis may occur (20 per cent of 200 cases, ANDERS). As sequelæ, chronic bronchitis and pulmonary tuberculosis may be found. Gastro-intestinal catarrh is not an infrequent following, and hernia may occur as a result of strain.

Diagnosis.—The disease can not be recognised until the paroxysmal stage, when the "whoop" is unmistakable. The paroxysmal cough of influenza may closely simulate pertussis, but the onset in the former disease is sudden, and the spasmodic character of the cough is usually marked from the outset. Mild cases in which the whoop does not develop may remain doubtful. The swollen face and eyes are of much diagnostic importance. The *mortality* of pertussis, with its complications, amounts to 7 or 8 per cent, especially during the first 2 years of life.

X. EPIDEMIC PAROTITIS (MUMPS)

Symptoms.—After an incubation of 2 to 3 weeks there is lassitude, with fever (rarely above 101°), and pain under one (usually the left) ear, increased by taking an acid. Headache and nausea are sometimes present. Swelling begins on the affected side, which in 2 or 3 days becomes very extensive. The bulk of the swelling lies under the lobe of the ear, which is pushed outward, and extends forward in front of the ear and backward under the sterno-mastoid muscle (anatomical situation of the parotid gland). The opposite gland usually follows suit within a day or two. The pain is tensive rather than acute, the mouth can scarcely be opened, and there is difficulty in mastication, swallowing, and speaking. Tinnitus and slight dulness of hearing are very common. The swelling lasts from 6 to 10 days. As a rule the disease is of a mild type, but in very exceptional cases there may be high fever (103° to 104°), with delirium, prostration, and other symptoms of the typhoid status.

Complications and Sequelæ.—Orchitis, usually after the parotitis has subsided, sometimes resulting in atrophy; and at the same period, ovaritis (rare), mastitis or tenderness of the breasts, and vulvovaginitis, almost invariably after puberty. Other complications are meningitis, acute mania, insanity, facial paralysis, convulsions, hemiplegia, peripheral neuritis, endocarditis, arthritis, jaundice, albuminous urine, acute uræmia, otitis media (not uncommon), disease of the auditory nerve with permanent deafness, disease of the lachrymal gland, and optic atrophy. The majority of these are either very rare or occur very infrequently. Suppuration of the gland in epidemic parotitis seldom occurs. The gland may become chronically enlarged.

Differential Diagnosis.—Mumps occurs mainly in children. The diagnosis is usually readily made by noting that the bulk of the swelling is below and in front of the ear, and that the lobe of the ear is pushed outward. Inspection of the throat will show the absence of a tonsilitis or other inflammation which might cause swelling of the cervical glands and thereby simulate a parotitis. A parotitis may result from various septic inflammations, but the gland usually suppurates, an occurrence which alone will almost invariably separate it from the epidemic form, and the presence of the causative infection, as a rule, will have been recognised. The *prognosis* is favourable in almost all cases.

XI. SMALLPOX (VARIOLA)

Four varieties of smallpox are recognised: the discrete, of moderate severity; the confluent, of great severity; the hemorrhagic or malignant; and varioloid, or smallpox as modified by vaccination.

Symptoms.—(1) *Discrete Form.*—After an incubation of from 8 to 15 days the disease usually begins with a chill (perhaps recurrent) in adults, and a convulsion in children, followed by intense headache, excessive lumbar pain, nausea, and vomiting. The temperature rises rapidly during the 1st and 2d days, reaching 103° to 105°. The face is flushed, eyes bright, and delirium present, espe-

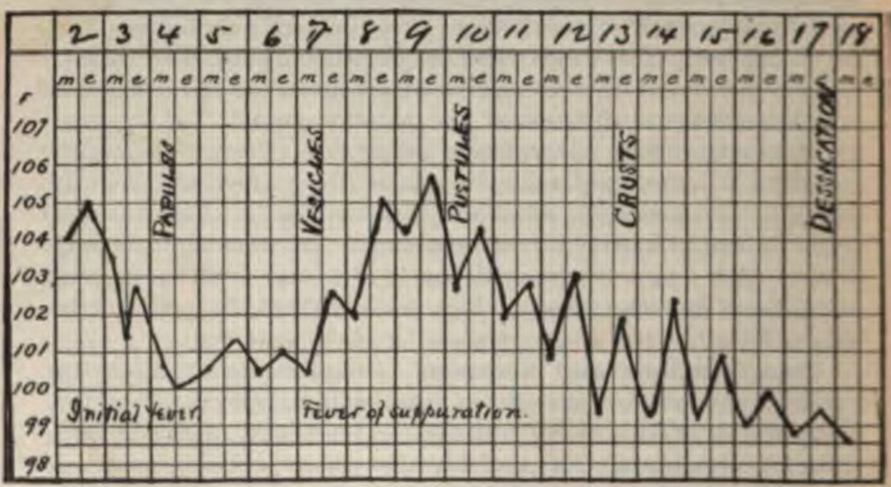


CHART XV.—Smallpox.

cially if the fever runs high. The pulse is rapid (120 to 140), full, seldom dicrotic. Generally the bowels are constipated, the urine scanty and albuminous. The initial fever continues high until the 3d or 4th day, when a decided remission occurs, lasting until the 7th or 8th day, when it again rises (fever of suppuration), fluctuating more or less, and in favourable cases gradually falls to the normal by the 18th day of the disease (Chart XV).

The characteristic eruption appears on the 3d or 4th day of the disease as small red spots, first on the forehead and wrists, appearing within the next 24 hours upon other parts of the face and limbs, and to some extent upon the trunk. With the appearance of the eruption the fever falls and there is an improvement in the general symptoms. The rash is at first macular, changing rapidly into rounded papules which feel like shot under the skin. The lesions may occur also in the mouth, pharynx, and larynx. About the 5th or 6th day of the disease they become vesicular and umbilicated, and about the 8th day are converted into non-umbilicated pustules. Each pustule is surrounded by a red areola, and the intervening skin is swollen. With pustulation the fever returns and the general symptoms reappear. About the 11th or 12th day of the disease the pustules begin to desiccate and form scabs or crusts, which cling to the skin for a week or longer. The maturation of the eruption occurs first upon the face and follows the order of its appearance. More or less pitting of the skin results. In the discrete form the lesions are not very abundant and do not coalesce.

(2) *Confluent Form.*—The papules are numerous, thickly set, and soon coalesce, although the confluence may not take place until the stage of pustulation. They are most abundant on the face and hands, less so on the limbs, and may be scattered and discrete on the trunk. The initial symptoms of the confluent form resemble those of the discrete variety, but are usually of greater severity. Delirium, stupor, subsultus; salivation, diarrhœa; extreme swelling of the cervical glands; hoarseness, cough, and offensive discharges from the nose and throat due to the existence of the eruption in the nose, mouth, pharynx, and larynx, are present to a varying extent.

(3) *Malignant or Hemorrhagic Form.*—This presents 2 varieties. *Black* or *purpuric smallpox*, beginning with intense fever, lumbar pain, and prostration, and an unusually rapid respiration rate. On the 2d or 3d day small ecchymoses appear on various portions of the skin, growing in size and number, so that in the severest cases the greater portion of the cutaneous surface may be purple or blackish in colour. Extensive conjunctival ecchymoses may also be present. Hemorrhages from the mucous membranes are common, viz.,

hæmaturia, hæmatemesis, intestinal hemorrhage, hæmoptysis, or metrorrhagia. As death occurs between the 3d and 6th day, the variolous eruption may not have time to appear, except that scattered papules may be found.

In *hemorrhagic pustular smallpox* the disease progresses like an ordinary severe case up to the vesicular or pustular stage, at which time the pocks become hemorrhagic, bleedings from the mucous membranes occur, and death usually takes place from the 7th to the 9th day.

(4) *Varioloid.*—The initial symptoms are mild, a few papules appear on the face and hands, and the fever of suppuration does not occur (Chart XVI).

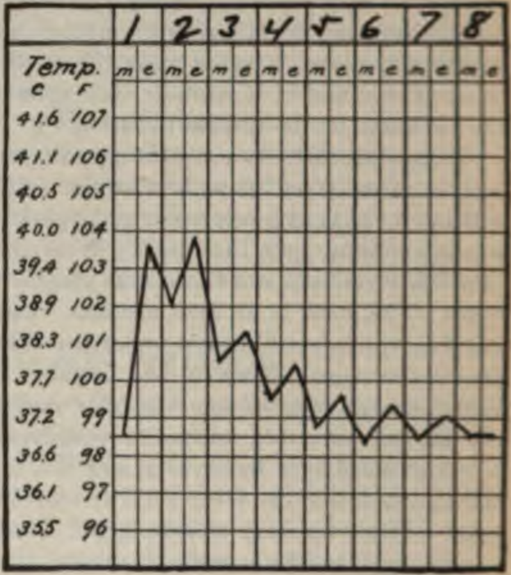


Chart XVI.—Varioloid.

Complications and Sequelæ.—Broncho-pneumonia (common), lobar pneumonia (rare), pleurisy (common); laryngitis, œdema of glottis, perichondritis or necrosis. Endocarditis and pericarditis are rare, myocarditis not infrequent. Diarrhœa not uncommon, especially in children; parotitis is rare; vomiting usually not protracted; swollen spleen and liver, albuminuria common, acute nephritis infrequent; hæmaturia, orchitis, and ovaritis very seldom seen. Neuritis, local or general; diffuse myelitis with paraplegia; hemiplegia and aphasia due to encephalitis (rare); convulsions, common in children; active delirium; coma; insanity (post-febrile) seldom met; epilepsy rarely. Arthritis, perhaps suppurative; acute necrosis of bone. Boils (frequent); local gangrene seldom; acne. Otitis media; conjunctivitis, iritis, and corneal inflammation.

Differential Diagnosis.—The so-called "initial" rashes may cause error. There are 2 forms, one resembling the rash of scarlet fever, the other that of measles. Generally they are limited to the lower abdomen, the inner surface of the thighs, or to the axillary regions and sides of the chest, and appear upon the 2d or 3d day of the disease. Until shotty papules form it is difficult or impossible to recognise the true nature of these rashes, although the prodromal symptoms may be sufficiently characteristic in each instance.

Ordinarily if an epidemic is present the diagnosis of smallpox is readily made; but in sporadic cases, or during the period prior to the appearance of the papules or vesicles, variola may require differentiation from the following diseases:

(1) *Cerebro-spinal Fever.*—The hemorrhagic form of smallpox may be mistaken for the purpuric spots and hemorrhages of cerebro-spinal fever, and irritative meningeal symptoms may also be present. If death occurs early a correct diagnosis may not be made, but otherwise the papular and pustular development of the eruption will make the discrimination.

(2) *Typhus Fever.*—The onset of this disease resembles that of smallpox, but may be discriminated by the fact that in typhus fever the macular or petechial rash appears first on the trunk and does not become papular or pustular; nor is there a remission of the fever coincidently with the appearance of the eruption.

(3) *Varicella.*—It is at times exceedingly difficult to discriminate between varicella and varioloid or very mild cases of discrete variola.

In *varicella* the eruption usually appears first upon the trunk, front or back, beginning more rarely on the forehead and face. The vesicles vary in size, are often oval in shape, are rather superficial without a marked red areola, and not infrequently flattened or umbilicated. They arrive in successive crops, so that by the 4th day lesions in the various stages of papules, vesicles, and pustules can be seen side by side, which, together with their abundance upon the trunk, constitute most important differential points. Moreover, the slightness of the constitutional symptoms (fever, backache, prostration) is notable in varicella, and there is no fever of suppuration. The knowledge of previous exposure to the disease may be helpful.

In *varioloid* or mild *variola* the eruption usually appears first upon the forehead and hands, the lesions are of uniform size, not superficial, are more pustular, and the various stages do not exist side by side. The eruption is most abundant on the face, hands, and extremities. The constitutional symptoms are, as a rule, of a more decided character. There may be a history of exposure to a known case of smallpox.

Prognosis.—Varioloid recovers; in discrete smallpox the prognosis is good; in confluent forms, grave; in the malignant and severe hemorrhagic forms death is almost inevitable.

XII. VACCINIA (COWPOX)

Symptoms.—Two or three days subsequent to vaccination a papule with a red areola is seen at the point of inoculation. It increases in size, and by the 6th day becomes an umbilicated circular vesicle, which grows larger and is distended with lymph. On the 10th day it becomes purulent, and on the 12th day desiccation begins. The scab separates in from 3 to 4 weeks from the date of inoculation, leaving a pitted scar. There is usually some fever and constitutional disturbance, beginning on the 3d and terminating on the 9th day. The axillary or inguinal glands, according to the site of the operation, become swollen and tender.

Irregularities and Complications.—A non-characteristic, over-rapid development of the vesicle, with the formation of a crust by the 7th or 8th day, or a retarded formation, requires revaccination. Injury of the vesicle causes inflammation or the formation of an ulcer. Secondary vesicles may form in the neighbourhood of the pock; and very rarely there is a general pustular eruption in various parts of the body, successive crops appearing for several weeks.

The possible complications are as follows (Acland):

1. During the first 3 days: Erythema; urticaria; vesicular and bullous eruptions; invaccinated erysipelas. 2. After the 3d day and until the pock reaches maturity: Urticaria; lichen urticatus, erythema multiforme; accidental erysipelas. 3. About the end of the 1st week: Generalized vaccinia; impetigo; vaccinal ulceration; glandular abscess; septic infections; gangrene. 4. After the involution of the pocks: Invaccinated diseases—for example, syphilis.

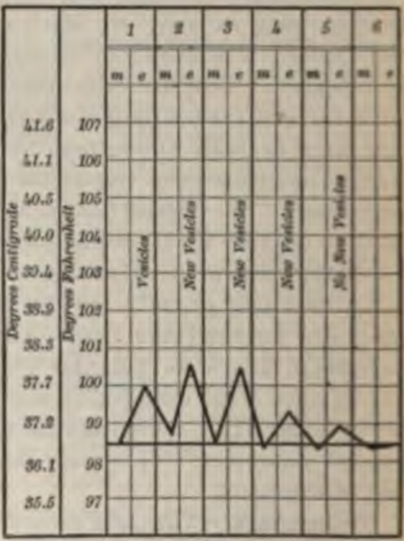


Chart XVII.—Varicella.

XIII. VARICELLA (CHICKEN POX)

Symptoms.—After a period of incubation (10 to 15 days) there is a slight fever, sometimes chilliness, vomiting, and aching in back and legs, followed in 24 hours by the appearance of a papular eruption, which in a few hours becomes vesicular. The lesions are discrete, often ovoid, somewhat superficial, flattened, not infrequently umbilicated, and contain clear or cloudy fluid. In 36 or 48 hours the contents become purulent, and during the 3d and 4th days shrivel into dark-brown crusts, which become detached, usually without leaving scars. The vesicles appear in successive sets during the first 2 or 3 days of the disease, so that lesions in various stages of development may lie side by side (Chart XVII).

Complications.—Ordinarily the disease is mild. If the vesicles are disturbed by scratching, ulcers may form, and in some cases the vesicles may be unusually large (bullæ). In rare instances varicella may be hemorrhagic, with bleeding from the mucous membranes and the presence of ecchymotic spots on the skin. Other occasional complications are acute nephritis, infantile hemiplegia, and gangrene immediately around the vesicles in tuberculous or weakly children. The *diagnosis* is from smallpox (page 761), and the *prognosis* is almost invariably favourable.

XIV. SCARLET FEVER (SCARLATINA)

Symptoms.—Period of incubation varies from 1 to 7 days (usually 2 to 4). The disease begins, as a rule, abruptly, rarely with a chill, but in the majority of cases with vomiting. In young children convulsions are common. The fever rises rapidly (perhaps 104° to 105°) during the 1st day, and the child complains of sore throat.

At the end of the 1st or the beginning of the 2d day the rash appears, first on the neck and chest, whence it spreads over the whole body, usually within the next 24 hours. From a distance the skin is uniformly and brilliantly scarlet, but on close inspection the rash is seen to consist of fine, closely set red points, the intervening skin being diffusely red. It disappears on pressure, the anæmic spot having a slightly yellowish tint. Not infrequently the rash is coarsely punctiform, the intervening skin remaining white; or the rash may occur in patches; or it may be finely papular; or with an intense rash there may be punctate petechiæ; or sudamina may form. The rash persists for 2, 3, or 4 days, and then slowly disappears.

The mucous membrane of the mouth and throat is brilliantly red and more or less swollen; the tonsils are reddened, and often present a punctate or membranous exudate (lacunar tonsilitis). The tongue is at first covered with a white fur through which the swollen red papillæ project (strawberry tongue). In a few days the fur exfoliates (raspberry tongue). The pulse is unduly rapid (in children from 120 to 150 or over) in comparison with the temperature, and the respiration is accelerated. The spleen is somewhat enlarged. Headache, restlessness, insomnia, and nocturnal delirium are present i cases of notable severity, especially at the onset, disappearing as th rash becomes established. Aside from the initial nausea gastro-intestinal symptoms are not common. The urine is scanty, high-coloured, and often slightly albuminous. A few, usually hyalin casts are not uncommon. A leucocytosis is usually present.

In favourable cases the temperature gradually falls as the ras disappears, so that by the 7th or 8th day it reaches the normal (Char XVIII). At this period desquamation begins, the cuticle becoming

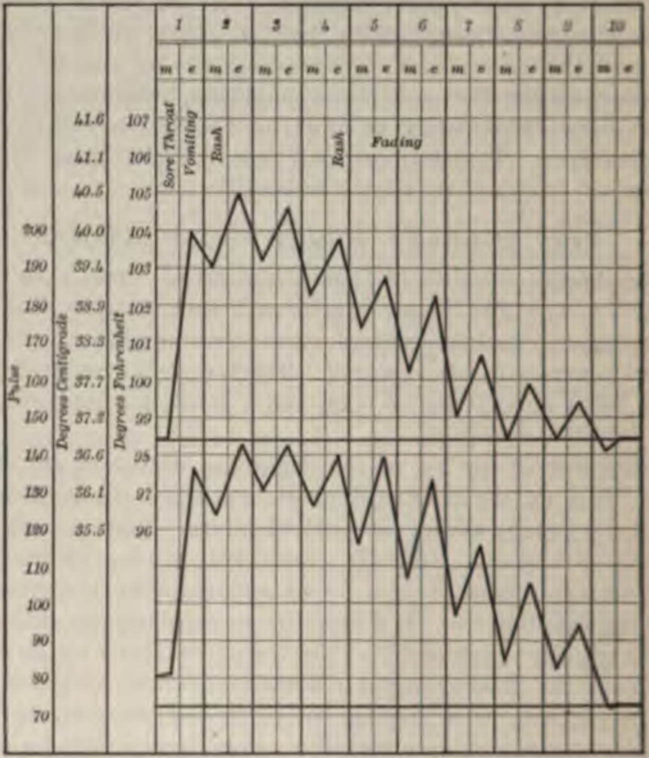


Chart XVIII.—Scarlet fever.

detached in small scales or large flakes. Beginning with the neck and chest, the desquamation is usually completed by the end of the 3d or 4th week, extending more rarely into the 7th or 8th week.

As scarlet fever varies greatly in severity, certain clinical types are recognised, which differ as follows from the cases of average intensity just described:

(1) **Mild Form.**—The rash may be present without vomiting, fever, or inflammation of the throat; or the tonsils may be inflamed without a rash; or fever, sore throat, and strawberry tongue may exist, and the rash be lacking. The presence of an epidemic or an indubi-able history of exposure may be the only evidence by which the nature of such cases may be surmised, unless the occurrence of some haracteristic sequel, acute nephritis in particular, throws an unhappy ight upon the primary attack.

The following forms are often spoken of as *malignant*:

(2) **Anginose Form.**—In this variety the throat symptoms are prominent. There is an intense inflammation of the tonsils, fauces, pharynx, and nasal chambers, with swelling and an extensive mem-ranous exudate. The cervical glands are enormously swollen, and he tissues of the neck become the seat of a brawny induration. The ischarges from the throat and nose are extremely offensive. There re high temperature (105° to 107°), cyanosis, stupor, diarrhœa, rapid, weak, and irregular pulse, and the child dies of an acute toxæmia, ometimes within 24 hours. If death does not ensue at such an early period, abscesses and sloughing of the tonsils and cervical tissues may ake place, and be further complicated by pneumonia and profound exhaustion.

(3) **Hemorrhagic Form.**—The rash is petechial, the spots steadily developing into large ecchymoses; and there may be hemorrhages from the mucous surfaces, particularly hæmaturia and epistaxis. The patient may die on the 2d or 3d day.

(4) **Atactic (Malignant) Form.**—The onset is sudden and intense, with chill or convulsion, and vomiting. The fever is high (107° to 108°), and there is headache, restlessness, and delirium, soon followed by coma. Death may take place within 24 or 36 hours, before the rash has had time to appear.

Complications and Sequelæ.—Nephritis of varying degrees of severity, occurring during the 2d or 3d, rarely the 4th, week of the disease, is usually recovered from, but may be fatal from acute uræmia or œdema of glottis. Endocarditis, pericarditis, and myocarditis are not uncommon. Pneumonia, pleurisy (often purulent). Otitis media (frequent and serious), sometimes followed by mastoiditis, necrosis, labyrinthine disease, thrombosis of the lateral sinus, meningitis, or cerebral abscess. Arthritis (scarlatinal "rheumatism"). Adenitis (of the submaxillary or cervical glands), leading in severe cases to suppuration, and in rare instances to chronic enlargement. Chorea occasionally occurs, with arthritis and endocarditis; and convulsions, hemiplegia, and spinal paralysis, and thrombosis of the cerebral veins have been noted very infrequently. Cancrum oris, single or double phlegmasia alba dolens, symmetrical gangrene of the extremities, and perforation of the soft palate are very rare complications.

Differential Diagnosis.—In the majority of cases the sudden onset, vomiting, high fever, extraordinarily rapid pulse, strawberry tongue, and the early appearance of the rash render a diagnosis of scarlet fever easily made. The following may require differentiation:

(1) **Acute Follicular (Lacunar) Tonsilitis.**—The onset of this disease may be quite indistinguishable from that of scarlet fever, but the appearance of the rash upon the 2d day will declare the presence of the latter.

(2) **Diphtheria.**—In certain cases of this disease there may be an erythema, but the rash is darker, generally confined to the trunk, and disappears earlier. The onset is less abrupt, there is usually a more marked degree of prostration, and a culture from the throat reveals the Klebs-Loeffler bacillus. Nevertheless, if the two diseases coexist, it may be quite impossible to affirm the fact.

(3) **Rubella.**—The face in this disease may strikingly resemble that of scarlet fever, but the rash is not punctiform, the fever is slight, the pulse is not so rapid—in short, the mildness of the constitutional symptoms will usually decide against scarlet fever.

(4) **Measles.**—In this disease the sore throat is usually absent; so also is a leucocytosis. The papular and blotchy character of the rash and its darker tint, its abundance upon the face, its later appearance (3d or 4th day of the disease), and the prodromal coryza and catarrhal symptoms will declare for measles.

(5) **Acute Exfoliating Dermatitis.**—This disease may furnish a very faithful clinical picture of mild scarlet fever. The vomiting, relatively rapid pulse, strawberry tongue, sore throat, ear complications, and nephritis of scarlet fever are, however, absent; and recurrent attacks are not uncommon. I consider it a good clinical rule to label as scarlet fever every case presenting a scarlatinous rash *plus* an exudate, punctate or membranous, upon the tonsils.

(6) **Drug Eruptions.**—Quinine, belladonna, potassium iodide, copaiba, and certain foods (lobsters, crabs) may cause an intense diffuse scarlatiniform erythema, but the sudden onset and rapid pulse of scarlet fever is lacking and the temperature is normal.

Prognosis.—Uncomplicated cases of moderate severity usually recover; severe and malignant forms are apt to prove fatal. The mortality varies with the epidemic from 5 to 30 per cent, mainly in children under 6 years of age. Hemorrhages, severe throat symptoms, œdema of the larynx, early delirium and subsultus, and hyperpyrexia are unpromising events.

XV. MEASLES (RUBEOLA)

ymptoms.—After an incubation of from 7 to 18 days (usu- 4) the disease begins with chilliness, sneezing, lachrymation, a, drowsiness, and, in 24 hours, cough. There is usually red- of the eyes, lachrymation, and photophobia. Headache, nausea, omiting may also be present. The temperature rises steadily he first 2 days, and may then remit to rise again with the appear- of the rash (Chart XIX).

he exanthem appears on the 4th day, first upon the face, as , red, flattened papules which grow larger and invade the trunk xtremities. The countenance has a mottled, swollen appearance, which, with the reddened and photophobic eyes, affords a very characteristic facies. The blotchy character of the rash is best seen upon the trunk. The eruption, except in severe cases, is distinctly discrete and disappears upon pressure. The exanthem can be seen upon the mucous surfaces of the mouth and throat. Koplik's spots (page 238) may be observed as a forerunner of the rash. The general symptoms do not lessen until the 6th day, at which time the fever generally falls by crisis, and the catar- symptoms slowly disappear. The rash lasts until the 6th or 7th nd then gradually fades. Desquamation occurs in the form of branny, almost unnoticeable scales.

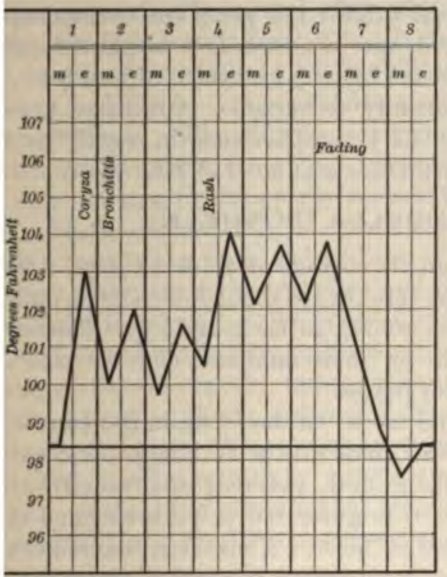


CHART XIX.—Measles.

he form of this disease known as hemorrhagic or "black" mea- ccurring in bad hygienic surroundings, may be grave and fatal. onstitutional symptoms are more violent, there is marked pros- n, the rash becomes petechial, and there are hemorrhages the mucous membranes. Death results from an overwhelming mia. Irregular cases in which the rash is present by the 2d day, or on the other hand is delayed until the 6th day, or in which the prodromal symptoms are present but the rash fails to appear, are not uncommon during an epidemic.

Complications and Sequelæ.—Catarrhal pneumonia from extension of the bronchitis which is a part of the disease, and lobar pneumonia (less common); laryngitis (not infrequent); otitis (frequent), diarrhœa (frequent). Rarer complications are tuberculosis, cancrum oris, stomatitis, vulvitis, and paraplegia or hemiplegia due to myelitis or neuritis.

Diagnosis.—(1) *Rubella.*—Prodromata, fever, and catarrhal symptoms are much milder, the rash appears earlier (1st or 2d day) and is more evenly distributed than in measles.

(2) *Scarlet Fever.*—In this disease the onset is more sudden, the fever is higher and does not exhibit the pre-eruptive remission, the sore throat is more marked, and the rash is a bright-red, uniform erythema, contrasting with the blotchy exanthem of measles.

Prognosis.—The mortality is variable. Ordinary, uncomplicated cases recover; pulmonary complications in weakly or tuberculous children are responsible for a number of fatal cases.

XVI. RUBELLA (RÖTHELN)

Symptoms.—After an incubation of 10 or 12 days, "German measles" begins with slight sore throat, slight fever (100°), chilliness, headache, coryza, and some aching in back and legs. These symptoms are often remarkable for their mildness, and the rash is frequently the first evidence of illness.

The rash appears on the 1st or 2d day, first on the face, whence it spreads over the trunk and limbs within 24 hours. It consists of slightly elevated, rounded, or oval, pale-red, and usually discrete spots; occasionally it is of a brighter red or pink tint and diffuse, resembling the rash of scarlet fever. A macular, rose-red eruption on the throat is a constant symptom. It persists for from 3 to 5 days, often leaving a slight pigmentation, and may be followed by a moderate branny desquamation. The glands, especially the cervical and posterior auricular, are swollen. Occasionally in cases the fever rises to 103° and the constitutional symptoms are well marked.

Complications.—Bronchitis, pneumonia, gastro-intestinal catarrh, albuminuria, and jaundice may occur, but complications of any kind are infrequent.

Differential Diagnosis.—(1) *Measles.*—From measles, rubella is to be distinguished mainly by the mildness of its symptoms. As the symptomatology is in many respects exactly similar it may be difficult to discriminate between light measles and severe rubella.

A rose-red tint of the rash and prompt swelling of the cervical and especially of the posterior auricular glands, point to the latter.

(2) *Scarlet Fever.*—The more sudden onset, the initial vomiting, the erythematous character of the rash, the longer duration and severer course of the disease will serve to separate it from rubella.

(3) *Urticaria.*—The characteristic wheals, intense itching, and usual absence of catarrhal symptoms will discriminate this disease from rubella.

(4) "*Fourth Disease.*"—The existence of a fourth exanthem, claimed by Dukes, is still unsettled. It is described as an independent disease of a benign type, closely simulating mild scarlatina.

XVII. DIPHTHERIA

Symptoms.—The period of incubation of this disease (Klebs-Loeffler bacillus) varies from 2 to 10 days; usually 2 or 3. The onset is with fever (102° to 104°), chilliness, headache, and pain in the back and limbs. A convulsion may occur in infants or very young children. Albuminuria is common. There is nothing distinctive in the initial symptoms. According to the site of the specific inflammation the following forms of diphtheria are recognised:

(1) *Pharyngeal Diphtheria.*—Sore throat, dysphagia, swollen and tender glands in the neck, and stiffness of the cervical tissues become manifest. The tonsils are swollen and covered with grayish or yellowish white membrane, which is usually adherent, and leaves a bleeding surface when forcibly stripped off. By the 2d or 3d day the membrane extends to the faucial pillars, perhaps also to the uvula and the posterior pharyngeal wall. In favourable cases the symptoms subside and the throat becomes clear of membrane by the 10th day. The membrane may be punctate and confined to the tonsils (lacunar or tonsillar diphtheria), simulating in every respect an ordinary follicular tonsilitis; or there may be a soft non-membranous exudate upon the tonsils alone; or the case may be so extremely mild that membrane or exudate does not appear, the throat presenting only the redness of an ordinary catarrhal inflammation.

(2) *Laryngeal Diphtheria.*—Membranous croup is in the large majority of cases laryngeal diphtheria; in a small minority a streptococcus inflammation. It is usually secondary, by extension from the pharynx, occasionally primary. A croupy cough, hoarseness, or aphonia, and, above all, evidences of progressive laryngeal stenosis, constitute the leading symptoms. As the narrowing of the glottic opening proceeds the breathing becomes stridulous, and dyspnœa and cyanosis become manifest. The supraclavicular, episternal, intercostal, and epigastric spaces are deeply retracted with inspiration and

bulge with expiration. The child is excessively restless, the nostrils work violently, and the sterno-mastoids become prominent during inspiration. Shreds of membrane may be coughed up. If the stenosis is not relieved the child passes into a semicomatose state and finally dies. The fever is usually slight and the general condition of the child is good. The membrane may extend into the trachea and the bronchial tubes.

(3) *Nasal Diphtheria.*—This may be primary, but is, as a rule, secondary by extension from the pharynx.

The general symptoms are generally, but not invariably, severe; high fever, prostration, and extensive swelling of the cervical glands. There is an offensive, often bloody, discharge from the nostrils, or an epistaxis, and the orifices of the nose and upper lip are excoriated. The membrane may usually be found by inspection. In certain cases, as yet somewhat inexplicable, there may be thick membranes in the nose containing diphtheria bacilli, but constitutional symptoms are absent and the disease is quite harmless (fibrinous rhinitis).

(4) *Miscellaneous Sites of Diphtheria.*—If a wound or an ulcerated or excoriated surface becomes infected, a pseudo-membrane may form at the point of lodgment of the germs. Diphtheria of the conjunctiva may occur primarily, or by extension from the nose; of the external ear, from the discharge of a diphtherial otitis media; of the mouth and lips, by extension; of wounds, ulcers, and the genitals, by direct infection from contaminated hands, instruments, or dressings.

Variations in Intensity.—The case may be *mild*, with slight fever and little if any prostration. In *severe* cases attended by extensive and intense local changes, the prostration is extreme, the temperature is subnormal, the face is ashy, the pulse rapid and feeble. In *malignant* diphtheria the general symptoms are grave from the very onset of the disease. The fever is high, the prostration extreme, the pulse rapid and weak, and death occurs in 2 or 3 days from the excessive toxæmia. In such cases the diphtheritic lesions in the nose and pharynx are usually of an aggravated type, and there may be subcutaneous ecchymoses.

Complications and Sequelæ.—Broncho-pneumonia and pulmonary collapse are notably frequent; rarely pulmonary gangrene. Acute nephritis is common; so also is otitis media. Epistaxis may be serious. Endocarditis and heart failure (usually due to a neuritis of the cardiac nerves) are not very rare.

Among the *sequelæ*, aside from chronic catarrh of the nasopharynx and anæmia, by far the most important and characteristic is diphtheritic paralysis. It occurs in 10 to 15 per cent of the cases, and comes on during convalescence (2d, 3d, or 4th week). It is as characteristic of diphtheria as nephritis is of scarlet fever. Like the latter, it may follow the mildest case of the disease and perhaps be the first symptom indicating the nature of the primary sore throat.

Diphtheritic paralysis is a toxic neuritis, general or local, and according to the particular nerves involved the symptoms may show a great variety. The most common form is that affecting the palate and pharynx, and is indicated by the nasal character of the voice, regurgitation of fluids through the nose, and dysphagia, with which there often coexists a moderate weakness of the legs. Next in frequency the ocular muscles are involved, causing ptosis, strabismus, and loss of accommodative power. The special nerves of taste or hearing may be involved. There may be a multiple neuritis affecting both legs (paraplegia), with absent tendon reflexes, although the last sign does not of necessity imply a neuritis; or the extensors of the feet; or one or both arms; or all four extremities; or the face; or the respiratory muscles (diaphragm in particular); or the cardiac nerves. The abnormally rapid or slow pulse, dilated heart, and the occasional advent of dangerous or fatal syncope, met with either at the acme of the disease or during convalescence, result in all probability from an involvement of the cardiac nerves, less commonly from an infectious myocarditis.

Differential Diagnosis.—The only absolute proof of the existence of diphtheria is the presence of the Klebs-Loeffler bacillus. In the severer forms of anginal diphtheria, where the exudate is not confined to the tonsils but spreads to the pillars of the fauces, soft palate, or pharynx, the clinical diagnosis is readily made; but when the picture and the course of the disease is exactly that of a follicular tonsilitis, or the membrane is absent as in the milder forms of diphtheria, a reliable diagnosis can be made only by a bacteriological or microscopical examination of the exudate. The membranous anginas occurring in scarlet fever, measles, pertussis, and scarlet fever, and usually due to the streptococcus, can not be differentiated from true diphtheria except by proving the absence of the Klebs-Loeffler bacillus. These forms of membranous sore throat are very appropriately qualified as diphtheroid.

For making cultures in suspected cases of diphtheria the Department of Health of New York city furnishes an outfit (free) consisting of a box in which there are 2 test tubes, one containing a sterilized cotton swab, the other a slant of coagulated blood serum. Both are plugged with sterilized cotton. The directions which accompany the outfit are as follows:

"The patient should be placed in the best light attainable, and, if a child, properly held. In cases where it is possible to get a good view of the throat, depress the tongue and rub the cotton swab gently, but freely, against any visible pseudo-membrane or exudate.

"In other cases, including those in which the exudate is confined to the larynx, open the mouth and pass the swab back till it reaches the pharynx, and then rub it freely against the mucous membrane. Without laying the swab down, withdraw the cotton plug from the culture tube, insert the swab, and rub that portion which has touched the exudate gently back and forth along the surface of the blood serum. Do not break the surface of the blood serum, for it is a surface growth of bacteria that is desired. Then replace the swab in its own tube, plug both tubes, fill out inclosed blank, and return the whole outfit at once to the station from which it was obtained. Do not use tubes which are contaminated, or in which the contents have liquefied or dried up. Do not make culture within two hours after any antiseptic wash or spray has been used in the patient's throat."

Prognosis.—The mortality varies from 10 to 50 per cent, and the prognosis should be guarded. In moderately severe cases of pharyngeal diphtheria recovery may be expected; if the nose is involved the gravity increases; and laryngeal diphtheria (membranous croup) is a very fatal disease. The antitoxine treatment has produced a notable diminution in the mortality.

XVIII. ERYSIPELAS

Symptoms.—This disease—due to infection by the *Streptococcus pyogenes*—has a period of incubation varying from 3 to 7 days. The form usually seen by the internalist is that which affects the face and head. The attack begins more or less suddenly with a chill or chilliness, followed by a rapid rise of temperature (103° to 105°). In previously strong and healthy persons the constitutional depression may be slight; but in the old or debilitated, or in chronic alcoholics, there may be great prostration, dry tongue, feeble pulse, and delirium.

Coincident with, or a little later than, the onset of the general symptoms a reddened spot is to be seen, usually on the bridge of the nose or upon one of the alæ, less frequently upon other parts of the face, or the ear, or head. This rapidly extends, the skin becomes swollen, red, and tense, and is often studded with vesicles or small bullæ, especially on the eyelids, ears, and forehead. The pain is burning and tensive. The inflammatory area has a well-defined, somewhat raised margin, and, as it extends, the areas first attacked become somewhat lighter in colour and less swollen. When the greater portion of the face is involved the eyes are nearly or quite closed, the nose is bulbous, the lips and ears are thickened and edematous. The cervical glands are enlarged. Small cutaneous abscesses are common on the neck, cheeks, and forehead, and there may be extensive suppuration under the scalp. The mucous membrane of the mouth and throat is reddened, and in rare cases the inflammation may extend to the larynx and cause œdema. Leucocytosis is present, and the urine is often albuminous.

The fever continues high with slight remissions for 6 or 7 days, and usually terminates by crisis. The local redness and swelling subside, and desquamation follows. In the wandering form of the disease (*E. migrans* or *ambulans*) the inflammation disappears from one part and appears in another, the temperature runs an irregular course, and the patient may exhibit the symptoms of the typhoid status.

Complications and Sequelæ.—Actual meningitis, as proved by autopsy, rarely occurs, the meningeal symptoms (delirium, coma) arising from the fever or the toxæmia. Pericarditis, pleurisy, acute nephritis (occasional), pneumonia (occasional), otitis media. Septicæmia, pyæmia, and ulcerative endocarditis are not uncommon. Articular rheumatism is a relatively frequent complication (ANDERS).

Differential Diagnosis.—The mode of onset, the fever, and the swollen, well-defined edge of the erysipelatous inflammation render the diagnosis easy in a developed case.

(1) *Acute Eczema.*—The absence of fever, the lack of a definite border or of marked swelling, and the presence of intense pruritus will declare the disease to be an acute eczema.

(2) *Erythema.*—The absence of fever, swelling, or local heat separates erythema from erysipelas.

Prognosis.—The mortality varies from 4 to 7 per cent. In healthy adults recovery is the rule. In persons over 60, in chronic topers, or in the debilitated, the prognosis is serious; and erysipelas of the navel in the newborn is usually fatal. Death occurs from exhaustion or toxæmia.

XIX. TOXÆMIA, SEPTICÆMIA, AND PYÆMIA

Definitions.—(*a*) *Toxæmia* results from the introduction into the system of toxines formed by micro-organisms, mainly by those which are pathogenic. *Sapræmia* is a toxæmia due to the absorption of toxines formed by the bacteria of putrefaction. In either case the organisms develop at some local site, whence the poisonous substances are absorbed—e. g., the throat in diphtheria, or a moist gangrene of the leg.

(*b*) *Septicæmia* (bacteræmia) is a condition in which there is an incursion of pathogenic germs into the blood and tissues of the body in general, with or without a discoverable avenue of entrance, but in which there are no foci of suppuration.

(*c*) *Pyæmia* is a septicæmia due to pyogenic organisms, *plus* one or more foci of suppuration.

Causes and Symptoms.—(*a*) **Toxæmia.**—(1) *Causes.*—This condition attends the invasion of many of the infectious diseases, especially erysipelas, diphtheria, tetanus, and pneumonia, in which the pathogenic germ lodges and multiplies in some special locality. The constitutional symptoms are due to the absorption of the toxines manufactured at the site of infection and not to the entrance of the germ into the blood. At a later period the pathogenic organisms may invade the blood and tissues, and the condition then becomes a septicæmia. Of the form of toxæmia known as sapræmia (absorption of the products of putrefaction) the most common examples are moist gangrene of the extremities, gangrene of the lungs or other portions of the body; auto-intoxication from the intestinal tract; the ingestion of tyrotoxicon or other ptomaines resulting from the decomposition of meat, milk, or cheese; the inhalation of the gases of putrefaction (causing nausea, fever, and diarrhœa); or absorption from a retained and offensive placenta.

(2) *Symptoms of Toxæmia.*—General malaise, prostration, restlessness, headache, chill, and fever. Of these the last is the most constant. The symptoms referable to the heart (rapid, weak pulse) and the nervous system constitute the best criteria of the severity of the toxæmia. There is usually a leucocytosis.

(*b*) **Septicæmia.**—(1) *Causes.*—The septicæmic condition may originate from a recognisable focus of infection, as in autopsy wounds, puerperal septicæmia, anthrax, pneumonia, gonorrhœa, and typhoid fever, a general infection resulting from what was originally a local process. In the majority of cases the septicæmia is due to the streptococcus or staphylococcus; but there are often combined or mixed infections—e. g., the septicæmia may be caused by the simultaneous presence of the *Streptococcus pyogenes* and the *Bacillus typhosus*, the *Diplococcus pneumoniæ*, the *Bacillus tuberculosis*, or the Klebs-Loeffler bacillus.

In other cases the local focus of infection is not recognisable during life and perhaps not at autopsy (cryptogenetic septicæmia). Generally in these cases the patient is already ill of some acute or chronic malady; but in a certain proportion the infection occurs in those who are in good health. Of the germs causing the infection the *Streptococcus pyogenes* is the most common, next the *Staphylococcus pyogenes*, the pneumococcus, the *Bacillus proteus*, and the *Bacillus pyocyaneus*.

Here also may be considered certain local or general infections occurring very frequently as secondary or intercurrent events in chronic diseases, especially in chronic nephritis, arteriosclerosis, chronic valvular disease, cirrhosis of the liver, Hodgkin's disease, leucæmia, chronic tuberculosis, and other chronic maladies. Because of the fact that these infections are apt to close the scene they are spoken of as "terminal." The micro-organisms found in the terminal infections are, in addition to those previously mentioned, the gonococcus, the gas bacillus, and the *Bacillus tuberculosis*. The local affections to which they give rise are acute meningitis, pericarditis, endocarditis, or pleurisy; acute miliary tuberculosis of the peritonæum or pleura; and entero-colitis.

(2) *Symptoms of Septicæmia.*—When septicæmia starts from a local infective focus, whence the micro-organisms enter the blood, the symptoms of the invasion may begin on the 2d, 3d, or 4th day, rarely later. At the onset there is usually chilliness rather than rigour, with fever, moderate at first, but which rises and tends to become of the continued type, with decided daily remissions. There is headache, anorexia, prostration, delirium, or marked apathy. The pulse is rapid, small, and compressible. The tongue shows a marginal redness, and may become dry and brown; there may be nausea, vomiting, and diarrhœa; and the spleen may be palpably swollen. Petechial spots are not uncommon; slight toxæmic jaundice usually develops; and scarlatiniform rashes or herpes may appear. The urine frequently contains albumin, leucocytes, red cells, and tube casts. If the septicæmia is cryptogenetic, and especially if it is due to the presence of the streptococcus, the fever is high, irregular, with recurrent slight chills, and of a more decided septic cast.

(*c*) **Pyæmia.**—(1) *Causes.*—Pyæmia is a septicæmia complicated by multiple abscesses arising from an original focus of suppuration. The abscesses, primary or secondary, may be seated in any part or organ of the body. The organisms most commonly causing suppuration are the streptococcus and staphylococcus, although other germs may be pyogenic, viz., the pneumococcus, the *Bacillus typhosus*, the *Bacillus coli communis*, Pfeiffer's bacillus (of influenza), the *Bacillus proteus*, the *Bacillus pyocyaneus*, and probably the gas bacillus.

The vessels in the neighbourhood of the original suppurative focus become inflamed, with the formation of thrombi; in the majority of cases the veins are involved (phlebitis), less commonly the arteries (arteritis). From these infected thrombi particles of varying size become detached, and are carried as emboli by the blood stream to various parts or organs, where they become impacted. Instead of causing simple infarctions, as do non-infected emboli, they give rise to new foci of suppuration because of the masses of pyogenic organisms conveyed by them. The veins are the usual channels by which the septic emboli are transmitted to various parts of the body; particles from an arterial thrombus are arrested in the capillary areas in which the branches of the thrombosed artery terminate.

The sites of the secondary (metastatic, embolic) abscesses vary according to the vascular area in which the original suppurative focus is situated; thus, if it lies in the intestines, multiple metastatic abscesses are found in the liver, the septic particles being conveyed by the portal system of veins; if in the skin, muscles, or bones, the embolic abscesses are found in the lungs; or if the emboli are very small they may pass through the pulmonary circulation and lodge in the kidney, spleen, and joints; or may be detained in the heart and produce an ulcerative endocarditis, furnishing septic particles which, travelling in the arterial stream, become the cause of abscesses in various parts of the body.

(2) *Symptoms of Pyæmia.*—A severe chill or rigour usually marks the onset of the disease, the temperature rapidly rises to 103° or 104° or over, and is followed by a remission, or perhaps an intermission, with profuse sweating. The chills, high fever, and sweating recur at irregular intervals (Chart XX), the temperature curve in the meantime showing a slightly elevated and remitting line. There are anorexia, nausea, and vomiting. In the acute cases there is rapid emaciation, and as the dieases advances prostration becomes marked, the

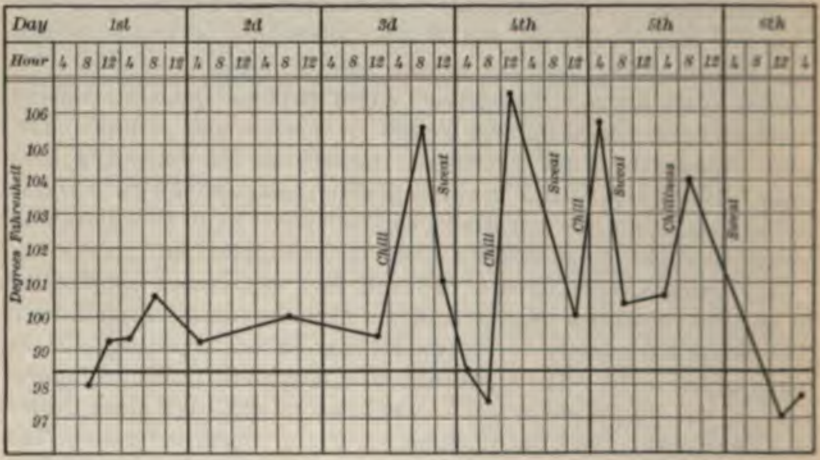


CHART XX.—Irregular chills and fever in pyæmia due to gangrene of lung following an inhalation pneumonia.

skin is moderately jaundiced, the pulse is rapid and feeble, and the dry, brown tongue, delirium, coma, and other symptoms of the typhoid status develop. In chronic cases the fever is irregular, and the chills recur at long intervals. There is a leucocytosis.

In the majority of cases local symptoms, due to multiple metastatic inflammations, become manifest sooner or later, as follows: In the *lungs*, dyspnœa, cough, perhaps purulent or rusty expectoration, and the physical signs of consolidation, cavity, or pleural effusion; in the *spleen*, enlargement of the organ, with pain, tenderness (perisplenitis), and perhaps splenic friction sounds on auscultation; in the *liver*, symptoms are slight, liver may be enlarged and tender, perhaps with friction sounds (perihepatitis); in the *heart*, endocarditis, perhaps ulcerative (*q. v.*); in the *kidneys*, the urinary evidences of multiple renal abscesses or infarcts, see (8) (*c*), page 708; in the *joints*, pain, redness, fluctuation.

Diagnosis of Pyæmia.—The cardinal symptoms are the irregular chills, fever, and sweats, together with the discovery of a primary suppurative focus. Of the latter the most common are: Suppurating wounds, surgical or accidental; childbirth; prostatic abscess, chronic ulcerative cystitis, gonorrhœa, chancroids, and bubo; appendicitis, suppurating hemorrhoids, or intestinal ulceration, perhaps with a pylephlebitis; suppurative mastoiditis resulting from an acute or chronic otitis media; osteomyelitis, or septic arthritis. Ulcerative endocarditis, while usually secondary, may be the primary cause of pyæmia. In doubtful cases of pyæmia or septicæmia, especially of the cryptogenetic form, a most careful search is to be made for a wound, abrasion, or local lesion in any part of the body. An examination of stained blood films may possibly result in the discovery of pyogenic organisms.

Differential Diagnosis of Pyæmia.—As compared with septicæmia, pyæmia exhibits recurring chills, deeply remitting fever, and sweats, with great prostration, rapid wasting, moderate icterus, and evidences of embolic abscesses; while in septicæmia there is usually but a single chill, the fever is of the continued type, sweats are uncommon, the jaundice is much lighter, and there are no metastatic abscesses. Moreover, in septicæmia, delirium and coma are early symptoms; in pyæmia they become manifest later in the disease.

(*a*) **Diseases Simulated by Pyæmia.**—(1) *Malaria.*—Because of the chills, fever, and sweats, pyæmia may be mistaken for an intermittent malarial fever, but the finding of the plasmodium and the prompt effect of quinine in the latter offer ready means of differentiation.

(2) *Typhoid Fever.*—The more chronic cases of pyæmia in which there is an irregular fever with delirium, prostration, diarrhœa, and a swollen spleen, may simulate typhoid fever, but the presence of rose spots, the absence of leucocytosis, and the finding of a positive Widal test will declare for the latter.

(*b*) **Diseases which may simulate Pyæmia.**—Among these are pyelitis (especially if due to renal calculus); renal tuberculosis; empyema; beginning pulmonary tuberculosis; abscess of the liver; lodgment of a gallstone in the common duct; Hodgkin's disease; grave anæmia; and swiftly advancing malignant disease.

Prognosis of Pyæmia and Septicæmia.—The prognosis varies with the cause. If the latter is removable before serious inroads have been made upon the strength, or if irremediable complications have not occurred, recovery takes place. Otherwise, especially in pyæmia, the prognosis is always grave.

XX. YELLOW FEVER

Symptoms.—The specific organism of this disease has not yet been identified. It is transmitted through the bite of the mosquito, *Stegomyia fasciata.* Incubation period, 1 to 7 days.

(1) *First Stage.*—Onset sudden, with chilliness and a rapid rise of temperature (100° to 106°), accompanied by headache and intense pain in the back and limbs. Sore throat, a furred tongue, and epigastric tenderness are present; nausea and vomiting are usually persistent. Constipation generally exists. The pulse is remarkable for its slowness in comparison with the temperature (75 : 103°), and may become less frequent as the temperature rises. Albuminuria, which may be transient, occurs early in the disease—i. e., on the 2d or 3d day. The facies is said to be quite characteristic. The face is flushed, the eyes are redly injected and intolerant of light, and the eyelids and lips slightly swollen. Careful inspection shows, even on the first day, a subicteric tint. The fever keeps high, with slight variations, for from 1 to 3 days, and then falls, with an amelioration of the symptoms, and the patient enters upon the

(2) *Stage of Remission.*—This lasts from a few hours to a day, and in favourable cases marks the beginning of convalescence. More commonly the fever rises again and the patient passes into the

(3) *Second Stage.*—The skin becomes yellow or almost bronzed. The vomiting persists, may be projectile and painful, and in severe cases the vomitus contains dark blood (black vomit); or the blood may be bright and unaltered. The stools may be tarry, and there may be bleeding from the nose and gums, as well as petechial spots on the skin. The urine is scanty, containing blood, albumin, and

casts; or there may be complete suppression, giving rise to uræmic coma or convulsions. The tongue is dry and brown, or raw and fissured. Unless uræmic dulness is manifested the mind usually remains unclouded. Great prostration, or even collapse, with cold skin and extremities, is present. In favourable cases the secondary fever, after lasting from 1 to 3 days, falls by lysis; in bad cases the temperature goes rapidly upward to a higher point than in the first stage and death soon follows.

The entire duration of the disease is about one week. Convalescence may be hindered by a relapse (not common), diarrhœa, parotitis, or abscesses.

Differential Diagnosis.—The cardinal symptoms of yellow fever are the facies, the reduction of the pulse rate although the temperature remains high or rising, and the *early* albuminuria.

Very mild cases (slight fever for 24 to 48 hours) can not be recognised unless an epidemic is prevailing; and the early cases of an outbreak are apt to present difficulties in diagnosis. The intensity of the disease varies from *mild*, to *severe* (prostration, vomiting, hemorrhages), to *malignant*, in which a fatal result occurs in 2 or 3 days from the virulence of the poison. The disease is to be discriminated from

(1) *Dengue.*—See page 749.

(2) *Malarial Fever.*—In the æstivo-autumnal variety of this disease, which is the only form likely to be confused with yellow fever, jaundice rarely appears until the 4th or 5th day; albumin is very seldom found as early as the 2d day; black vomit is very rare; hæmaturia is much more common; the spleen is usually enlarged; and the characteristic crescentic or, more commonly, the small ring-shaped forms of the plasmodium may be found in the blood. In the absence of the plasmodium good authorities may differ.

Prognosis.—The mortality varies from 15 to 85 per cent, showing a wide range in the virulence of different epidemics. An initial fever exceeding 104° is of bad omen; so also is black vomit; while anuria, delirium, coma, and convulsions usually mean death. On the other hand, moderate fever, slight jaundice, an ample flow of urine, and freedom from hemorrhages offer a favourable prognosis. When death occurs it is generally from exhaustion or uræmia.

XXI. DYSENTERY

Five varieties of this disease are recognised: catarrhal, acute specific, amœbic, diphtheritic, and chronic dysentery. All of these have as a symptom frequent, usually mucous and bloody, stools; and in the acute forms abdominal griping and severe tenesmus.

Varieties and Symptoms.—(1) **Catarrhal Dysentery.**—Usually after 1 or 2 days of a painless moderate diarrhœa, griping and colicky abdominal pains are manifest. The stools become frequent, and are expelled with severe straining and tenesmus. The desire to defecate is unremitting, and there is a constant and distressing feeling of rectal fulness, pressure, and bearing down. The stools are at first partly fæcal, but soon come to consist purely of small amounts ($\frac{1}{2}$ oz.) of gelatinous mucus, muco-pus, and blood, varying in number from 15 to 200 in 24 hours. In mild cases there is slight fever; in the severer forms the temperature may reach 102° or 103°. The tongue is at first furred, later becoming red and smooth; thirst is excessive; nausea and vomiting are not usually present; and the abdomen is often flat and resistant. In severe cases there is marked prostration, with a frequent pulse, and rapid emaciation.

If the case is of moderate severity, in 7 or 8 days the stools become less frequent and less bloody, the mucus diminishes, brownish shreds of necrosed mucous membrane may be found, fæcal matter makes its appearance, and the stools gradually assume a normal character. While the milder forms of the disease terminate in 1 week, the severer types endure for at least 4 weeks before convalescence is established.

(2) **Acute Specific Dysentery.**—This prevails in Japan and the Philippines as a disease of adults and in temperate climates as an infantile disease. It is due to the bacillus dysenteriæ of Shiga, which is now recognised as the exciting cause of most of the forms of infantile diarrhœa. Two varieties of the organism are known—the Shiga (not fermenting mannite), and the Harris (fermenting mannite). The latter is the form most commonly met with in the United States.

This bacillus has rounded ends and possesses slight motility. It decolorizes by Gram's method. These organisms rarely can be discovered in the stools until the fifth to the seventh day of the disease; and then only by the skilled laboratory worker. Like the bacillus of typhoid fever in the Widal test, the bacillus dysenteriæ will, except in very mild cases, agglutinate with the blood-serum of the patient. To be considered reliable the reaction should occur with a dilution of not less than 1:200. It is positive in about 80 per cent of all cases.

Symptoms.—Sporadic cases are frequent and epidemics may be looked for here in the summer months, and toward the end of the rainy season in the tropics. In the severe cases there is a sudden onset with fever, abdominal pain, and diarrhœa. The stools are at first watery, then mucoid, and finally bloody. Tenesmus is constant and severe. With the increase in intensity of the local symptoms there is a corresponding constitutional disturbance with rapid, feeble pulse, high temperature (103° to 104° F.), dry, furred tongue, and thirst. Delirium may supervene, followed by death in 48 hours. Milder cases tend to improve after 2 or 3 days with amelioration of all symptoms and recovery after 15 to 20 days. The disease may, however, pursue a subacute or chronic course of weeks or even months.

Unfavourable symptoms are great emaciation, nervous adynamia, low delirium, hiccough, and the finding of gangrenous sloughs in the stools.

In *infants* we have to deal with the well-known symptoms of acute enterocolitis (*q. v.*). We owe this important advance in our knowledge of the etiology of the summer diarrhœas of infants (especially cholera infantum and dysentery) to the excellent work of Bassett, Duval, Park, Dunham, and others.

The *diagnosis* rests on the presence of a severe enterocolitis with the demonstration of Shiga's bacillus in the stools, which requires the skill of the expert; perhaps also a positive result from the agglutination test, if such is available. This will serve to differentiate it from Asiatic cholera and amœbic dysentery.

(3) **Amœbic (Tropical) Dysentery.**—The invasion may be sudden, usually gradual, beginning with a moderate diarrhœa. There is slight fever (which may be entirely absent), nausea and vomiting are uncommon, and abdominal griping and tenesmus present only at the onset. The stools are at first mucous and bloody, but later become fluid and yellowish gray, containing mucus and at times blood and sloughs, and vary from 6 to 12 in 24 hours. The diarrhœa is somewhat characteristic in that it, although persistent, alternately remits and relapses at irregular intervals. There is a steady loss of strength and weight. If complications do not occur, the disease lasts from 6 to 12 weeks; convalescence is slow because of weakness and anæmia, relapses are frequent, and the disease is prone to become chronic.

(4) **Diphtheritic Dysentery.**—This form of the disease, in which there may be necrosis and ulceration of the mucosa of the colon, with the formation of a more or less marked croupous exudate or pseudomembrane, is not uncommon. It may be primary or secondary.

In the rare *primary* cases the disease is usually acute and of sudden onset, with extreme prostration, delirium, abdominal pain, tenderness and distention, frequent stools, and high fever. This condition may be mistaken for typhoid fever.

The *secondary* form is more frequent, and is found as an intercurrent and often terminal event in chronic nephritis, chronic cardiac disease, pulmonary tuberculosis, and in various cachexiæ, as well as in certain acute ailments, especially typhoid fever and pneumonia.

The intestinal symptoms are slight, simply a moderate diarrhœa. The stools vary from 2 to 4 daily, are often copious and weakening, and at first may contain a little mucus and blood. There is little, if any, tormina or tenesmus.

(5) **Chronic Dysentery.**—Usually a sequel of an acute attack, except in the amœbic variety, which, from its beginning, may be subacute and tend to chronicity. The symptoms are not especially characteristic. The stools, varying from 4 to 12 or more in 24 hours, may be fluid and frothy, or semifluid, yellowish or brown, occasionally containing mucus and undigested food, rarely blood, pus, or necrotic shreds; constipation may alternate with diarrhœa; and acute exacerbations are not uncommon. Except during the exacerbations tormina and tenesmus are rarely present. There is usually tenderness along the course of the colon, often flatulence; the tongue is red and glazed, or dry and fissured; and the loss of flesh and the anæmia are extreme.

Complications and Sequelæ.—Hepatic abscess (*q. v.*), perhaps with a secondary abscess of the lung, is the most frequent and gravest complication, occurring as a rule only in the amœbic variety and in the tropics (20 per cent). In cases of amœbic dysentery in this country its frequency does not exceed 3 per cent. Local peritonitis by extension may occur, or an ulcer may perforate, causing, according to its location, perityphlitis, or periproctitis. There may be during long-continued and severe cases, or as sequelæ, pleurisy, pericarditis, endocarditis, painful and swollen joints, pylephlebitis, œdema (due to anæmia), chronic nephritis, and paraplegia (due to neuritis). The debility of chronic dysentery predisposes to tuberculosis and pneumonia, and occasionally to corneal ulceration. Any form of dysentery may leave the patient with impaired digestion and a liability to diarrhœa. Dysentery and malaria may coexist.

Differential Diagnosis.—In *catarrhal dysentery* the cardinal symptoms are the frequent stools, composed of blood and mucus, and the tenesmus. In the *specific form* the blood serum agglutinates with the Shiga bacillus. In *amœbic dysentery* the course of the disease is characteristically slow, irregular, and chronic, presenting remissions and exacerbations; the pathognomonic test is the finding of the amœbæ in the stools, or in the sputum if an hepatic abscess has perforated into the lung. The existence of the *secondary diphtheritic* variety may be suspected, but the diagnosis is generally made at autopsy. The *primary acute diphtheritic* variety is not seldom mistaken for typhoid fever (see (4) following). *Chronic dysentery* is difficult to distinguish from chronic diarrhœa, but the dysenteric character of the symptoms (tormina, tenesmus, bloody and mucoid stools) in the initial attack, and during the exacerbations as well, may establish the diagnosis.

Dysentery is to be separated from:

(1) **Diarrhœa.**—The absence of tenesmus and stools composed purely of mucus and blood are sufficient for the discrimination.

(2) **Local Affections of the Rectum.**—Cancer or syphilitic disease of the rectum, or inflamed and strangulated hemorrhoids, may cause tenesmus and bloody, mucoid discharges, but the history and a physical examination of the rectum will, as a rule, readily determine the cause of the symptoms.

(3) **Intussusception.**—In this condition, although defecation may be frequent and tenesmic, there is usually persistent and increasing vomiting, the stools are bloody rather than mucoid, laxatives are not effectual, fever is not an early symptom, and examination of the abdomen may reveal a tumour.

(4) **Typhoid Fever.**—In this disease the fever does not rise so rapidly and to such a height as in primary diphtheritic dysentery; the intestinal symptoms are less, and the stools are rarely bloody. Moreover, in dysentery the spleen is not enlarged, the rose rash is absent, and a positive Widal reaction is not obtained.

Prognosis.—In the *catarrhal form* recovery is the rule. In the *specific form* the mortality in some epidemics is very high. Isolated cases usually recover. In the *amœbic form* the mortality varies; in epidemics during campaigns in the tropics it rises to 70 or 80 per cent, while occasional cases in civil life and temperate zones give a death rate of 5 or 6 per cent only. In the *diphtheritic forms* the prognosis for life is unfavourable. In dysentery death usually results from exhaustion. The symptoms which justify a bad prognosis are a dry tongue, feeble and rapid pulse, delirium, stupor, and evidences of collapse.

XXII. CHOLERA ASIATICA

Symptoms.—This disease—due to the *comma bacillus*—has an incubation period of from 2 to 5 days.

(1) *Stage of Invasion.*—Commonly there is, during this period, slight diarrhœa, and colicky abdominal pain, with headache, mental depression, perhaps nausea and vomiting. These symptoms may progress no further (cholerine), but usually the two other stages of the disease supervene, viz., collapse, and reaction.

(2) *Stage of Collapse.*—This may come on without prodromata, but ordinarily an existing looseness of the bowels is suddenly succeeded by frequent copious stools, which soon lose their fæcal character and become liquid, serous, or "rice-water" discharges, usually

passing without pain, sometimes with tormina and tenesmus. Persistent and severe vomiting soon appears. The vomitus is copious, and becomes serous, like the stools. Thirst is excessive, the tongue is furred and dry, and recurring and severely painful cramps in the legs and feet constitute an early symptom. The patient becomes rapidly exhausted or collapsed. The skin is cold, shrivelled, and wet; the lips and finger tips are profoundly cyanotic, the face is gray, pallid, and pinched, the eyeballs recede, and the cheeks become sunken. While the internal temperature (by rectum) may be elevated (102° to 104°), the axillary or mouth temperature may drop to 95° or below. The voice is husky or whispering, and there may be mental dulness merging into stupor and coma, although consciousness is often preserved to the close. The pulse becomes feeble, running or, in bad cases, imperceptible at the wrist. The urine is scanty, albuminous, and contains tube casts; complete suppression may occur. This stage lasts from a few hours to 2 days. If death does not occur during the collapse, the disease passes into the

(3) *Stage of Reaction.*—The skin becomes warm, the flow of urine is re-established, vomiting ceases, the stools become less frequent and more fæcal in character, the pulse strengthens, the fever departs, and in favourable cases convalescence begins.

Varieties and Terminations.—The attack may be mild (*cholerine*), with nausea, griping, copious stools, and cramps, but with very slight collapse symptoms, recovery occurring before the graver conditions develop. In the severest cases the patient is overwhelmed and dies in a few hours, before the onset of diarrhœa (*cholera sicca*), or perishes just before the stage of collapse. The stage of reaction and apparent convalescence may be interrupted by a relapse, during which death occurs; or the symptoms may merge into those of *cholera typhoid*—i. e., fever, dry, brown tongue, feeble and rapid pulse, delirium, coma, and death. There may be erythematous, macular, or purpuric rashes.

Complications and Sequelæ.—Acute nephritis, anuria, and uræmia; pneumonia and pleurisy; diphtheritic inflammations of the mucous membranes (colon, pharynx, genitals) are not uncommon; suppurative parotitis (not uncommon); ulceration of the cornea; abscesses in various parts, erysipelas, local gangrene (rare); and, during convalescence, muscular cramps in arms and legs.

Differential Diagnosis.—During an epidemic the diagnosis is readily made. Sporadic cases may be mistaken for cholera morbus (or nostras), which in the severest form may present exactly the same symptoms as Asiatic cholera. A positive differentiation is possible only by the microscope and cultures, whereby the presence or absence of the comma bacillus is ascertained. Attacks resembling Asiatic cholera may arise from poisoning by arsenic, bichloride of mercury, antimony, or other metals, but the history and, if suspected, the chemical tests of the stomach contents usually serve for differentiation.

Prognosis.—The mortality varies from 20 to 80 per cent, but the prognosis must always be guarded. The chronically debilitated, the alcoholic, and the extremes of age show a large mortality.

XXIII. BUBONIC PLAGUE

Symptoms.—This disease—caused by the *Bacillus pestis*—has an incubation period varying from 2 to 5 days.

The initial symptoms are headache, backache, muscular stiffness, vertigo, mental depression and uneasiness, rapid respiration, and perhaps epistaxis or hæmoptysis. In 24 hours, more or less, there is chilliness or a chill, and the temperature rises to 104° to 106°, with delirium, great thirst, dry, brown tongue, and oftentimes nausea and vomiting. Ecchymoses and petechial spots are very commonly present. About the 3d to the 5th day the inguinal glands become swollen (buboes), less commonly the axillary, cervical, and popliteal, and may either undergo resolution or proceed to suppuration (favourable) or gangrene (rare). Carbuncles may appear, especially on the legs, gluteal regions, or back. In the severest cases hæmatemesis, intestinal hemorrhages, and hæmaturia may occur.

Varieties.—There are 4 varieties: the *mild form*, with slight fever and constitutional symptoms (rarely fatal); the *ordinary bubonic form*, with glandular swellings and severe disturbance (may recover); the *malignant form*, usually without glandular enlargements, the poison localizing itself in the lungs, kidneys, brain, stomach, or intestines, or acting as a general toxæmic agent without localization (almost always fatal); and the *pneumonic form*, the sputum containing the bacilli in enormous numbers. This variety is even more fatal than the malignant form. The *mortality* is enormous, 70 to 95 per cent.

XXIV. MALARIAL FEVER

I. Varieties and Symptoms.—Four varieties of malarial poisoning are recognised: intermittent malarial fever, remittent malarial fever, pernicious malarial fever, and the malarial cachexia. For a description of the life history and the method of detection of the causative organism (*Plasmodium malariæ*), see pages 631 and 633.

Intermittent Malarial Fever.—(*a*) Symptoms.—A paroxysm of intermittent fever embraces 3 parts, chill, fever, and sweating.

(1) *The Chill.*—Premonitory symptoms are headache, languor, yawning, gastric unease, perhaps nausea and vomiting. The patient begins to feel cold, and soon shivers and shakes more or less violently, the teeth chatter, and the face and finger nails are cyanotic. Although the skin is cool and pallid, the thermometer in the mouth or rectum may rise to 105° or 106°. The pulse is rapid, small and hard. In from 10 minutes to 1 hour the cold stage is succeeded by:

(2) *The Fever, or Hot Stage.*—The coldness disappears, the skin becomes excessively hot and reddened, the face is flushed, the eyes injected, there is a throbbing headache and, possibly, active delirium. The pulse is full and bounding. The fever may reach its maximum during the chill, and in any case does not continue to rise very rapidly. In from 2 to 6 hours the fever falls by crisis.

(3) *Sweating Stage.*—As the temperature descends the patient begins to sweat, slightly or profusely, first about the head and neck, finally from head to feet; the headache disappears; and in 2 or 3 hours he feels comfortable and may fall asleep.

The duration of the entire paroxysm varies as a rule from 8 to 12 hours.

Types of Intermittency.—(See Charts II and III, page 112.)

(1) *Tertian.*—When 1 group of the tertian parasites is present the paroxysm is tertian—i. e., recurs every other day; with 2 groups it is quotidian, recurring every day—i. e., double tertian. In the Northern and Middle States the double tertian is the type most commonly encountered.

(2) *Quartan.*—When 1 group of the quartan parasite is present the paroxysm occurs every 4th day (quartan type); with 2 groups the paroxysms occur 2 days in succession, the 3d day none; with 3 groups, daily or quotidian paroxysms occur as in the double tertian.

Æstivo-autumnal Fevers.—**Symptoms.**—(1) *Of Remittent Malarial Fever.*—This type of fever is caused by the æstivo-autumnal parasite which may not only occur in ill-defined and multiple groups, but has as well an uncertain and probably varying period of development. Consequently the type of the fever is often very irregular. The paroxysms may be of an intermittent quotidian periodicity, but are longer (20 hours) than with the tertian or quartan infection, and exhibit a tendency to anticipate—i. e., the intervals between the paroxysms grow shorter. Chill is often absent, and the fever rises and falls more slowly. In the severer forms of æstivo-autumnal fever the paroxysms lengthen, each one treading closely upon the heels of that which precedes it, until the fever becomes almost continuous (102° to 103°), with slight remissions. Sharp intercurrent paroxysms of fever (105° to 106°), perhaps with chills, may occur.

In a case of average severity the 1st day of the disease begins with a chill or chilliness, followed by fever, which remits with sweating usually in the early morning of the 2d day, to be followed by another paroxysm in the afternoon. The face is flushed, the pulse full and bounding but seldom dicrotic, the tongue is furred, moderate jaundice is often present, there is a bronchitis, and slight delirium may become manifest. In the mildest cases the symptoms are light, and the fever ceases in about 7 days; in others it lasts from 10 days to 2 weeks; in the severest attacks the temperature is almost continuously high, and the disease may be protracted for 3 or 4 weeks (especially if quinine is not given), simulating typhoid fever; or the characters of the pernicious form may appear.

(2) *Symptoms of Pernicious Malarial Fever.*—This type of malarial fever is also, like the remittent form, due to the æstivo-autumnal parasite. It is rare in the temperate zone. The symptoms of the various forms are due to a more or less overwhelming toxæmia. Three varieties of the pernicious form are recognised: algid, comatose, and hæmorrhagic.

Algid Form.—Sudden onset of vomiting, often purging or choleriform diarrhœa, great prostration or symptoms of collapse, coldness, perhaps with no distinct chill, and a normal or even subnormal temperature, are the symptoms of the algid form. There is oliguria, sometimes anuria. After persisting for several days, with slight rises of temperature, the patient may die from profound exhaustion.

Comatose Form.—The disease begins abruptly, with high fever, and either active delirium or a rapidly developing coma. The patient may perish in the attack, or more commonly regain consciousness in 12 to 24 hours. Recurrent attacks are often fatal.

Hemorrhagic Form.—There may be cutaneous ecchymoses, epistaxis, bleeding from the gums, hæmatemesis, intestinal hemorrhage, metrorrhagia, and hæmaturia. Jaundice is very frequent. The hemorrhagic cases often begin with a severe chill, followed by high fever, and perhaps delirium. The urine may contain albumin and casts—granular, epithelial, and blood. Anuria and uræmia may occur. Hæmaturia is the most common of the hemorrhagic features of malaria, sometimes reaching epidemic dimensions, and not infrequently causing death. It is mainly a hæmoglobinuria (p. 697). The hæmoglobinuria may be preceded by a severe chill, fever, and sweat; or a mild malarial paroxysm; or occur without notable fever. It may be intermittent; or continuous, with remissions.

Malarial Cachexia.—In the majority of cases this condition is a legacy from repeated attacks of some variety of the acute malarial fevers; repeated either from reinfection or from lack of proper and energetic treatment. In excessively malarial regions the preceding febrile paroxysms may have been slight.

Symptoms.—The cardinal symptoms are anæmia and enlargement of the spleen, sometimes also of the liver. The patient is pale and thin, the face is of a dirty yellow tint, sallow and cachectic. There may be no fever, or the temperature vary irregularly from 99.5° to 103°. The red cells may sink to but 1,000,000 to the cubic millimetre. The spleen is greatly enlarged and indurated. Dyspnœa, palpitation, œdema of the feet and ankles, headaches, and neuralgias are common because of the anæmia. Chronic gastro-intestinal catarrh, vertigo, insomnia, tremor, paraplegia (excessively rare), slight bronchitis, and painful stiffness of the muscles and joints may be present. Hemorrhages (retinal, hæmaturia, hæmatemesis, etc.) may occur.

II. COMPLICATIONS OF MALARIAL FEVER.—Anders states that complications occur in about 10 per cent of malarial cases. They are, in order of frequency, enteritis, nephritis, rheumatism, typhoid fever (but 8 times in 1780 cases), lobar pneumonia, jaundice, and dysentery.

III. DIFFERENTIAL DIAGNOSIS OF MALARIAL FEVER.—The only indubitable proof of the presence of malaria lies in the discovery of its parasite in the blood. There is no doubt that the term "malaria" has been and is used to cover a multitude of diagnostic sins, an evasion which is perhaps pardonable in view of the technical skill and experience which is requisite to give value to a negative result of a blood examination for the plasmodium. It is a good practical rule to suspect malaria, but to be extremely chary in making a positive diagnosis unless the symptoms are absolutely typical, and perhaps not even then without the finding of the hæmatozoon. That an intermittent fever is not malarial may be affirmed with an almost absolute certainty if it does not promptly cease when quinine is given in sufficient quantity (20 to 30 grains daily).

The characters of the blood in malaria are worthy of special notice. Owing to the fact that the red corpuscles are the hosts of the plasmodia there is an extensive destruction of hæmoglobin and a diminution in the number of the red cells, with consequent anæmia—the extent of the latter depending upon the duration and continuity of the malarial poisoning. The rapid supervention of anæmia is, indeed, very characteristic of this disease. The leucocytes are as a rule diminished in number and some are pigmented. In certain cases the blood has the characters of pernicious anæmia (p. 649). The great frequency of herpes in malaria is notable.

Differential Diagnosis of Intermittent Fever.—It is of great importance to bear in mind that various conditions are accompanied by an intermittent fever. The simple remembrance of this fact will in many cases prevent mistakes in diagnosis. Intermittent fever requires differentiation mainly from the following conditions:

(1) *Tuberculosis.*—The chills and intermittent fever sometimes present in beginning pulmonary tuberculosis are not arrested by quinine, the plasmodium is absent, and the physical signs or the finding of the tubercle bacillus will serve for differentiation.

(2) *Pyæmia or Concealed Suppurations.*—In this disease the chills, fever, and sweats occur at irregular intervals; there is more prostration, a marked leucocytosis exists, the malarial organism is not found, and recurrences are not prevented by quinine. Moreover, there is often some local lesion which will indicate the pyæmic nature of the intermittent pyrexia.

(3) *Pyelitis.*—This may closely simulate intermittent fever, but the presence of pyuria, leucocytosis, and perhaps of lumbar pain and tenderness and swelling of the kidney, together with the results of the blood examination and the therapeutic test, will declare the pyelitic origin of the fever.

(4) *Ulcerative Endocarditis.*—In addition to the history, clinical symptoms, and physical signs, the blood examination shows the absence of the plasmodium and the presence of a leucocytosis, and quinine does not arrest the chill and fever.

(5) *Gallstones.*—The chill and intermittent pyrexia sometimes associated with cholelithiasis may usually be distinguished from malaria by the history, the characteristic hepatic colic, the tender and perhaps palpable gall bladder, the frequent jaundice, and the absence of the plasmodium.

Differential Diagnosis of Remittent Fever is to be made from typhoid fever (p. 741), and **of Pernicious Malarial Fever** from yellow fever (p. 779). The algid form of pernicious malaria may resemble Asiatic cholera, but the absence of an epidemic, the finding of the plasmodium, and finally, in suspected cases, the non-discovery of the comma bacillus in the stools, will exclude the latter.

IV. PROGNOSIS OF MALARIAL FEVER.—In intermittent fever, always favourable with proper treatment; in remittent fever, usually favourable with proper treatment, but death may occur in very severe cases from exhaustion, or hæmaturia, anuria, and uræmia; in pernicious malarial fever the mortality runs from 20 to 25 per cent.

XXV. RHEUMATIC FEVER

Symptoms.—(1) **Acute Rheumatic Fever.**—There are wide variations in the intensity of the symptoms. The attack may be preceded by malaise, indefinite joint pains, and sore throat, especially tonsillitis. In the majority of cases the onset is sudden, with slight chilliness, rapid rise in temperature (rarely over 103°), and coincident joint symptoms. The affected articulations, usually the wrists, elbows, ankles, or knees, become swollen, hot, reddened, tender, and excessively painful upon movement. Generally 2 or more joints are involved, the inflammation leaving one and simultaneously appearing in another. Rarely one joint alone is affected. No joint is exempt in severe cases, but ordinarily the vertebral, sterno-clavicular, and phalangeal articulations escape. The swelling affects mainly the periarticular tissues, and there is rarely evidence of much fluid in the joint. The muscles in severe cases may be tender and rigid, and the tendinous sheaths of the wrists and ankles may be involved and cause considerable swelling. Profuse sweats, at first acid, are characteristic; the urine is scanty, loaded with urates, and may contain albumin; the bowels are constipated, and the tongue is heavily furred. Sudamina and a red miliary eruption are of frequent occurrence. Anæmia develops very rapidly, and there is a marked leucocytosis. The fever is very irregular, rising and falling in correspondence with the severity and extent of the joint inflammations. Free sweating lowers the temperature.

The duration of the disease varies from a few days to 6 or 8 weeks. In the protracted cases short periods of improvement alternate with relapses.

(2) **Subacute Rheumatism.**—This resembles the acute form, except that all the symptoms are of a milder character; the fever does not exceed 101°, the joint inflammations are less intense, and not so many articulations are involved. The subacute cases are often protracted for weeks or months, and may ultimately merge into a chronic type of the disease.

Complications and Sequelæ.—The most common and important complication is an endocarditis (rarely of the ulcerative form), the frequency of which is variously estimated at from 25 to 40 per cent, or even more, of all cases. The liability increases in proportion to the number of attacks. The valve most frequently affected is the mitral, and the importance and seriousness of this complication depends upon the progressive sclerotic and deforming changes of the valve segments which it initiates. Pericarditis, fibrinous, serofibrinous, or purulent (in children), sometimes attended by delirium, may occur, so also may myocarditis. Other complications are pneumonia, pleurisy; hyperpyrexia (106° to 110°), with or without delirium, and attended by great prostration, rapid and feeble pulse, and final stupor; cerebral complications—*e. g.*, delirium with or without hyperpyrexia or pericarditis, convulsions (not common), coma (uræmic, hyperpyrexial, toxæmic), chorea, and very rarely meningitis. Small firm, painless, and rapidly developing subcutaneous nodules, attached to the tendons and fasciæ, especially of the fingers, wrists, back of elbow, patellæ, and malleoli, are sometimes found in the course of the disease or during convalescence. They may last for weeks or months. Urticaria, purpura, erythema nodosum, and extensive ecchymoses may be present.

Differential Diagnosis.—The cardinal symptoms of rheumatic fever are the sudden onset of a polyarthritis, flitting from joint to joint, with fever and sweats, and the rapid occurrence of anæmia. Ordinarily the diagnosis is readily made, but in some instances the following affections require to be differentiated. (See also page 101. In children the arthritic symptoms may be very slight, while endocarditis, which is common, may in consequence of the lightness of the other symptoms be overlooked; and paroxysmal pain, without tenderness, in the upper abdomen is frequent.

(1) *Pyæmia with Suppurative Arthritis.*—The irregular chills and fever, the greater prostration, the slight jaundice, the fact that the joint symptoms do not shift from one articulation to another, and that the joint as a rule proceeds to suppuration, together with the finding of a purulent focus, are in favour of pyæmia. The *acute arthritis* of nursing infants (usually pyæmic, or due to gonorrhœal ophthalmia or vaginitis) is generally limited to one joint (knee or hip), and rapidly passes to suppuration. Acute *osteomyelitis* is usually located in the epiphysis of the bone rather than in the joint, and the greater severity of both local and constitutional symptoms will aid in separating it from rheumatism.

(2) *Gonorrhœal Rheumatism* (*Arthritis*).—This is frequently monarticular (often involving the knee) from the beginning, the constitutional symptoms are generally not pronounced in comparison with the local signs, it is very intractable, and there is a history of an immediately preceding gonorrhœa.

(3) *Gout.*—In typical cases of this disease the involvement of the great toe and the absence of decided fever and sweats will differentiate it from rheumatism. But when, as is not uncommon, several of the joints are affected, it may be very difficult to make a positive diagnosis. Gout usually occurs later in life than rheumatic fever, and tophi may be found.

Prognosis.—Favourable with regard to life, but a certain number of cases become chronic, and endocarditis often lays the foundation for subsequent irremediable valvular disease.

XXVI. LOBAR PNEUMONIA

In a very large proportion of cases of lobar pneumonia the *Diplococcus pneumoniæ* (or *lanceolatus*) is present; in a small number, other organisms, especially the streptococcus, are found.

SYMPTOMS AND PHYSICAL SIGNS.—**Typical Cases.**—In a typical case the onset is abrupt, with a severe and often prolonged chill, followed by headache, general aching, and a rapid rise of temperature to 104° or 105°. Very soon a short, dry, restrained, and distressing cough sets in, with a sharp, stabbing pain near the nipple or in the axilla of the affected side. The respirations become rapid (30 to 60 in adults, 80 or over in infants), with an expiratory grunt, and the alæ of the nose dilate with each inspiration. The face is flushed, and there is often a circumscribed redness upon the cheek of the affected side. The expectoration becomes rusty or blood-stained, and is excessively thick and tenacious. The pulse is rapid (100 to 120), full, and bounding, and the normal pulse-respiration ratio of 4 to 1 becomes 3 to 1, 2 to 1, or even 1 to 1. Herpes labialis is more frequent in pneumonia than in any other disease (12 to 40 per cent of all cases). The tongue is furred, there may be nausea and vomiting, the bowels are usually constipated, and tympanites is not uncommon. There is in most cases a leucocytosis (12,000 to 50,000). The urine is scanty, high coloured, and often contains a trace of albumin. A great diminution or entire absence of the chlorides is a striking feature. The *fever* having risen to its maximum, usually within the first 24 hours, continues high, with remissions, until the 5th to the 10th day, when in typical

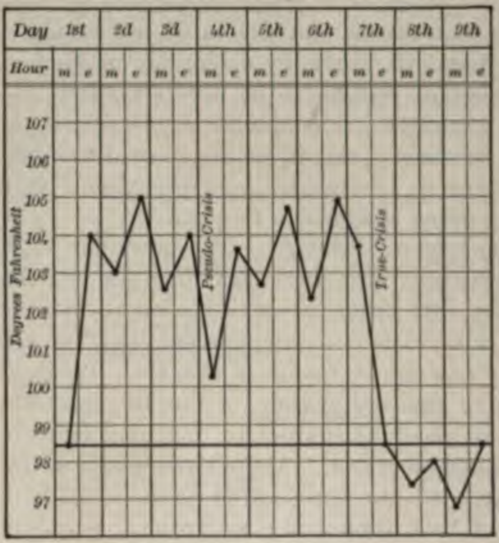


CHART XXI.—Pneumonia (lobar) with pseudo-crisis and true crisis.

cases it falls in a few hours (5 to 12) by crisis to normal or below (97° to 96°), with a corresponding drop in the pulse and respiration (Chart XXI).

The *physical signs* are modified by the stage of the disease (engorgement, red hepatization, gray hepatization).

(1) **Inspection.**—Increased (compensatory) motion of the unaffected side may be observed, and confirmed by palpation and mensuration, after the disease is well established; so also may the excessive action of the accessory muscles of respiration.

(2) **Palpation.**—After consolidation has occurred, unless the bronchial tubes are filled with thick secretion or fibrinous exudate (which may perhaps be removed by coughing) or pleural effusion is present, the vocal fremitus over the affected portion of the lung is increased, and a friction fremitus also may be felt.

(3) **Percussion.**—In the first stage the note may be somewhat high-pitched, perhaps hyper-resonant (Skodaic resonance), and a similar note may be elicited from healthy lung lying above an area of consolidation. Wintrich's change of sound (p. 438) may be found with pneumonia of the apex. Under similar circumstances the note may have a cracked-pot quality. If the consolidation is deep-seated the resonance may simply be impaired. Ordinarily there is marked dulness over the solidified portion of the lung.

(4) **Auscultation.**—In the early stage the breath sounds are often weak, and at the end of inspiration the crepitant râle may be heard. As engorgement merges into consolidation the breath sounds become broncho-vesicular, and finally intensely and typically bronchial. If the larger bronchi are plugged, bronchial breathing may be absent, sometimes temporarily. The voice sounds, both spoken and whispered, are transmitted with great distinctness, but this also may be prevented by filling of the bronchi.

During the 2d stage friction sounds are often heard, but no râles may be present. During the 3d or resolving stage small moist crepitations (*râle redux*), later mixed with moist râles of all sizes, become audible.

Special Symptoms and their Variations. — (1) The patient often lies on the affected side. Orthopnœa is not very frequent.

(2) The *fever* may be very slight or entirely absent in old persons and chronic alcoholics. I have seen cases of intense pneumonic infection with a temperature not exceeding 100°. In a few cases the crisis occurs on the 3d day. The termination may be by lysis, especially in children; and in delayed resolution the febrile movement may last for weeks. A pseudo-crisis 2 or 3 days previous to the final

fall is not uncommon. A rise just before and just after the true crisis (precritical and post-critical) is occasionally seen.

(3) *Cough* and *expectoration* are not seldom absent or very slight in infants, old people, topers, and those already ill with some acute infectious, or serious chronic, illness. If violent cough persists after the crisis, a pleural effusion is to be suspected. The sputum may be bright-red, rusty, bright yellow, or, in adynamic and typhoid cases, of a dark-brown colour (prune-juice). It is always very glutinous and clinging. Hæmoptysis is an uncommon early event.

(4) *Pain* is absent if the pleura is not involved, as in pneumonia affecting only the centre of the lung, and is absent or slight in apical consolidation. In children the pain is almost always referred to the abdomen. Abdominal and epigastric pain is not rare in adults, often exceptionally severe, and is generally due to involvement of the diaphragmatic pleura in pneumonia of the lower lobes.

(5) *Tympanites* is not uncommon, and may be so excessive, in conjunction with abdominal pain, as to suggest peritonitis or appendicitis. The *spleen* is generally swollen, occasionally also the *liver*.

(6) The *pulse* in severe cases may be dicrotic; or small and rapid; or full but soft ("gaseous"), and followed by serious cardiac weakness. Because of the obstruction to the pulmonary circulation the right heart may have an excessive amount of work thrown upon it, and the character of the pulse is no indication of the manner in which the right ventricle is standing the strain. A better criterion is the character of the *pulmonary second sound*. If this is accentuated the lesser circulation is being maintained; a disappearance or weakening of this sound is significant of right heart weakness and dilatation. An *absent leucocytosis*, if persistent, is a bad prognostic omen, except in very mild cases.

(7) Symptoms referable to the nervous system are common. In children convulsions frequently initiate the disease; and, also usually in children, the symptoms may closely resemble those of meningitis and the pneumonia be undiscovered. Delirium occurs in the adynamic cases and may be active or maniacal, especially in drunkards. Deafness, not dependent on otitis, is not infrequent.

CLINICAL VARIETIES OF PNEUMONIA.—The variations which are of more or less clinical importance depend in part upon the location and extent of the pulmonary lesions, but mainly upon differences in the virulence of the infective agent and the resisting power of the individual.

(1) *Pneumococcus Toxæmia (or Septicæmia)*.—Under this heading are embraced the cases which have been called adynamic, low, or typhoid pneumonia. The clinical picture is that of a more or less

profound and sudden blood-poisoning, with symptoms indicating the involvement of the nervous mechanisms which preside over the most important functions of the organism. The physical signs are often well marked, but may be slight. There is delirium or stupor, early severe prostration and cyanosis, and often slight jaundice. The tongue is dry and brown, the pulse and respiration are usually rapid, the expectoration prune-juice or very bloody, the fever may or may not be high, and subsultus and carphologia may be present. The spleen is often palpable, and nausea, vomiting, tympanites, and occasionally diarrhœa may be present. This form of the disease may be a mixed infection (pneumococcus *plus* streptococcus). It occurs especially in drunkards and persons debilitated from previous disease. Rare cases are those in which the toxæmia is so sudden and overwhelming that death may occur in from 24 to 48 hours.

(2) *Latent Pneumonia.*—The characteristic symptoms are hidden —indeed, may be altogether absent. The pulse is weak, not necessarily rapid, and the pulse-respiration ratio may deviate little, if at all, from the normal. Cough and expectoration may be absent. There may be delirium. In spite of the paucity of symptoms there are some points which will lead to an exploration of the chest: 1. Old age or chronic alcoholism. 2. A curious and at first inexplicable muscular weakness. 3. Patient inspection discloses a trifling amount of dyspnœa, manifested by an abnormal increase of respiration after moderate exertion such as turning in bed, or slight breathlessness in talking. 4. A trace of cyanosis in the lips and finger nails.

(3) *Abortive* (*or Larval*) *Form.*—Cases of pneumonia are encountered whose duration is so short that they deserve the term abortive. These are seen particularly in children, although they occur not infrequently during epidemics of pneumonia in military camps, hospitals, and jails. They differ from the ordinary form of pneumonia only in duration and rapidity of convalescence. In the shortest personal case there was unmistakable consolidation within 12 hours after its onset, and defervescence took place at the end of 48 hours (Chart I, page 7). The termination is by a rapid crisis.

(4) *Obstructive Form.*—This term was popularized by A. H. Smith. It is applied to cases in which there is great obstruction to the passage of blood through the lungs, arising from the extent of the consolidation or from the existence of intense pulmonary congestion, with more or less œdema. Unusual strain is thus put upon the right ventricle, which is unable to propel the blood with normal velocity through the pulmonary circulation. The right auricle and ventricle are overdistended, while the aorta and its branches are

underfilled. The superficial veins are also empty, having passed the major portion of their contents into the large venous trunks.

A certain amount of pulmonary obstruction is an element in every case of pneumonia, but in this form it is a predominant factor. The amount of obstruction is measured in great part by the pulmonary second sound. If the obstruction is marked and the power of the right ventricular systole is adequate, the pulmonary arterial tension will be increased and the closure sound of the pulmonary valve loud and accentuated. It is further obvious that this accentuation of the pulmonary second sound will cease under two conditions, viz., lessening of the pulmonary obstruction, or decrease in the power of the right heart. Whether it is due to the former or the latter cause must be determined by the general condition of the patient, and the evidence of lessened or increased rapidity and ease of breathing.

Extensive involvement of lung tissue, some dulness and numerous râles over the uninvaded portion of the lungs, marked cyanosis, rapid and embarrassed breathing, together with a pulmonary second sound at first accentuated, later becoming extinct, with progressive increase of the unfavourable symptoms, constitute the clinical picture of the obstructive form of pneumonia.

(5) *With Nervous Symptoms Predominating.*—This form of pneumonia, simulating meningitis, occurs mainly in children, and at one time was thought to be associated especially with pneumonia of the apex. It sets in with headache, high fever, delirium, convulsions, tremor of the muscles, and perhaps cervical retraction. The chest symptoms may be so overshadowed by the meningeal, that it is an excellent rule to consider head symptoms in a child as demanding an examination of the chest. The possible existence of true meningeal inflammation as a not uncommon complication requires some reservations, but it occurs at the height, or toward the close, of pneumonia, whereas the pseudo-meningitis initiates the disease.

Pneumonia occurring in chronic alcoholics may at first glance be quite unsuspected, the symptom group being that of delirium tremens. An increased pulse, respiration, and temperature will usually prevent misinterpretation, although in a considerable proportion of cases there is no pain, cough, or sputum, and the respiration rate may be normal.

(6) *With Gastro-intestinal Symptoms.*—In children, vomiting and diarrhœa may be so marked and persistent as to divert attention from the chest. This rarely happens with adults

(7) *With Marked Peritoneal or Abdominal Symptoms.*—In some cases there are violent abdominal pain with constipation and great

meteorism, simulating intestinal obstruction; or vomiting, abdominal tenderness, marked tympanites, and rigidity of the abdominal muscles—symptoms identical with those of peritonitis.

An involvement of the diaphragmatic pleura will account for the pain. This pain may be felt in the hypochondriac, umbilical, and lumbar regions. It may also in part explain the rigidity of the abdominal muscles, the latter contracting to limit the movements of the diaphragm. It is somewhat significant that the pneumonia in these cases almost invariably involves the lower lobe or lobes. It is also possible that there may be an actual peritonitis by extension, although this is a rare complication in pneumonia. In other instances the subsequent course of the disease justifies the diagnosis of a pneumo-typhoid, the pulmonary inflammation, as an initial event, coexisting with unusually violent abdominal symptoms.

(8) *With Delayed Appearance of Physical Signs.*—These are extremely perplexing cases when encountered, unless the rusty sputum is present, and are not very rare. The physical signs may not be manifest until the 5th day, or even until the 8th day, of the rational symptoms. The old explanation is doubtless correct, viz., that the consolidation begins centrally and spreads slowly to the periphery.

(9) *Secondary or Intercurrent Pneumonia.*—The pneumonia which occurs during the course of certain acute infections may be latent. The respiration rate may be only slightly increased, cough absent, and the physical signs of consolidation often lacking. The percussion note, usually over one of the bases, may be slightly dulled, the breath sounds feeble and accompanied by a few moist or crackling râles. This form of pneumonia occurs most frequently in typhoid fever, typhus fever, diphtheria, influenza, and the bubonic plague.

(10) *Terminal Pneumonia.*—Patients who are very ill and soon to die of some chronic disease, especially diabetes, chronic nephritis, arteriosclerosis, cardiac disease, or pulmonary tuberculosis, may have a pneumonia which is practically latent and is usually discovered post mortem. The respirations may be a little more rapid than normal and the temperature slightly raised.

(11) *Pneumonia as Modified by Age.*—In the old the disease is often latent, without chill, and with slight cough or expectoration. The general prostration is notable, while the physical signs are obscure and indefinite.

In infants and young children the cerebral symptoms are often prominent; an initial convulsion is not uncommon, and may be followed by stupor or coma. Pneumonia of the apex is more frequent than in adults, and rusty sputum is seldom seen.

(12) *Post-operative and Ether Pneumonia.*—The majority of cases of pneumonia following etherization or operation are broncho-pneumonic and not lobar. When the lobar form occurs it does not differ in any respect from the cases met with in medical practice. In a number of instances seen by request in the surgical wards, the pneumococcus was always found in the sputum.

(13) *Streptococcus Pneumonia.*—This form of pneumonia is said (Denny) to be characterized by special involvement of the upper lobe, a tendency for the inflammation to wander, a protracted and irregular fever with long-delayed resolution, and is finally determined by finding streptococci in the sputum.

(14) *Epidemic Pneumonia.*—Pneumonia may assume the proportions of an epidemic. The mortality is greater, and each outbreak may be characterized by a predominance of some one of the special types which have been described (cerebral, septicæmic, etc.).

(15) *Variations in Localization.*—The lower lobe of the right lung is the most frequent seat of the disease, and when the pneumonia is double, both lower lobes are those usually involved. Pneumonia of the apex is most common in children. *Wandering, creeping,* or *migratory* pneumonia is a variety in which there is a steady advance of consolidation from lobe to lobe. It can be readily followed by the resulting physical signs and recurring elevation of pulse, respiration, and temperature, and is apt to be protracted. *Massive pneumonia,* in which not only the air cells but the bronchi of one or more lobes are filled with exudate, is a rare variety. Because of the filling of the bronchi the physical signs are almost exactly those of pleurisy with effusion. *Central pneumonia* is not uncommon. The consolidation begins in the centre of a lobe, or at the root of the lung. The rational symptoms of pneumonia may be present for several days before the physical signs are rendered distinctive by the advance of the consolidation to the surface. See also (8) preceding. Indeed, resolution may take place without the development of more than barely satisfactory signs.

Here may be included cases of which I have seen several examples—*limited* or *incomplete hepatization.* The main points of these cases are: a moderately sudden onset, with chilliness but no marked chill, slight cough, slight non-localized pain in chest, pulse-respiration ratio of 3:5, temperature fluctuating irregularly between 100.5° and 102.5°, duration 3 to 4 weeks. A cursory examination of the chest is usually negative, but if a minute search is made, a single strip or patch of dulness and bronchial breathing will be found, usually in the right axillary region, corresponding to the adjacent borders of the middle and upper lobes, but not at any time involving the entire lobe.

(16) *Acute Pulmonary Congestion.*—This may be seen in numerous cases of epidemic influenza, especially in children. Strictly speaking it does not belong in this classification, because intense congestion is the first and last of the process, pneumonia not developing. Clinically its onset is sudden, temperature high, pulse-respiration ratio eminently pneumonic. There is no perceptible alteration of the percussion note, and the respiratory murmur is normal except for a very moderate harshness over both sides of the chest. The crepitant râle is occasionally heard. It subsides either with or without treatment in 12 to 24 hours. It is very apt to come on at night, and to subside by morning. There may be a recurrence on 1 or 2 subsequent nights. As this condition is undistinguishable from the first stage of pneumonia, the physician frequently receives praise to which he is not entitled.

TERMINATIONS, RELAPSES, AND RECURRENCES.—(1) *Delayed Resolution.*—It is well known that in many cases the physical signs of consolidation persist for a varying period, even for 2 or 3 weeks, with normal or nearly normal pulse, respiration, and temperature. Such cases do not come under this heading. The delayed resolution may follow an apparently typical frank pneumonia, but is more commonly a sequel to the septicæmic, latent, or incomplete forms. It is attended by a remittent, almost suppurative, temperature curve; not, however, with such wide excursions as may attend the latter. The pulse and respiration remain unduly rapid, 8 or 10 weeks elapsing before resolution is complete.

(2) Abscess, gangrene, and chronic interstitial pneumonia are rare terminations. (3) *Recurrences* are common; 3d or 4th attacks are not infrequent; and cases have been reported in which the recurrences numbered from 8 to 10 or more. (4) *Relapses* are rare, and the cases in which, after the temperature has been normal for 1 or 2 days, an apparent 2d attack runs its course, are probably instances of an irregular delayed resolution (OSLER).

COMPLICATIONS.—(1) *Pleurisy and Empyema.*—Aside from the common inflammation of that portion of the pleura which covers the surface of a consolidated area, there are cases in which the pleurisy is so severe and extensive that it rivals or surpasses the pneumonic element—pleuro-pneumonia. The effusion may be large, and is unusually rich in fibrin. Not infrequently the fluid is purulent, even though the pneumonia has been slight.

As pleurisy, serous or purulent, is by no means a rare sequel of pneumonia, and is not seldom overlooked, the possibility of its occurrence during convalescence should be borne in mind. A slight, persistent, and irregular rise of temperature, after it has been normal for several days, with dulness, absent or weak respiration and voice sounds, at the base, without râles, will constitute grounds of suspicion. A persistent leucocytosis is important and suggestive. Whether these signs are due to thickening of the pleura or to effusion may be ascertained by puncture.

(2) *Endocarditis.*—The ulcerative or malignant form of endocarditis originates, in from 15 to 25 per cent of cases, from a pneumonia. It affects mainly the left heart. The symptoms of ulcerative endocarditis are uncertain and sometimes absent. If the fever is irregular and prolonged, and chills, fever, and sweats occur, it is suspicious; if evidences of septic embolism become manifest and, in addition, meningitis occurs, together with the development of a loud diastolic murmur not previously present, the diagnosis of ulcerative endocarditis is quite certain.

(3) *Pericarditis.*—This is not so frequent as endocarditis, and occurs particularly in the double or left-side pneumonia of children. It is usually fibrinous, sometimes serous, rarely purulent. Præcordial pain, increased dyspnœa, and weak pulse may declare its existence. The physical signs of pericardial effusion may then be found, It is often overlooked or latent.

(4) *Meningitis.*—This very serious and fatal complication is fortunately rare, and when found is often associated with ulcerative endocarditis. Its existence is usually not recognised if, as is so often the case, it affects the convexity. If basilar, intense headache, marked cervical retraction, delirium, stupor, and coma will announce its presence.

(5) *Jaundice.*—Toxæmic icterus is very common in some epidemics.

(6) *Other Complications.*—These are: nephritis (rare), parotitis (occasional), peritonitis (rare), otitis media in children (not infrequent), peripheral neuritis (rare), diphtheritic colitis with diarrhœa (not uncommon), venous thrombosis in protracted cases (occasional), embolism of the femoral or other large artery (rare), and cerebral embolism with aphasia and hemiplegia (rare). Pneumonia may occur in a malarial subject, or malaria be manifest during the course of a pneumonia. The existence of the malaria is to be decided by the blood examination. Pneumonia not infrequently occurs in phthisical subjects. Rheumatic fever may precede an attack of pneumonia; or rheumatism may occur during an attack, although the redness, swelling, and pain in one or more joints which sometimes occurs, either at the height of the disease or after the crisis, may proceed to suppuration, and the pneumococcus has been found in the pus from the diseased joint. The articu-

lar complications are often accompanied by pleurisy or endocarditis.

DIFFERENTIAL DIAGNOSIS.—As a rule the diagnosis is readily made. Frank, typical cases present extremely distinctive and unmistakable symptoms. It may be overlooked in the very old or the very young, and in chronic alcoholics; so also with secondary, terminal, central, or incomplete pneumonias.

Certain diseases may simulate lobar pneumonia, or the latter may simulate other diseases.

Diseases Simulating Lobar Pneumonia.—(1) *Acute Pulmonary Congestion.*—This may simulate a beginning pneumonia (page 783).

(2) *Hypostatic Congestion.*—This develops in long-continued fevers, or diseases requiring a prolonged dorsal recumbent posture and attended by a weak heart. There is slight dulness at both bases, with a few moist crackling râles at the end of deep inspiration, with perhaps some harshness of the breath sounds. It can usually be distinguished from pneumonia by the absence of rusty sputum or of a rise in temperature, and is bilateral. Similar signs may, however, announce a secondary or terminal double pneumonia.

(3) *Pulmonary Œdema.*—In this condition, usually in connection with nephritis or cardiac valvular disease, there is sudden dyspnœa, cough, and expectoration, but it may be distinguished from pneumonia by the absence of fever, the presence of weak breath sounds and numerous fine and coarse moist râles on both sides of the chest, and the absence of marked dulness and bronchial respiration. The sputum in œdema is fluid, frothy, and not rusty.

(4) *Acute Bronchitis.*—In children this may simulate pneumonia, but there is no chill or convulsion, the respiration is not so rapid nor the fever so high as in pneumonia, dry and moist râles are heard over both chests, and there is no dulness or bronchial breathing.

(5) *Pulmonary Infarctions (or Apoplexy).*—These occur in connection with pyæmic or local septic processes, attended by thrombosis; or in chronic disease of the heart. There is more or less sudden dyspnœa, cough, and expectoration, but the sputum is fluid and often very bloody (resembling a small hæmoptysis) rather than tenacious and rusty, and may be expectorated once or twice only. If the infarct is large, signs of a circumscribed consolidation, usually in one of the lower lobes, may be manifest, but there is less fever than in pneumonia. If the embolus causing the infarct is septic, a true localized pneumonia may develop, usually terminating in abscess or gangrene. The sputum becomes rusty, and on examination the streptococcus, or, as in a case of my own, the staphylo

coccus (from an ulcerative cystitis) may be found by cultural tests, instead of the pneumococcus.

(6) *Broncho-pneumonia.*—This disease is usually preceded by bronchitis, or an acute infection (e. g., measles); dyspnœa and cyanosis are well marked; the sputum is thick, and streaked with blood rather than rusty; the fever is irregular, does not terminate by crisis, and may last for weeks. The physical signs are those of a diffuse bronchitis, dry and moist râles over both chests; the evidences of consolidation (dulness, broncho-vesicular or bronchial breathing) are often indefinite, and when well marked are usually limited to a vertical strip on each side of the spine, whereas in lobar pneumonia they are quite as well, and indeed often more distinctly, perceived over the lateral aspect of the chest.

(7) *Acute Pneumonic Phthisis.*—The onset, symptoms, and physical signs of this disease may exactly resemble those of lobar pneumonia until the 8th or 10th day, when the fever continues, the patient grows worse instead of better, and ultimately the signs of softening gradually develop, the expectoration becomes muco-purulent and greenish, and contains tubercle bacilli and elastic tissue. A differential diagnosis is impossible until sufficient time has elapsed for the appearance of the distinctive symptoms. The evidences of this disease as contrasted with lobar pneumonia may be summarized as follows: Tuberculous family or personal history, recurring chills and sweats common, fever more often distinctly remittent, more rapid loss of flesh, herpes labialis not present, crisis absent, usually affects one apex and extends downward; opposite apex often involved; disease continues with unabated severity beyond the 10th day, evidences of softening (gurgling râles, cavernous, or amphoric breathing, etc.) occur, and elastic tissue and tubercle bacilli (numerous examinations required) appear in sputum, which is abundant, purulent, and green.

(8) *Pneumo-typhus.*—See pages 732 and 741.

(9) *Pleurisy with Effusion.*—Mistakes rarely occur except in children. The onset of pleurisy is not so sudden, there is usually chilliness rather than a rigour, the fever is not so high and declines by slow lysis, the cough is frequent and dry, with scanty or absent expectoration, and the general prostration is comparatively slight. Herpes is absent or rare. The physical signs differ materially. In pleurisy the affected side is distended, vocal fremitus is absent (not increased), there is flatness, with a marked sense of resistance, rather than dulness, and the line of flatness may change position as the patient moves. Auscultation shows absent or diminished voice sounds or egophony; absent or diminished respiratory murmur, or *distant* bronchial breathing, and usually an absence of râles. There is evi-

dence of displacement of the heart or the liver. Finally, puncture will afford positive proof of the presence of fluid.

Diseases Simulated by Lobar Pneumonia.—(1) *Gastro-intestinal Disturbance.*—A routine examination of the chest in children will do away with any mistake of this kind.

(2) *Delirium Tremens.*—Make a routine examination of the chest.

(3) *Meningitis.*—Usually as in (1) preceding. If, however, the pneumonia is complicated by meningitis, the symptoms of the latter usually occur at the height, or toward the end, of the disease, whereas the simulated meningeal symptoms are present from the onset. In true meningitis the presence of marked cervical retraction, Kernig's sign (page 300), exaggerated reflexes, hyperæsthesia, strabismus, paralysis and other pressure signs, and perhaps lumbar puncture will serve to determine its existence.

(4) *Cerebro-spinal Fever.*—See page 753.

(5) *Typhoid Fever.*—In pneumonia with rapid supervention of the symptoms of the typhoid status, a differential diagnosis may be quite impossible if the case is not seen from the outset, unless the rose spots appear, or a positive Widal reaction is obtained.

PROGNOSIS OF LOBAR PNEUMONIA.—The disease is very fatal in drunkards, and in people of 60 years or over (60 to 80 per cent). The younger the patient (except in infants under 1 year) the better the prognosis. Meningitis as a complication is always fatal; endocarditis adds much to the gravity. Toxæmic or septicæmic ("typhoid") symptoms are especially unfavourable, so also are hyperpyrexia (105° or over), great dyspnœa, marked cyanosis, rapid spread of consolidation from one lobe to another, or involvement of both lungs, and steadily increasing rapidity and weakness of the heart with pulmonary œdema.

XXVII. TUBERCULOSIS

I. ACUTE GENERAL (DISSEMINATED) TUBERCULOSIS

Acute miliary tuberculosis results in many cases from the rupture into a vein of a tuberculous nodule, after which the bacilli are carried by the blood stream to various organs of the body. Whatever its origin, the condition is usually at first an acute generalized infection, and may so remain; but later in its course the infection may be predominantly localized either in the meninges (most common) or the lungs; or it may be thus localized from the onset. Three varieties are recognised, as follows:

General or Typhoid Form.—(1) *Symptoms.*—The general symptoms are those of an infectious disease without localizing symptoms, closely resembling typhoid fever. Malaise, weakness, chilliness, and fever, sometimes occurring rather abruptly, initiate the disease. Epistaxis is uncommon. The fever is irregular, and is often the first notable symptom. It rises in the evening (often to 103° or 104°) with decided morning remissions, or it may be of the inverse type; occasionally the temperature may be nearly normal for several days, and rise again; in exceptional cases it may be normal or subnormal. As the disease advances the pulse becomes rapid and feeble (rarely dicrotic), the face is flushed, the tongue dry and brown. Moderate bronchitis and somewhat hurried respiration are common. There are prostration, mental dulness, mild delirium or stupor, and, occasionally, diarrhœa. The spleen is somewhat enlarged; the urine is scanty, high coloured, often with a trace of albumin, and the diazo-reaction may be present; a reddish eruption, or toward the end petechiæ, may appear. There may be a moderate leucocytosis. Jaundice is rare.

(2) *Differential Diagnosis.*—Acute general miliary tuberculosis so closely simulates typhoid fever that the most acute diagnostician may be at a loss to decide which is present until the lapse of time permits the finding of some distinctive symptoms. The differential points are given elsewhere. (See page 742.)

Acute Tuberculous Meningitis.—This may occur as a localization of a general miliary tuberculosis, or the meningeal symptoms may be so pronounced that it should be classed as a meningitis rather than a general infection.

(1) *Symptoms.*—The onset may be abrupt. More commonly after an illness (measles, pertussis), a fall, or indefinite failure of health, the patient (usually a child) loses appetite, flesh, and strength, and may become irritable and peevish. In a week or so the initial or irritative stage comes on, manifested by headache (often intense), nausea and vomiting, chilliness, and fever. If the onset is sudden, convulsions, coma, or delirium may occur. Light and noise aggravate the headache, which is so severe that the child cries out suddenly (hydrocephalic cry) or screams continuously. The pulse is at first rapid, becoming slow and irregular; the respirations are usually unaltered. Constipation is the rule. Sleep is restless, and disturbed by starting or waking in alarm; muscular twitchings are common; vertigo and cutaneous hyperæsthesia are often manifest; cervical retraction is frequently observed, and the pupils are usually contracted. During the first stage the fever may rise to 102° or 103°. Leucocytosis is usually present throughout.

The symptoms slowly shift into those of the paralytic (or pressure) stage. The child becomes dull and apathetic, and is delirious if disturbed; the vomiting may persist, but more commonly lessens; the bowels are obstinately constipated, and the abdomen becomes scaphoid. The pupils are unequal or dilated; the respiration sighing or irregular; the pulse slow and intermittent, or irregular. The *tache cérébrale* can be produced. Convulsions are not infrequent, and nystagmus, strabismus, ptosis, or optic neuritis (blindness) may become manifest, and deafness may develop. Cervical retraction or spasm, and various forms of paralysis (hemiplegia, monoplegia), are common. Gradually a typhoid state ensues, with dry tongue, diarrhœa, rapid pulse, and involuntary passage of urine and fæces. Cheyne-Stokes respiration is not uncommon. The eyes are partly open and the eyeballs rolled upward. The temperature varies, rising and declining at short intervals. In some cases a rise to 106° or 107°, followed by a sudden fall to normal or below, heralds the fatal ending.

The duration of the disease, which is probably always fatal, varies from 2 to 4 weeks.

(2) *Differential Diagnosis.*—To distinguish tuberculous meningitis from the other meningitides may be difficult unless some pre-existing tuberculous focus (lungs, glands, bones) can be found, or choroidal tubercles are discovered by the ophthalmoscope. Lumbar puncture may afford a fluid containing tubercle bacilli. If the fluid obtained is free from any variety of micro-organism it also argues for a tuberculous affection. The onset in the tuberculous form is slower and the fever is not so high as in the suppurative variety, and in the latter there is a marked leucocytosis, which is slight or absent in the former.

Acute Miliary Tuberculosis of the Lungs.—(1) *Symptoms.*—When the general infection is pre-eminently localized in the lungs the onset may be sudden. More commonly, to the symptoms of the general infection are added cough, rapid breathing (even to 60 per minute), and perhaps pleuritic pain. The expectoration is muco-purulent, and may be rusty or streaked with blood. Dyspnœa and cyanosis are marked and much more severe than would be expected from the physical signs, which are those of a bronchitis of the smaller tubes. The percussion note is usually normal or hyper-resonant, especially over the front of the chest. If old tuberculous foci or broncho-pneumonic patches are present, especially in children, there may be slight dulness at the bases. On auscultation, fine or coarse, dry or moist râles are heard, and occasionally fine, soft pleural frictions or rubbing sounds. The respiration may be either weak or bronchial over the areas of localized dulness, if such be present. The spleen is swollen in the very acute cases. The pulse is rapid and weak. The fever runs from 102° to 103°. The duration of the disease varies, according to its intensity, from 10 or 12 days to weeks or months.

(2) *Diagnosis.*—The following circumstances point to the tuberculous nature of the disease: Dyspnœa, cyanosis, and diffuse bronchitis in adults; the presence of tubercles in the choroid; the occurrence of meningeal symptoms; the occasional finding of tubercle bacilli in the sputum; a history of cough, or tuberculous disease of glands, bones, or lungs; and in children a recent attack of pertussis or measles.

II. ACUTE PULMONARY PHTHISIS

Two varieties are recognised:

Pneumonic Form.—Clinically the *symptoms* are those of a prolonged lobar pneumonia. It may follow inspiration of an hæmoptysis, without a suspected lesion. The duration of the disease varies from 2 to 6 weeks, but rarely may be protracted to 3 or 4 months. The *differential diagnosis* is from lobar pneumonia (page 802).

Broncho-pneumonic Form.—(*a*) **Symptoms.**—Repeated chills, rapid pulse and respiration, high and irregular fever, and perhaps hæmoptysis, initiate the "galloping" form of phthisis. Night sweats and rapid loss of flesh and strength ensue. A muco-purulent sputum, at first scanty, later profuse, appears, containing elastic tissue and tubercle bacilli. The *physical signs* are at first indefinite, or are those of a diffuse bronchitis, but, shortly, areas of slight or marked dulness, with harsh broncho-vesicular or bronchial respiration and numerous moist and crackling râles will be found, usually first at the apices. These signs may be either unilateral or bilateral, more commonly the latter. Subsequently the evidences of softening and cavity formation supervene. If the disease is very acute the patient may develop typhoid symptoms and die in 3 weeks; more commonly the duration is from 6 or 8 weeks to 2 or 3 months; occasionally the acute symptoms subside and the case becomes one of chronic phthisis. The fatal broncho-pneumonia following measles and pertussis is, in the majority of instances, tuberculous, and an example of this form of acute phthisis.

(*b*) **Differential Diagnosis.**—(1) *Broncho-pneumonia.*—A personal or family tuberculous history, progressive emaciation, the finding of tubercle bacilli in the sputum, and the eventual occurrence of signs of softening will declare for tuberculosis.

(2) *Typhoid Fever.*—Typhoid fever with extensive bronchitis may be confused with this form of phthisis, but the presence of the rose spots, meteorism, and positive Widal reaction, with absence of tubercle bacilli in the sputum or of signs of cavity formation, will exclude phthisis.

III. CHRONIC PULMONARY PHTHISIS

The onset of chronic phthisis is gradual, except in the few cases, previously described, of general miliary tuberculosis with pulmonary localization, or acute pulmonary phthisis, which become protracted and merge into the chronic form. Owing to the immense therapeutic value of an early diagnosis it is best to describe the disease as in two stages, viz., initial or *incipient*, and *advanced*.

Incipient Phthisis.—(1) **Symptoms.**—In the majority of cases there is a gradually increasing sense of languor or weakness, with some loss of flesh and pallor of the face, unaccustomed shortness of breath on slight exertion, chilly feelings, loss of appetite, perhaps some pleuritic pain, and digestive disturbance. Then slight fever and moderate night sweats may occur. There may or may not be slight cough and expectoration, and physical examination of the chest may be either practically negative or reveal signs of disease which are just sufficiently marked to be recognisable.

The onset and manner of invasion may vary. The initial symptom in many instances is an *hæmoptysis*, an occurrence which should be considered indicative of tuberculosis unless subsequently proved to be otherwise, with which physical signs may coexist or, perhaps, not become manifest for weeks or months after. *Gastro-intestinal symptoms*, such as epigastric distress, acidity, eructations, flatulence, or vomiting, may be so prominent as to overshadow the pulmonary disease. The gastric disturbances are sometimes associated with *anæmia* and its symptoms in the young. Such cases are very common and are often misdiagnosed. A regularly *intermittent fever*, with chills and sweats, is not uncommon, and frequently leads to a mistaken initial diagnosis of malaria. *Pleurisy* with effusion, or a *dry pleurisy* at one apex, occur not infrequently as early symptoms, followed at varying intervals by evidence of pulmonary involvement. Most commonly *bronchitis*, following exposure, and occurring in persons who have catarrh of the nasopharynx or who have suffered from repeated colds, initiates the disease. The cough continues and the physical signs of a localized bronchitis at one apex are found. In some instances the presence of *tuberculous lymph glands* antedates the pulmonary disease, which is often latent. In other cases the earliest symptoms relate to the larynx. Finally, the disease may be *latent* and progress even to cavity formation before the occurrence of symptoms (perhaps hæmoptysis) sufficiently marked to attract attention; or it may be quite overshadowed by the presence of grave disease elsewhere.

(2) **Physical Signs.**—The physical signs of the incipient stage may be at first negative or very indefinite, but sooner or later become plainly manifest. Inspection may show the phthisical chest, with deficient expansion, either general or limited to one apex. There may be impaired resonance or slight dulness over an apex, more commonly the right. Auscultation over this apex shows the presence of fine, moist, crackling râles at the end of inspiration. The breath sounds are frequently interrupted or cogwheel, and feeble, or harsh and broncho-vesicular, or the expiration may be high-pitched and prolonged. The voice sounds, especially the whispered voice, are transmitted with more than normal intensity. As time passes and consolidation becomes more extensive, perhaps with cavity formation, the case enters the:

Advanced Stage.—(1) **Symptoms.**—The cough increases and may keep the patient awake at night. At times slight, it may be so violent and paroxysmal as to cause vomiting. The sputum, at first mucous, becomes greenish-gray, and finally takes on the nummular form (said to signify cavities). Pain over an apex, or more commonly in the lower chest, anteriorly or posteriorly, perhaps felt only upon coughing and due to pleurisy, is not uncommon. Hæmoptyses occur in from 60 to 80 per cent of all cases; in the early stages usually slight, from erosion or congestion of the bronchial mucosa; in the later stages often copious, from erosion of small vessels. The bursting of an aneurism of the pulmonary artery may cause a large and immediately fatal hæmorrhage in far-advanced cases.

The respiration is quickened, but not in proportion to the extent of the disease. Marked subjective dyspnœa is not common. The pulse is usually rapid, often running to 120 or 130. Anæmia, with a relatively great diminution of hæmoglobin, is always marked. Nausea, vomiting, anorexia, gastric disturbances, and perhaps diarrhœa, are common. Night sweats may be drenching and frequent. The loss of flesh is progressive. In women, menstruation usually ceases at an early period; rarely it becomes profuse. Pregnancy may cause a temporary arrest of the disease. The urine may present a febrile albuminuria; or if amyloid disease supervenes, polyuria and tube casts; or if renal tuberculosis is present, pyuria or hæmaturia. The notoriously hopeful mental attitude of the patient throws an interesting sidelight upon the efficacy of "mental healing" in serious organic disease.

Fever is not only the earliest symptom of the disease, but constitutes the most reliable index of its activity and progress. It may be intermittent or remittent. In some cases, even with extensive disease, the temperature may remain normal, or perhaps subnormal, for varying periods. When consolidation is advancing the intermittent and remittent types may alternate irregularly from day to day. If the type is intermittent, normal or below normal in the morning with sweating, rising to 102° to 105° in the evening with a flush upon the face, it is usually an indication of large cavities and progressive breaking down of lung tissue (hectic, septic, or suppurative fever).

(2) **Physical Signs in the Advanced Stage.**—*Inspection* shows an unusual prominence of the clavicles, scapulæ, and ribs. In many instances the congenital phthisical chest is seen. The supraclavicular and infraclavicular spaces are depressed, more so on the side of the lung which is most affected, and on the same side there is deficient expansion. Visibility of the cardiac impulse over a large area (2d to 5th left interspaces) is indicative of disease at the left apex, with retraction of that portion of the lung which is normally interposed between the heart and the chest wall. Clubbed fingers are frequent, and there may be yellowish-brown patches of pityriasis versicolor over the front of the chest.

Palpation confirms the existence of deficient expansion, and reveals an increase of vocal fremitus over consolidated portions of lung or over cavities. Pleural effusion or great thickening causes it to be absent or diminished.

Percussion should be practiced upon the supraclavicular and infraclavicular, supraspinous, and interscapular spaces, in the last on a level with the fifth dorsal spine in particular, always comparing both sides. Normally the note over the right apex is a little higher pitched than that over the left. More or less marked dulness indicates consolidation. If the dulness is very slight it is most easily determined at the end of inspiration. Dulness of a "wooden" quality usually indicates extensive fibrosis of the lung. Absolute flatness with retraction of the chest signifies a thickened pleura and widespread consolidation, or, if the intercostal spaces are bulging and motionless, a large pleural effusion. A tympanitic or dull tympanitic note over the upper part of the lung may be due either to a superficial cavity, or to an area of consolidation reaching from the chest wall to the main bronchi. In miliary tuberculosis, or numerous minute spots of consolidation, or a large number of small cavities, the percussion note may be nearly normal. Myoidema (page 552) may be observed.

Auscultation shows the presence of pleural or pleuro-pericardial friction sounds. According to the amount of consolidation, the breath sounds vary from prolonged expiration, through broncho-vesicular up to tubular or bronchial breathing. Jerking or "cog-wheel" inspiration is common, but not always significant. Early in the disease fine, crackling, or subcrepitant râles are heard, especially toward the end of inspiration; at a later period fine or coarse râles, either moist or dry, indicate increased secretion due to the accompanying bronchitis; and large mucous or gurgling râles indicate the presence of cavities. A cardio-respiratory murmur is not infrequent; so also is a systolic murmur in one or both subclavian arteries. The voice sounds, both spoken and whispered, are intensified (bronchophony, pectoriloquy) over areas of consolidation or over cavities.

The presence of a *cavity* can not always be determined with certainty, as any one who controls his clinical diagnosis, when possible, by the results of the autopsy room, will freely admit. Several of the signs of a cavity may be most accurately imitated by an area or shaft of consolidated lung extending from chest wall to main bronchi. The signs which usually indicate the existence of a cavity are: a tympanitic, dull tympanitic, amphoric, or cracked-pot percussion note, or Wintrich's and Gerhardt's phenomena (pages 438 and 440); bronchial, cavernous, or amphoric respiration, whispering pectoriloquy, or amphoric bronchophony, and large gurgling or bubbling râles which may have a ringing, metallic, or amphoric quality.

The *duration* of the disease varies from many months to many years.

Complications of Chronic Phthisis.—Laryngeal phthisis, usually secondary, evidenced by aphonia and dysphagia; gangrene (occasional); pneumonia (not infrequent as a final event); pleurisy, usually dry, sometimes with serous effusion; pneumothorax or pyopneumothorax (common); amyloid disease of the liver, spleen, kidneys, or intestines; intestinal tuberculosis; endocarditis (not common), meningitis; peripheral neuritis.

Diagnosis of Chronic Phthisis.—The diagnosis of an advanced case is readily made by the discovery of the tubercle bacilli in the sputum, conjoined with the well-marked physical signs. The disease is, of course, most difficult to discover at the very time when its recognition is of the greatest importance, namely, at the beginning. It is probable that if the physician resisted his lifelong habit of optimism and erred in the opposite direction in cases of suspected incipient pulmonary tuberculosis, the mortality list would be shorn of a measurable proportion of its bulk. Phthisis often masquerades under the guise of "malaria" or "bronchitis," and hæmoptysis is simply "from the throat." A gradual and unaccountable loss of strength and flesh, with a slight evening rise of temperature, even without physical signs, should be treated exactly as if the examiner felt sure that tubercles were forming. Repeated examinations of the sputum for bacilli should be made. The presence of localized moist or crackling râles at one apex, especially if conjoined with slight dulness, and harsh respiration or prolonged expiration, with or without the finding of bacilli, will in the vast majority of cases decide for tuberculosis. So also with the discovery of a dry pleurisy at one or the other apex. It should be remembered that while the disease usually starts in the apices, especially the right, it may first appear in the lower lobes or any other part of the lung.

(1) *Apical Catarrh.*—Rarely there is a non-tuberculous catarrh of one apex evidenced by fine moist râles, mucous sputum, and slight fever, which passes away in a few weeks, repeated examinations failing to reveal tubercle bacilli. The diagnosis of this condition should be made with the greatest caution.

(2) *Bronchiectasis.*—The chronic cough of bronchitis and the physical signs of cavity (caused by dilatation of the bronchial tubes) may be mistaken for those of chronic tuberculosis, but as the error can arise only in the later stages of phthisis, the discrimination is readily made by finding the bacilli in the sputum.

Prognosis of Chronic Phthisis.—In the earliest stages, with proper care, the outlook is not unpromising; in advanced cases, almost hopeless. Unfavourable symptoms are anorexia, rapid pulse, persistent high fever, steady loss of weight, and chronic diarrhœa. Such complications as involvement of the larnyx, pyopneumothorax, tuberculous meningitis, and amyloid disease are usually forerunners of the end.

IV. FIBROID PHTHISIS

In certain cases of chronic phthisis there is an extensive formation of fibrous tissue in one apex or one lung. When fully developed the clinical condition is practically the same as in chronic interstitial pneumonia (*q. v.*), from which fibroid phthisis can be distinguished only by the finding of tubercle bacilli in the sputum. The affected side is retracted and shrunken, the spine is laterally curved, and the shoulder of the same side prominent. The expansion is poor or absent; the percussion note dull and characteristically wooden; the heart is uncovered (if the left lung is affected) or drawn to the right; the breathing at the base is weak, absent, or distant bronchial, perhaps with râles of all sizes; the vocal fremitus is diminished. The duration of the disease varies from 10 to 20 years, or longer.

V. TUBERCULOSIS OF THE SEROUS MEMBRANES

The signs and symptoms of tuberculosis of the *pleura* and the *pericardium* are the same as those of pleurisy (*q. v.*) or pericarditis (*q. v.*) from other causes.

Tuberculosis of the Peritonæum.—Most common between the ages of 20 and 40, and twice as frequent in women as in men.

(1) **Symptoms.**—Extremely variable. The disease is often latent and is discovered accidentally during a cœliotomy for some other condition; or the symptoms resemble those of a typhoid fever; or may begin acutely. An early symptom is abdominal discomfort or pain, localized or general; and if the pain is severe it is apt to be continuous, with marked remissions of varying duration, and associated with tenderness. In the acute cases the fever may rise to 103° or 104°; in the more chronic cases the temperature is usually subfebrile, or may be normal, or even steadily subnormal, for days at a time. The fever is apt to rise coincidently with the attacks of pain. Effusion into the peritonæum (ascites), which may be hæmorrhagic, is common, usually moderate, but may be so abundant as to cause very considerable distention. Tympanites in varying degree is found, especially in the more acute cases, but occurs also in the chronic forms. Digestive disturbances, anorexia, nausea (perhaps vomiting), and either diarrhœa or constipation may be present. The latter may be very intractable. The skin may become pigmented, and there is usually a progressive loss of flesh and strength. In addition to ascites and meteorism the physical examination of the abdomen may reveal tumourlike masses, usually situated in or near the median line. Coils of intestine may become adherent, forming a rounded mass, which is tympanitic or dull tympanitic on percussion; or portions of the fluid effusion may become encapsulated by adhesions forming cystic collections, flat or dull on percussion; or the omentum may become shrivelled and thicken into a sausage-shaped transverse cord; or the mesenteric glands are enlarged and matted together; or indurated masses are found in the pelvis.

(2) **Differential Diagnosis.**—In the latent cases a diagnosis, at least in the early stages, may be difficult or impossible. If with the signs of a chronic peritonitis there is evidence of tuberculous disease in the lung, kidney, testes, Fallopian tubes, lymphatic glands, or bones, and especially if pleurisy with effusion coexists, the peritonitis is probably tuberculosis. The ascites, if present, is to be distinguished from that due to cirrhosis of the liver by the presence of marked abdominal pain, tenderness, and tumourlike masses, and an irregular fever; and the absence of enlarged spleen and gastric or intestinal hæmorrhages. It may be extremely difficult or impossible to decide between an encysted effusion and an ovarian cyst. In the latter case the growth is slower and more clearly defined, there is little or no loss of strength or flesh, usually no fever, and not so much abdominal pain and tenderness. No evidence of past or present tuberculous lesions can be found.

Cancer of the peritonæum may produce puckering and cording of the omentum, and tumourlike masses, as in tuberculous peritonitis, but the more rapid progress of the former and the finding of malignant disease (especially visceral) will aid in differentiation. Acute tuberculous cases can not be distinguished from ordinary acute peritonitis, or in some instances from enteritis or strangulated hernia. Those with slow onset, abdominal tenderness, meteorism, and continued low fever can not be differentiated from typhoid fever except by the Widal test, rose spots, or lapse of time. The tuberculin test in all these cases may be of service.

Under favourable conditions (cœliotomy?) and in latent and ascitic cases the *prognosis* is better than was formerly supposed.

VI. Tuberculosis of the Genito-urinary Organs

I. **Tuberculosis of the Kidney.**—This is more common in men than in women (3 to 1), and in mid-life.

(1) *Symptoms.*—The clinical manifestations are those of a pyelitis. There is frequent urination, pain or discomfort over one or both loins, with tenderness, and palpable, though rarely very marked, symmetrical enlargement of one or both kidneys. The urine is acid and contains pus, albumin, epithelium, occasionally small caseous particles, and tubercle bacilli. Hæmaturia may occur. In advanced cases, and especially when both kidneys are involved, chills, sweats, irregular fever, and progressive emaciation ensue.

(2) *Diagnosis.*—Tuberculous pyelitis, evidenced by pyuria, may exist for many years without causing much discomfort. The diagnosis of a renal tuberculosis depends (in addition to the symptoms just described) upon the discovery of tuberculous disease elsewhere (especially in the testis), and of tubercle bacilli in the urine. Ureteral catheterization, when practicable, will determine whether one or both kidneys are involved. The tuberculin test is helpful.

(3) *Prognosis.*—Usually but not invariably fatal in from one to three years after a diagnosis has been made. Many recoveries have occurred after nephrectomy when but one kidney is diseased.

II. **Tuberculosis of Ureter, Bladder, and Prostate Gland.**—Usually secondary to renal tuberculosis. The symptoms are those of a chronic cystitis without apparent cause. For the urinary characters see page 712. There is apt to be persistent fever and progressive loss of flesh in well-developed cases. The kidneys and testes should be carefully examined, and the absence of a vesical calculus assured. If the prostate is involved, there is extreme irritability of the bladder, hard nodules may be felt by rectal examination, and the passage of a sound is excessively painful. The urine should be examined for tubercle bacilli.

III. **Tuberculosis of the Testis.**—The finding of a tuberculous testicle is of much importance in making a diagnosis in suspected renal tuberculosis or tuberculous peritonitis. The epididymis is greatly enlarged, in some instances equalling the gland itself in size, and it may be painful. Syphilis of the testicle, with which tuberculosis may be confounded, affects the body rather than the epididymis, and the enlargement is more irregular.

IV. **Tuberculosis of the Fallopian Tubes and Ovaries.**—The symptoms are those of ordinary salpingitis and ovaritis, but if occurring in a weakly, especially unmarried, woman, with a tuberculous family history, and associated with extensive adhesions, ill-defined abdominal swellings, and continuous slight afternoon fever, are suspiciously indicative of the tuberculous character of the affection. Ultimately it tends to a fatal termination.

VII. TUBERCULOSIS OF THE LYMPHATIC GLANDS (SCROFULA)

Tuberculous adenitis occurs mainly in children, and is almost always local, involving special groups of lymph glands; very rarely nearly all the lymph glands in the body are implicated. The gland groups most commonly affected are the cervical (frequently), bronchial, and mesenteric, less frequently the axillary and inguinal.

Cervical Tuberculous Adenitis.—(1) *Symptoms.*—The submaxillary glands are usually the first to be involved, subsequently the postcervical, supraclavicular, and axillary may follow suit, and the disease may extend downward to the bronchial glands as well. Both sides are usually affected, one to a greater degree than the other. Unilateral enlargement of the supraclavicular and axillary glands may herald the beginning of a tuberculous pleurisy or pulmonary phthisis. The affected glands slowly enlarge from the size of a bean to that of an English walnut or a hen's egg. They are smooth, firm, and at first separate, but later the several tumours coalesce and become matted together, forming rounded but irregular masses. The overlying skin is freely movable, unless one or more glands suppurate, at which time the skin becomes adherent and the abscess (unless operated) points and discharges, leaving a persistent sinus. During rapid growth or suppuration, fever, anæmia, and loss of flesh become manifest.

(2) *Diagnosis.*—Slowly enlarging glands in a child are probably tuberculous if there exists at the same time chronic naso-pharyngeal catarrh, enlarged tonsils, suppurative otitis media, eczema of the

scalp, ear, or lips, conjunctivitis or keratitis, or known tuberculous disease elsewhere.

Tuberculosis of the Tracheo-bronchial Glands.—Usually occurs in children and, in the majority of cases, pulmonary tuberculosis coexists, although the glands alone may be involved.

(1) *Symptoms.*—These are mainly mechanical and arise from the pressure exerted when the mass of diseased glands becomes sufficiently large—i. e., constitutes a mediastinal tumour. From pressure on the recurrent laryngeal nerve arises a paroxysmal cough; and dyspnœa, also paroxysmal, which may be either croupy or asthmatic in character. Pressure on the superior vena cava causes cyanosis, puffiness, or œdema of the face. Distinct physical signs are not common. Anteriorly there may be dulness over the first piece of the sternum; or posteriorly on either side of the spine from the 2d to the 5th dorsal vertebræ. The mass of glands may transmit the breath sounds from the trachea and bronchi with such intensity that they resemble those heard over a cavity, viz., bronchial, cavernous, or amphoric respiration. A suppurating gland may rupture into a bronchus, and a considerable quantity of pus, blood, and cheesy matter may be suddenly expectorated.

(2) *Diagnosis.*—The occurrence of paroxysmal cough and dyspnœa, with swelling of the face, in a child who is suffering from phthisis or other form of tuberculosis, enables a diagnosis. In the absence of evidences of tuberculosis elsewhere it may be impossible to say that the cough and dyspnœa are due to tuberculous bronchial glands. The physical signs *per se* are not reliable.

Tuberculosis of the Mesenteric Glands.—Generally these glands are involved secondarily to intestinal tuberculosis, but the infection may be primary. In children the glandular disease may greatly predominate over the intestinal lesions (*tabes mesenterica*). In this there is a chronic diarrhœa, the stools are fluid and offensive, the abdomen is swollen and tympanitic, and if the flatulent distention can be relieved, the enlarged glands may be felt as small, firm, rounded, somewhat movable tumours, most commonly lying near the navel or in the right iliac fossa. There is moderate fever, marked anæmia, and great emaciation. Tuberculous peritonitis is a common coexistent lesion.

VIII. TUBERCULOSIS OF THE ALIMENTARY CANAL

Tuberculosis of any portion of the alimentary canal except the intestines is rare. Tuberculous ulcers—sharply defined, irregular, indurated base, with yellowish floor, not inclined to bleed—are met with on the lips (very rare); on the tongue, in which case, in contradistinction to syphilis, the submaxillary glands are not enlarged and the iodides do no good; on the tonsils, often associated with cervical adenitis; on the pharynx, by extension from a laryngeal tuberculosis; and in the œsophagus and stomach. In accessible ulcers scrapings may be examined for tubercle bacilli, or inoculations made.

Intestinal Tuberculosis.—The infection is often primary in children (from milk); in adults almost always secondary to disease of the lungs. It occurs in one half of all cases of chronic phthisis.

(*a*) *Symptoms.*—Persistent diarrhœa, often alternating with constipation, is a prominent symptom. The stools are thin, offensive, and, if the large intestine is involved, may contain mucus and blood. There may be moderate colicky pain and localized tenderness.

(*b*) *Diagnosis.*—A chronic diarrhœa occurring in the course of chronic pulmonary phthisis is usually tuberculous, unless due to amyloid disease of the intestines, in which case evidences of amyloid disease of the spleen, liver, or kidneys may be found. Primary tuberculosis of the intestines is recognised with great difficulty. Anæmia, loss of flesh, fever, the discovery of enlarged mesenteric glands, and the finding of tubercle bacilli in the fæces (in the absence of pulmonary disease in which the germs may be swallowed) may lead to a diagnosis, but it may be necessary to wait for the development of tuberculosis in some other part of the body.

Chronic tuberculosis localized in the cæcum or appendix may, in consequence of the development of a tumorous mass, simulate a subacute, chronic, or recurring appendicitis, or a carcinoma of the cæcum. The discovery of tubercle bacilli in the stools may enable a diagnosis.

IX. TUBERCULOSIS OF OTHER ORGANS

Liver.—This is infrequent except in connection with general tuberculosis and tuberculous peritonitis. In the last the perihepatitis may involve the portal vessels and give rise to ascites. Otherwise there are no symptoms of clinical importance. *Spleen.*—Enlargement of the spleen is constant in acute miliary tuberculosis, because of its involvement in the tuberculous infection. *Suprarenal Capsules.*—Generally chronic, often associated with tuberculosis elsewhere. It is clinically important because of its frequent existence in Addison's disease (*q. v.*). *Brain and Spinal Cord.*—Acute tuberculous meningitis (page 804) has been considered. Chronic tuberculosis of the brain (meningo-encephalitis) and spinal cord causes symptoms of tumour of the brain (*q. v.*), or spinal tumour (*q. v.*), or spinal meningitis (*q. v.*). *Mammary Gland.*—Indicated by the presence of circumscribed nodules which often ulcerate and form fistulæ. The nipple is retracted, and the axillary glands are enlarged in most cases. Tuberculous (cold) abscess may occur. The diagnosis is to be made from the appearance of the ulcers and fistulæ, the finding of tuberculous disease elsewhere, and the demonstration, by the microscope or by inoculation, of the presence of tubercle bacilli in scrapings from the ulcer or fistula.

X. Diagnosis of Tuberculosis

Aside from the symptoms and physical signs of the various forms of tuberculosis, the diagnosis depends upon the following tests:

Finding Bacilli.—The presence of the tubercle bacilli in sputum, effusions, urine, pus, or scrapings from the lesions; or the results of inoculation of these substances into guinea-pigs.

Cutaneous Tuberculin Test.—*Method.*—That of Von Pirquet is to cleanse with ether, the skin of the inner forearm, and place 2 drops of concentrated old tuberculin about 4 inches (10 cm.) apart. First scarify a spot between the drops, then through each in succession, as uniformly as possible without drawing blood. A chisel-pointed borer about $\frac{1}{16}$ inch (2 mm.) in diameter is best for this purpose. Use by rotating it quickly between thumb and finger. After scarifying, place a small flake of absorbent cotton on each drop to prevent flowing, and, having left it 5 minutes, gently wipe off the drop. No covering is needed. There is no contraindication, and no danger. Fever reactions follow only when the scarification by carelessness is carried through the skin. The local irritation causes slight or no annoyance.

Reaction.—A slight traumatic swelling follows the test. The specific reaction appears within 24 hours as a red papule, at least $\frac{3}{16}$ inch (5 mm.) in diameter. Torpid reactions may develop later than 24 hours, and usually are found in clinically unsuspected adults. With a doubtful or negative result, the test, on repetition after a week in a different site, may be found positive. The reaction reaches its maximum in 48 hours and fades out slowly, ultimately leaving no trace.

The modification of Moro, the inunction of a 50 per cent tuberculin ointment, has no advantages, and is more uncertain.

Diagnostic Value.—A positive reaction has most significance in childhood, decreasing in diagnostic importance with increasing age. It should precede other tuberculin tests, and under the age of 15 is of equal value. Since 70 to 90 per cent. of apparently healthy adults react, the test is usually only valuable in confirming the diagnosis when the reaction is prompt and intensive. A negative result after repetition is valuable in excluding tuberculosis.

(4) Conjunctival Tuberculin Test.—A single drop of a 1 per cent solution of old, or a 0.5 per cent solution of precipitated, tuberculin, in sterile normal saline, is instilled into the eye by pulling down the lower lid and holding it until the drop is diffused without flowing out on the cheek. If no reaction occurs after 48 hours, a 2 per cent or 1 per cent solution, respectively, may be employed in the *other* eye. Only healthy eyes should be used, and a history of past disease of the eye or lids, and of scrofula, should be a contraindication to the test. It cannot be recommended for general use, since it has no advantage over the skin test except in producing fewer reactions among apparently healthy persons, and may be harmful, especially to children, and on repetition in the same eye (E. L. BALDWIN).

Subcutaneous Tuberculin Test.—A reaction after the hypodermic injection of old tuberculin (KOCH) of standardized strength.

Dosage and Administration.—The initial safe dose, of a proper tuberculin, for an adult, is 0.0005 c.c.; if no reaction occurs give 0.001, 0.003, 0.005, and 0.008, or even 0.01 c.c. In children 0.0001 c.c. is a safe initial, and 0.003 a safe maximal, dose. For diluting the tuberculin (in order to measure the dose) a 0.25 per cent phenol, or a physiological salt solution, may be used. A cylindrical graduate, and a pipette reading to hundredths of a cubic centimeter are required. All apparatus should be sterilized. Make the injection subcutaneously in the subscapular region, having cleaned the skin with alcohol.

For at least 2 days previously the temperature of the patient must be recorded every 2 hours. The patient is to be kept quiet afterward, but not necessarily in bed. The 2-hour record should be continued. If no reaction occurs the subsequent doses may be given on the evening of the 2d day following each negative injection.

Reaction.—The symptoms are malaise, headache, pain in back and limbs. Anorexia, sometimes nausea and vomiting, a greater tendency to cough, and, if the reaction is severe, marked prostration. The temperature often rises to 101°; sometimes not over 100°; exceptionally to 107°, without obvious bad consequences. It falls to normal the day after. Variable symptoms are dyspnœa, substernal oppression, increased expectoration, vertigo, faintness, or drowsiness.

Changes in the physical signs (harsher respiration, more abundant crepitations) may, or may not, occur. A tuberculous larynx, gland, or joint may exhibit a distinct local reaction. If tuberculosis of the bladder or kidney is present, pus, blood, and, perhaps, tubercle bacilli, may be found in the urine. Redness, swelling, and pain at the point of injection occur in differing degrees, and may be severe. Fever *immediately* following the injection is not due to tuberculin.

Marked general and localizing symptoms, even with but slight fever (1°), following a dose of 0.003 c.c., or less, of tuberculin, may be considered a positive reaction (L. BROWN).

Contraindications.—If the patient's temperature rises to 100° at any time of day tuberculin should not be used. It is contraindicated by severe dyspnœa, night-sweats, hæmoptysis (within a month), extensive glandular tuberculosis, cardiac disease, nephritis, meningitis, and epilepsy; or for 3 weeks after any febrile ailment.

Indications for Use.—"Tuberculin should be used diagnostically as a last resort. When a patient with suspicious symptoms, with indefinite physical signs, and with no tubercle bacilli in the sputum on repeated examinations, wishes a positive diagnosis, tuberculin should be administered" (L. BROWN).

Diagnostic Value.—Some advanced cases of pulmonary tuberculosis, with bacilli in the sputum, do not react; so also with healed or encapsulated lesions. But, so far, no case of early pulmonary tuberculosis has been reported as failing to react to a dose of 0.010 c.c., or less, of old tuberculin. It may, however, be stated that a person, *without symptoms or physical signs,* who reacts to tuberculin, does not require treatment for tuberculosis.

XXVIII. SYPHILIS

1. **Acquired Syphilis.**—(*a*) **Primary Stage.**—In from 2 to 4 weeks after inoculation the initial lesion appears as a small red papule which increases in size, becomes eroded, and finally forms a small ulcer with a raised, movable, and characteristically gristly or indurated base—the hard chancre. Usually seated upon the prepuce or the vulva, it may occur just inside the urethra, or upon the finger (especially in physicians), lip, tongue, or elsewhere. The lymph glands, by way of which the affected region is drained, undergo a painless enlargement, occurring about 2 weeks after the appearance of the initial lesion. Syphilis is caused by the *Spirochæta pallida.*

(*b*) **Secondary Stage.**—In from 6 to 12 weeks from the appearance of the primary lesion the constitutional symptoms become manifest. There is *fever,* usually mild (101°) and continuous; or it may be remittent, more rarely distinctly intermittent. There is weakness, headache, slight backache, and general aching, often with a reddened and sore throat. *Anæmia* with a sallow or slightly yellowish tint of the skin (syphilitic cachexia) is present and may develop quite rapidly. The lymph glands all over the body may become somewhat enlarged and indurated. *Skin eruptions* appear, first and most commonly the macular (roseolar) syphilide, reddish-brown "coppery" spots occurring especially upon the anterior aspect of the trunk and arms, and remaining for one or two weeks. It may recur at a later period. This rash when seated in the nose and throat causes coryza and angina. The papular syphilide, resembling acne, and occurring mainly upon the face and trunk, is next most common. If the papules suppurate, as they may in the severer forms of the disease, the condition may simulate smallpox.

Syphilitic psoriasis, a squamous or scaly, copper-coloured syphilide, occurs especially on the palms and soles. Mucous patches (characteristic and contagious), well-defined flattened growths with a moist surface covered by a grayish secretion, are found not only upon the mucous membrane of the lips, tongue, gums, cheeks, and pharynx, and the muco-cutaneous junctions of the anus, vulva, and penis, but also in the moist cutaneous folds of the perinæum, groins, axillæ, navel, and webs of the toes. The tonsils may show small ulcers; and papillary hypertrophy of the mucous membranes may cause soft warty outgrowths or condylomata, especially about the anus or vulva. Whitish spots (leucomata) may be seen upon the tongue, particularly in smokers. White patches (leucoderma) surrounded by an increased deposit of pigment may occur, especially on the neck. Loss of hair, perhaps including eyelashes and eyebrows (syphilitic alopecia), frequently takes place, usually 3 or 4 months after the infection. A painful periostitis, the pain growing worse at night, may become manifest. It is usually circumscribed (nodes or exostoses), and occurs especially upon the tibiæ, clavicles, and cranial bones. Syphilitic onychia or paronychia may deform or destroy the nail. Three to six months after the initial lesion iritis may develop; choroiditis and retinitis are rare. Epididymitis and parotitis may occur. Abortion or miscarriage is common.

(*c*) **Tertiary Stage.**—The lesions which are commonly recognised as tertiary may appear as early as 6 months from the occurrence of the initial sore, while secondary lesions are still present; ordinarily a latent period intervenes, and it is several years before the tertiary manifestations begin. The characteristic lesions of the third stage are certain cutaneous lesions, gummata (syphilomata) especially of the viscera, diseases of the bone, and amyloid degeneration.

(1) *Cutaneous Manifestations.* — The tubercular syphilide appears as small nodules, which break down, resulting in well-defined, rounded, and deep ulcers, which may coalesce with neighbouring lesions, healing in one part and extending at another (serpiginous ulcer), with a thick, yellowish discharge. After healing, the cicatrix resembles that of a burn. These lesions may be covered by a conical stratified crust (rupia) which is quite characteristic. Gummata originating in the subcutaneous tissues may grow to the size of a walnut. They may be absorbed, or may break down, forming a deep ulcer and a deep scar.

(2) *Gummata of the Mucous Membranes.*—These result in ulcers which may cause deforming scars; or destroy bone or cartilage, if such underlie the affected mucous membrane. Thus gummata of the mouth and nasopharynx may perforate the nasal septum and flatten the nose; or perforate and partly destroy the hard and soft palate; or cause the adhesion of the soft palate to the posterior wall of the pharynx. Gummatous ulceration of the rectum may result in cicatricial stricture.

(3) *Gummata of the Bones, Periosteum, and Muscles.*—The cranial bones in particular, less frequently the long bones, are subject to gummous periostitis. The gummata are at first hard, but subsequently soften, ulcerate, and expose carious bone; perhaps causing perforation of the skull when the cranial bones are affected. Gummata growing from the bodies of the vertebræ may impinge upon the spinal cord; and similar growths may involve the joints and periarticular tissues. Diffuse gummous infiltration of the fingers or toes constitutes dactylitis syphilitica.

Circumscribed, slightly movable gummata may occur in the muscles; and a chronic myositis may result from syphilis.

II. **Congenital Syphilis.**—The symptoms of inherited syphilis may be present at birth; usually they appear between 4 and 8 weeks after birth.

(1) *Symptoms at Birth.*—The infant is poorly developed, emaciated, and blisters or bullæ (pemphigus) are usually present on the wrists and hands, ankles and feet. The epiphyses of the long bones may be separated from the shaft (osteochondritis); the spleen and the liver are enlarged; the lips and angles of the mouth are fissured and ulcerated; and there is a marked coryza ("snuffles").

(2) *Early Symptoms after Birth.*—The infant may be born plump and healthy, and so remain for from 4 to 8 weeks, at which time it begins to snuffle, the skin around the nostrils ulcerates, and the nasal bones may become necrosed and the root of the nose flattened down. The skin is sallow, and usually first about the mouth, arms, and genitals, erythematous, eczematous, macular, or papular syphilides develop. The lips and angles of the mouth become characteristically fissured (*rhagades*) and ulcerated. Alopecia, ulcerations of the skin and mucous membranes, enlargement of the spleen (of diagnostic value), enlargement of the liver (not significant), slight swelling of the lymph glands, and rarely subcutaneous and mucous-membrane hæmorrhages may occur. The child cries, becomes peevish, sleepless, anæmic, and thin.

(3) *Late Symptoms.*—In cases which recover from the early manifestations, the late symptoms may appear either at the second denti-

tion or at puberty. The child's general development is faulty, and even at the age of puberty the patient may retain the characteristics of a much earlier period. The late symptoms are interstitial keratitis or iritis, rapid onset of deafness, perhaps due to labyrinthine disease, Hutchinson's teeth (page 241), chronic periostitis, especially of the tibia (sabre-shaped), and atrophy of the testicles.

III. **Visceral Syphilis.**—**Syphilis of the Brain and Cord.**—The symptoms are due to the presence of gummata; or a gummatous meningitis or arteritis; or localized sclerosis. *Cerebral syphilis* presents the symptoms of brain tumour (*q. v.*), epilepsy (*q. v.*), dementia paralytica, or apoplexy (*q. v.*). *Spinal syphilis* presents the symptoms of tumour of the cord (*q. v.*), or chronic spinal meningitis (*q. v.*).

Syphilis of the Lungs.—In rare cases this may simulate chronic phthisis, but the diagnosis is difficult. If the symptoms and signs of chronic phthisis, or of chronic interstitial pneumonia with bronchiectasis, occur in a person who is unquestionably syphilitic, and repeated examinations fail to reveal the presence of tubercle bacilli, it may be considered syphilis (gummata or fibrosis) of the lungs. Janeway has called attention to the occasional existence of fever in tertiary syphilis. Prior to the publication of his paper I had under my care a physician who had fever (101° to 102°), cough, expectoration, and some physical signs of chronic phthisis, but also presented gumma in the liver, amyloid kidneys, profound anæmia, and a rapid arteriosclerosis, with absence of tubercle bacilli in his sputum. There were also tibial nodes, pigmented scars, and a history of a very persistent sciatica. In this case I made a diagnosis of pulmonary syphilis.

Syphilis of the Liver.—Gummata form in the substance of the liver, and as a result of their absorption the liver becomes deeply lobulated. A chronic fibrous inflammation, affecting mainly Glisson's capsule, may coexist, causing a perihepatitis, with fibrous bands running inward along the portal canals. Amyloid disease of the liver may supervene.

(1) *Symptoms.*—There may be evidences of hepatic cirrhosis (ascites, slight jaundice, and digestive disturbances); or the liver may be enlarged and irregular, and the spleen also enlarged, with polyuria, albumin, and casts (amyloid disease); or the gummata may form a tumour in the right or left lobe of the liver. The disease may be latent. The ascites is due to pressure by gummata, or contraction of cicatricial tissue, upon the portal vein or its branches.

(2) *Diagnosis.*—There must be good evidence of a previous syphilitic infection. If with this the liver is found (after removing

ascitic fluid if necessary) to be enlarged, distinctly lobulated, and irregular, or presents several rounded prominences, and the general health is not greatly impaired, the disease is probably syphilitic. The therapeutic test (potassium iodide) is helpful.

An enlarged amyloid liver with irregular gummata closely simulates cancer of the liver, but in the latter ascites and jaundice are rare; there is marked cachexia; there is often a softening or depression in the centres of the nodules; usually a history of a primary malignant growth in the breast, stomach, intestines, or uterus, and it is found as a rule only after 40 years of age.

Syphilis of the Rectum.—Occurs most commonly in women, and consists in the development of diffuse submucous gummata above the internal sphincter. The *symptoms* are those of a slowly progressive narrowing or stricture of the rectum. The stools may be muco-purulent and passed with straining and tenesmus, simulating a chronic dysentery. In addition to the history or presence of other syphilitic lesions the *diagnosis* depends upon a rectal examination which reveals a firm fibrous ring, differing materially from the crateriform ulcer of rectal cancer.

Syphilis of the Heart.—This occurs in the form of gummata or chronic fibrosis, or both.

Symptoms.—A sense of oppression, palpitation, and extreme irregularity of the heart action, dyspnœa, and præcordial pain or anginal attacks, occurring in a patient who is known to be the subject of tertiary syphilis, may enable at least a probable diagnosis of cardiac syphilis. Sudden death occurs in 33 per cent of these cases.

Syphilis of the Arteries.—Endarteritis and periarteritis may play an important part in the production of arteriosclerosis (*q. v.*), aneurism (*q. v.*), and paralytic dementia.

Syphilis of the Kidneys.—The usual manifestation of renal syphilis assumes the form of amyloid degeneration of the kidneys (*q. v.*).

Syphilis of the Testicles.—This may appear in two forms: a uniform or irregular painless atrophy of the testicles, usually involving one more than the other, and gummata of the testicles. As compared with tuberculosis of the testicle, syphilitic orchitis presents nodules mainly in the body of the testis, the epididymis usually escaping, and there is no tendency to ulceration. Malignant disease of the testicle develops more rapidly, is painful, and may ulcerate.

IV. **Diagnosis of Syphilis.**—The tendency to concealment on the part of the patient is to be remembered. When endeavouring to ascertain the existence of a previous syphilitic infection, the result of which may explain the present condition, the following points are to be determined:

Is there a history of a primary sore (genital or extragenital), followed by skin rashes (without itching), sore throat, and alopecia?

In women, have there been frequent abortions or miscarriages?

In children, is there a history of "snuffles" and skin rashes within the first 3 months after birth?

Examine nose, mouth, and throat for lesions, such as destruction of nasal septum and flattening of nose, perforation of hard or soft palate, ulcers, mucous patches, etc.

Examine the cutaneous surface for scars (groin, leg); the genitals for the primary scar and for syphilitic atrophy or enlargement of the testicles.

Examine the bones (especially tibia, clavicle, and cranial bones) for nodes, or necrosis.

Examine the teeth (for Hutchinson's sign); the eyes for iritic adhesions or interstitial (ground-glass) keratitis.

Examine the lymph glands for universal moderate enlargement and induration.

Remember that paralysis of single cranial nerves, or anomalous or atypical symptom groups (especially in disease of the nervous system), are apt to be of syphilitic origin; also that visceral syphilis presents symptoms which usually do not differ from those of non-syphilitic disease, and that the diagnosis of their syphilitic origin depends upon the history of infection and the finding of lesions which are accessible and characteristic.

XXIX. GONORRHŒAL RHEUMATISM

This form of systemic infection, manifested mainly by an arthritis, occurs as a rule during or toward the end of a gonorrhœa, but may show itself at any time in the course of a subsequent gleet.

Symptoms.—These are variable. There may be fleeting joint pains, without redness or fever; or polyarticular inflammation, with fever, redness, and swelling of the affected joints; or one articulation, especially the knee joint, may become greatly swollen and excessively painful (rarely suppurates), with but moderate fever; or the knee joint may fill with fluid, often without pain, redness, or swelling; or the bursæ and tendon sheaths in the vicinity of an articulation may become inflamed and cause thick swelling above and below the joint. Endocarditis (sometimes ulcerative), pericarditis, or pleurisy may occur, usually in the polyarthritic form. If suppuration takes place (generally in the knee joint) or ulcerative endocarditis supervenes, the symptoms may shift into those of a pyæmia.

Diagnosis.—The presence of a recent gonorrhœa affords a direct clew to the nature of the disease; occurring during a gleet, the latter may be overlooked and an incorrect diagnosis result. Compared with rheumatic fever (page 791) fewer joints are affected, the fever and constitutional symptoms are less marked, the disease is intractable and tends to chronicity, relapses are common, and antirheumatic treatment is of little or no service.

XXX. ANTHRAX

This disease, usually affecting sheep and cattle, and due to the *Bacillis anthracis*, may develop in man from accidental inoculation. There are several forms of anthrax.

Symptoms.—(1) **Malignant Pustule.**—At the point of inoculation (face, hand), 1 to 3 days after infection, a small red, burning papule forms, rapidly developing into a vesicle containing bloody serum, which soon breaks, leaving a dark or black scab lying in the centre of a large area of brawny induration and œdematous swelling. Red lines (inflamed lymphatic vessels) radiate outward, and there are secondary vesicles surrounding the original pustule. By the second day the constitutional symptoms are severe; prostration, high fever, sweating, nausea and vomiting, delirium, splenic swelling, and in bad cases coma and collapse. Death may occur in from 5 to 8 days; or the vesicle may slough off and convalescence begin.

(2) **Anthrax Œdema.**—The constitutional symptoms are grave and instead of the formation of a pustule there is widespread brawny œdema which may lead to extensive and often fatal gangrene.

(3) **Internal Anthrax.**—This occurs in two forms.

Wool-sorters' Disease.—In this there is a sudden onset, with rigour, fever (102° to 103°), headache, pains in back and legs, faintness and prostration. The pulse is rapid and weak, and in bad cases there are vomiting, diarrhœa, delirium, coma, collapse, and death within 24 hours. In some instances the infection manifests itself principally in the lungs, affording the symptoms and signs of bronchitis, or of a rapidly spreading septicæmic (typhoid) pneumonia.

Intestinal Form.—Sets in with a chill, followed by pain in back and legs, nausea, vomiting, abdominal pain and diarrhœa, with moderate fever; in bad cases, cyanosis, dyspnœa, mucous membrane hæmorrhages, petechiæ, and toward the close restlessness, convulsions, and muscular spasms.

Diagnosis.—The occupation of the patient is an important diagnostic factor. Anthrax occurs in hostlers, butchers, tanners, shepherds, wool-sorters, ragpickers, weavers, carpet and blanket makers, and in general those who work in hides, hair, or wool. External anthrax is recognised by the appearance of the pustule. Internal anthrax is diagnosed with great difficulty unless external anthrax coexists. In cases suspected because of the occupation, blood or local products, if such can be obtained, should be inoculated into mice or guinea-pigs.

Prognosis.—If the malignant pustule is promptly operated recovery is the rule. Internal anthrax is usually fatal.

XXXI. GLANDERS (FARCY)

This is a disease of the horse, due to the *Bacillus mallei*, but may be transmitted by accidental inoculation to man. It is localized either in the nose (*glanders*) or beneath the skin (*farcy*). The essential lesion consists of nodules which break down, forming ulcers on mucous membranes and abscesses in the skin. The disease may be acute or chronic.

Symptoms.—(1) **Acute Glanders.**—After a period of incubation of 3 or 4 days, sometimes 14 days, swelling, redness, and inflammation of the lymphatics at the point of inoculation is observed. At the same time there are headache, malaise, anorexia, fever, and joint pains. In two or three days nodules form in the nasal cavities, followed by ulceration and a muco-purulent discharge. The nose swells, and the cervical glands are much enlarged. Coincidently an eruption of red papules comes out on the face and about the joints, occasionally over the trunk. The papules rapidly become pustules (resembling those of variola), a septicæmic condition, often with the development of pneumonia, ensues, and the disease invariably ends in death in from 6 to 12 days.

(2) **Chronic Glanders.**—The symptoms are those of a chronic coryza with recurring ulcers. There may be weakness, occasional fever, loss of flesh, and wandering pains in the limbs.

(3) **Acute Farcy.**—The nose is not involved, but with the general symptoms of an acute septicæmia or pyæmia, nodular enlargements (farcy buds), which quickly suppurate, form along the course of the inflamed lymphatic vessels. The joints are painful and swollen, and abscesses may develop in the muscles. The disease is usually fatal in from 12 to 15 days.

(4) **Chronic Farcy.**—There are numerous subcutaneous nodules which suppurate and sometimes form deep ulcers. The lymphatics are involved slightly if at all.

Diagnosis.—The occupation of the patient (stableman, groom, hostler) is an important factor in the diagnosis. In suspected cases the *Bacillus mallei* should be sought for in the discharges; or a portion of the latter, or of a culture made from the discharges, should be inoculated into the peritoneal cavity of a male guinea-pig. In two days, if the *Bacillus mallei* is present, the testes are greatly swollen and proceed to suppuration.

Prognosis.—Acute cases die; chronic cases have a mortality of 50 per cent.

XXXII. ACTINOMYCOSIS

A chronic disease, mainly of animals, occasionally of man, caused by the ray fungus (*Streptothrix actinomyces*).

Symptoms.—(1) *Actinomycosis of Mouth and Jaw.*—The lower jaw is enlarged, or the side of the face is swollen, or an abscess forms at the angle of the jaw and discharges pus, which contains the organisms.

(2) *Pulmonary Actinomycosis.*—The symptoms are those of a putrid bronchitis (*q. v.*), pulmonary abscess (*q. v.*), or a chronic tuberculosis, with irregular fever, and offensive sputum; or an empyema (*q. v.*) may result.

(3) *Cerebral Actinomycosis.*—This is rare. The symptoms are those of brain tumour (*q. v.*) or cerebral abscess (*q. v.*).

(4) *Intestinal Actinomycosis.*—Gastric disturbance, diarrhœa, localized pain or tenderness; or symptoms of secondary hepatic abscess (*q. v.*), perforation peritonitis (*q. v.*), pericæcal abscess or appendicitis (*q. v.*), may result.

Diagnosis.—To be made only by the discovery of the parasite in the sputum or pus.

XXXIII. LEPROSY

A chronic disease caused by the *Bacillus lepræ.* There are two clinical varieties, which, however, may coexist in the same person. The period of incubation is usually from 3 to 5 years.

Symptoms.—(1) *Tubercular Leprosy.*—In the early stage there are slightly elevated, hyperæsthetic patches of erythema, usually on the face, arms, and knees. The patches generally become pigmented; rarely white and anæsthetic. At a later period somewhat tender nodules of varying size form, at first especially on the face, hands, and feet, with thickening, induration, and scaliness of the skin. The nodules may break down and form ulcers, which, healing, give rise to deforming cicatrices. The eyelashes and eyebrows fall out. Owing to involvement of the mucous membrane of the mouth, throat, and larynx, ozæna, cough, dyspnœa, hoarseness, or aphonia may result; blindness may ensue from keratitis by extension from an affected conjunctiva.

(2) *Anæsthetic Leprosy.*—There are at first nerve pains and areas of hyperæsthesia or numbness. Later there are patches of anæsthesia, which may be whitish or scaly, preceded or not by maculæ.

The accessible nerve trunks may be hard and nodulated (peripheral neuritis), and trophic disturbances become prominent. Bullæ form, generally upon the extremities, and, breaking, result in destructive ulcers. The fingers and toes may become contractured, and the phalanges undergo necrosis and be lost.

Diagnosis.—The dusky-red hyperæsthetic maculæ of the early stage, with the subsequent development of anæsthetic areas, are quite characteristic; while in advanced cases there is rarely any doubt. The bacilli may be found in a nodule, or the secretion from an ulcer, in suspected cases.

Prognosis.—Ultimately hopeless; but the disease is extraordinarily chronic, lasting from 4 to 20 or even 30 years.

XXXIV. TETANUS

This disease—caused by the *tetanus bacillus*, which grows in the soil, and particularly in manure—arises usually by inoculation through wounds, especially of the extremities; but it may be idiopathic. The period of incubation varies from 1 to 2 weeks. Acute, chronic, and cephalic forms are recognised.

Symptoms.—(1) *Acute Tetanus.*—The onset may be abrupt, but more commonly headache, malaise, and perhaps chilliness, occur, with slight stiffness of the neck, or of the muscles of the jaw. Tonic spasm of the masseters (lockjaw or trismus) soon develops, and the face assumes the *risus sardonicus*. The head is drawn back, the legs rigidly extended, and the body may be thrown spasmodically into a condition of opisthotonos, emprosthotonos, or pleurosthotonos. The arms are, as a rule, little involved. During the paroxysms there may be spasm of the glottis, dyspnœa, cyanosis, or rapid respiration, with sharp pain along the costal margins. The skin is covered with perspiration, and the temperature may or may not be moderately elevated. Hyperpyrexia (108° to 112°) may occur. The pulse is usually rapid. The paroxysms of spasm are excessively painful and recur spontaneously, or, in severe cases, as a result of external irritants, and last a variable time. The muscles remain tonically contracted in the intervals. The mind is not affected. Usually fatal in about 10 days. Tetanus may affect the newborn, the infection entering by way of the navel.

(2) *Cephalic Tetanus.*—If the wound of inoculation is on one side of the head or face there is trismus, dysphagia, and paralysis of the facial nerve on the same side as the injury.

(3) *Chronic Tetanus.*—The symptoms are those of the acute form, but are milder, interrupted by periods of relief from paroxysmal and tonic spasm, and the course of the disease is prolonged for weeks.

Differential Diagnosis.—The cardinal symptoms are, a wound (usually), a period of incubation, trismus, and rigidity of the neck.

(1) *Hydrophobia.*—In this there is a history of a bite from a rabid animal, mental disturbances, spasmodic dysphagia and dyspnœa, and an absence of trismus and opisthotonos.

(2) *Tetany.*—No history of a wound (except, perhaps, thyroidectomy). The spasm is usually limited to the four extremities, very rarely there is trismus, and the attitude of the hands is characteristic (Fig. 215, page 550).

(3) *Strychnine Poisoning.*—There is a history of ingestion of the poison, and the symptoms rapidly follow. The involvement of the jaw muscles, if present at all, comes late, the spasms occur soon after the onset of the symptoms, and there is muscular relaxation between the paroxysms.

Prognosis.—Traumatic acute cases give a mortality of 80 per cent; chronic cases somewhat less; in the newborn it is always fatal; in the idiopathic cases 50 per cent, in chronic cephalic cases 25 per cent, may recover.

Promising indications are a long incubation, no fever, no spasm except in neck and jaw, and a chronic course.

XXXV. HYDROPHOBIA

A disease—due to an unknown organism—of the wolf, dog, cat, skunk, cow, and horse. Communicated to man usually by bite of the dog. Three stages of the disease are recognised.

Symptoms.—(1) *Prodromal Stage.*—The period of incubation varies from 2 weeks to 3 or 4 months, usually 6 weeks to 2 months. At the end of this period there are great depression of spirits, malaise, slight fever, headache, anorexia, insomnia, hyperæsthesia of the retina or the auditory nerve, and perhaps darting pain or numbness in the scar, husky voice, and slight dysphagia.

(2) *Stage of Excitement.*—The patient is excessively excitable, and there is intense hyperæsthesia of the special senses and general sensibility. The throat stiffens, and attempts to swallow produce attacks of violent spasm affecting the muscles of the mouth, pharynx, larynx, and upper chest, with severe dyspnœa. During the paroxysms, which may be excited by the sight of water because of the dread of a seizure on attempting to swallow, or by bright lights, noises, draughts of air, or even by suggestion, there may be maniacal delirium, excessive salivation, and the utterance of odd sounds. In the intervals between the seizures the mind is usually clear, but delirium or mental aberration may be present. There are, in most cases, slight fever (100° to

103°) and intense thirst. In from 36 hours to 3 days the symptoms gradually merge into those of the

(3) *Paralytic Stage.*—The spasms cease, coma ensues, and in from 6 to 18 hours the patient dies from cardiac weakness and final syncope.

Diagnosis.—This depends upon the history of a bite, the spasm on attempting to swallow, the intense hyperæsthesia, the mental disturbance, and the final paralysis.

Lyssophobia (pseudo-hydrophobia) is a condition developing in nervous or hysterical persons within a few months after having been bitten by a dog. There are mental irritability, despondency, a fear of becoming rabic, and emotional seizures, during which the patient alleges an inability to swallow; but there is no fever, no increase in severity, the course of the disease is prolonged, and recovery almost invariably occurs.

Prognosis.—When fully established the disease is always fatal. Preventive measures (immediate cauterization, Pasteur's treatment) are of great importance.

XXXVI. BERI-BERI

An epidemic multiple neuritis. Four forms are recognised.

Symptoms.—(1) *Rudimentary Form.*—This begins with a feeling of weakness in the extremities, with paræsthesias and some anæsthesia in the legs, sometimes with slight œdema and muscular tenderness; moderate dyspnœa, palpitation, and abdominal uneasiness may be present. These symptoms last from a few days to several months and then pass away. Recurrences are common.

(2) *Paralytic or Atrophic Form.*—This begins as does (1) preceding, but the muscles of the extremities (sometimes of the face) soon become painful, paralyzed, and undergo atrophy. The tendon reflexes are abolished. Cardiac symptoms and œdema are slight or absent.

(3) *Dropsical Form.*—Beginning as in (1) preceding, the œdema becomes universal (general anasarca), and effusions into the serous sacs may take place. Dyspnœa and palpitation are common, while anæsthesia and muscular atrophy are not marked.

(4) *Acute or Cardiac Form.*—This begins as does the rudimentary form, but the symptoms of cardiac weakness are predominant. Death may occur in 24 hours in the very acute cases, but the disease is usually prolonged for several weeks.

Diagnosis.—In patients living in, or just from, the tropics, the occurrence of general œdema with the evidences of multiple peripheral neuritis (*q. v.*) will suffice for the diagnosis.

Prognosis.—The mortality varies from 2 to 50 per cent.

XXXVII. MOUNTAIN FEVER AND MOUNTAIN SICKNESS

The cases described as *mountain fever* are for the most part typhoid fever; perhaps more rarely lobar pneumonia (CURTIN).

The symptoms of *mountain sickness*—due to rarefied air—are severe headache, vertigo, dry throat, excessive thirst, intense dyspnœa, anorexia, nausea and vomiting (occasional), and profound weakness.

XXXVIII. EPHEMERAL FEVER–FEBRICULA

(*Simple Continued Fever*)

These terms are applied to a brief fever, without discoverable local lesions, and depending upon various and oftentimes undetermined causes. If the elevated temperature lasts for not more than 24 hours it is an ephemeral fever; if for from 3 to 6 days it is spoken of as febricula or simple continued fever.

Symptoms.—The onset is usually abrupt, but is sometimes preceded by malaise. The temperature runs from 101° to 103°, in children perhaps to 104° or 105°. There are headache, flushed face, anorexia, furring of the tongue, constipation, and in children restlessness, and occasionally drowsiness and nocturnal delirium. In infants and young children there may be a convulsion. The urine is scanty and high-coloured. Herpes labialis is common. The fever terminates by an abrupt crisis in from 1 to 6 days, often with free sweating and an increased flow of urine.

Causes.—By far the most common example of the febrile states grouped under this heading is the one-night fever of infants due to an acute indigestion or gastro-intestinal catarrh. Other examples are the unrecognised or abortive forms of typhoid fever, scarlet fever, measles, rheumatism, pneumonia, or tonsilitis; intestinal auto-intoxication by ptomaines; inhalation of noisome vapours from decomposing material (sewer gas, offensive autopsies); and exposure to the sun, or excessive muscular fatigue.

Diagnosis.—It is doubtless a fact that the term febricula constitutes in one sense a confession of ignorance, and that the frequency with which the diagnosis of this fever is made is in inverse ratio to the diagnostic thoroughness and acumen of the observer. When a careful search fails to reveal the rashes of the exanthemata, or evidence of tonsilitis, pneumonia, or other local cause of fever, and the elevation of temperature disappears in from 1 to 6 days without the development of the symptoms which characterize any other recognised disease, the diagnosis of febricula is justified.

XXXIX. MALTA FEVER

A disease caused by the *micrococcus melitensis.*

Symptoms.—The period of incubation varies from 5 to 10 days. The onset is gradual, often with slight chilliness, malaise, headache, anorexia, and fever which proves to be of a remittent type. The course of the disease is marked by periods of pyrexia, lasting from 1 to 3 weeks. At the end of each period the fever disappears for 2 or more days and is followed by another pyrexial period of varying duration. Several of these relapses may occur. While the disease usually lasts about 3 months it may continue for 6 months or even for 2 years.

During the febrile periods there is debility, progressive anæmia, splenic enlargement, and severe constipation; less frequently chills, delirium, neuralgias, diarrhœa, and painful arthritic swellings with effusion. In severe or malignant cases there may be hyperpyrexia and death within from 7 to 10 days. The disease resembles malarial fever, but the blood examination will separate one from the other. The serum reaction is diagnostic. The mortality is about 2 per cent.

XL. WEIL'S DISEASE

An acute febrile jaundice of unknown origin.

Symptoms.—The onset is sudden, with headache, backache, severe pains in the muscles, especially of the legs and face, and fever of a markedly remittent type. Jaundice appears by the 2d day, either slight (toxæmic) or marked (obstructive, with clay-coloured stools). The liver is tender and swollen, the spleen enlarged, the urine often contains albumin and casts, and hæmaturia may occur. The *prognosis* is favourable.

A more complete account of this malady will be found in the section on Tropical Diseases.

XLI. GLANDULAR FEVER

An infectious disease, sometimes epidemic—of unknown origin—affecting children (usually from 5 to 8 years of age), and characterized by swelling, mainly of the lymph glands of the neck. Its existence as a nosological entity is denied by some authors.

Symptoms.—The onset is abrupt, with pain on movement of the head and neck, perhaps with nausea, vomiting, and abdominal pain. There is fever (101° to 103°), possibly with some redness of the tonsils, but the anginal symptoms are slight and of no consequence. On the 2d or 3d day the characteristic tender glandular swellings appear, varying from a pea to a goose-egg in size. The subcutaneous tissues of the neck may be somewhat edematous, but there is no redness of the skin. The fever is of short duration, but the glandular swellings do not subside for from 10 days to 3 weeks. Suppuration rare; acute otitis media, retropharyngeal abscess, and hemorrhagic ephritis have occurred. The carotid lymph-glands are most com- ionly involved, next most frequently the postcervical, axillary, and iguinal, and occasionally the tracheo-bronchial (chest-pains and aroxysmal cough) and mesenteric glands. Both spleen and liver iay be acutely enlarged. The prognosis is favourable.

XLII. MILK-SICKNESS

An infectious disease of cattle ("trembles") communicated to ian by the ingestion of the milk or flesh of diseased animals.

Symptoms.—The prodromal symptoms are malaise, headache, nd anorexia. In a few days there ensue burning pain in the stom- ch, nausea and vomiting, excessive thirst, and obstinate constipation. he breath has a characteristic foul odour, the tongue is swollen and remulous; and in severe cases there may be restlessness, hebetude, oma, or convulsions, with the development of the typhoid status nd an ultimate fatal result. Slight fever is usually present, but nay be lacking. The duration of the disease varies from 3 days to or 4 weeks, with an average of 10 days. Recovery is the rule, but n the grave acute cases death may occur in 3 days.

XLIII. MILIARY FEVER (SWEATING SICKNESS)

This disease (of unknown origin) occurs at present only in Europe. n *mild* cases the symptoms are slight fever, anorexia, epigastric ppression, drenching sweats, and on the 3d or 4th day an outbreak f miliary vesicles, which burst and are followed by a scaly desquama- ion. In *severe* cases with an intense type of infection there are in ddition high fever, delirium, hemorrhages, and extreme prostration, r fatal collapse. The mortality is 7 or 8 per cent.

XLIV. FOOT AND MOUTH DISEASE

This disease, known in man as epidemic stomatitis or aphthous ever, is an acute infection—origin unknown—of cattle, sheep, and igs. It may be communicated to man by the ingestion of milk, but- er, or cheese from diseased cattle, or by direct inoculation.

Symptoms.—After an incubation period of from 3 to 5 days here are chilliness, fever, digestive disturbances, and the appearance f a vesicular eruption upon the lips, the inside of the cheeks, and he pharynx. The mouth and throat are hot and reddened, and alivation is present. There may be, particularly in children, a niliary or pustular eruption on the skin, especially of the hands. In he severe cases hemorrhages may occur. The duration of the dis- ase is about 1 week. The prognosis is usually favourable, but in ne epidemic the mortality was 8 per cent.

SECTION II

DISEASES OF THE DIGESTIVE SYSTEM

I. DISEASES OF THE MOUTH

(*See also pages 238, 239.*)

I. **Catarrhal Stomatitis.**—(*a*) *Causes.*—Dentition or gastro-intestinal disturbances in children, irritating or too hot food, and the acute infectious diseases.

(*b*) *Symptoms.*—The mucous membrane of the mouth is more or less extensively reddened, dry, and hot, with associated salivation and swelling of the tongue. Feverishness and discomfort, or sharp smarting when food is taken, may be present. The duration of this condition is about one week, sometimes longer.

II. **Aphthous or Follicular Stomatitis.**—(*a*) *Causes.*—"Canker" sore mouth is most common in infants and young children, either as an idiopathic affection or as a result of indigestion or a febrile attack; and occurs in adults when the general health is impaired.

(*b*) *Symptoms.*—Small vesicles appear on the inner surface of the lips or cheeks or edges of the tongue and soon rupture, leaving small and very sensitive superficial grayish ulcers with red areolæ. There is feverishness, a heavy breath, salivation, and a disinclination for taking food. If complicating some other disease, the symptoms of that disease will coexist. There may be successive crops of vesicles which will protract the disease beyond its ordinary period, which is from 4 to 7 days.

It is to be discriminated from thrush. See (IV) following.

III. **Ulcerative or Fœtid Stomatitis.**—(*a*) *Causes.*—Putrid sore mouth occurs most commonly in children during the first dentition, and may be epidemic, even in adults, in asylums, jails, and camps, where the hygienic conditions are poor.

(*b*) *Symptoms.*—The gums become swollen, red, spongy, and bleed readily, and upon them form linear ulcers with gray, soft, and sloughing bases. The mucous membrane of the lips, cheeks, and tongue is swollen (rarely ulcerated), there are salivation and a peculiarly foul breath. The submaxillary glands are enlarged, and the teeth may become loosened, perhaps with necrosis of the alveolar process. Nausea, vomiting, and an ill-smelling diarrhœa may be present, and the general symptoms may be of a severe grade. Except in marasmic or greatly debilitated children with extensive ulceration and alveolar necrosis, recovery generally occurs in about one week.

IV. **Parasitic or Mycotic Stomatitis.**—(*a*) *Causes.*—This affection (thrush, soor, muguet) is dependent upon the *Saccharomyces* (or *Oïdium*) *albicans.* It occurs mainly in bottle-fed infants. Predisposing conditions are uncleanliness of the mouth and of feeding utensils, and cachectic or diseased states in general, in adults as well as children.

(*b*) *Symptoms.*—Small, slightly elevated, pearly white or curdlike spots are seen, first upon the tongue, which increase in size and may coalesce. Subsequently the patches spread to the lips, cheeks, and hard palate, perhaps invading the tonsils, pharynx, and esophagus, and in extreme cases lining the entire cavity of the mouth and throat. The patches can readily be removed, in the majority of cases, without excoriating the mucous membrane or causing it to bleed.

(*c*) *Diagnosis.*—Microscopical examination of a bit of the membrane shows the branching filaments, with their spore-bearing ends, of the causative organism. This disease may be confounded with aphthous stomatitis; but aside from the microscopical examination, the latter may be recognised by its distinct red-bordered ulcers, usually few in number and very painful; nor can the grayish base of the ulcer be removed except with difficulty and not without bleeding.

V. **Gangrenous Stomatitis.**—(*a*) *Causes.*—Cancrum oris or noma is a rare disease, affecting children of from 2 to 5 years of age, and occurring usually during convalescence from the acute fevers. More than fifty per cent of the cases follow measles, less frequently it occurs after typhoid fever, scarlet fever, variola, and whooping cough. Debilitated and cachectic states also predispose. The exciting cause is probably a yet unknown micro-organism.

(*b*) *Symptoms.*—The disease begins as an irregular, dark, sloughing ulcer, usually on the inside of one cheek, more rarely on the gum. The process spreads rapidly, the cheek becomes swollen and brawny, and externally red and glazed; and later by extension of the sloughing shows a dark, gangrenous spot. The cheek may be perforated, and the disease may involve the jaw bones, tongue, chin, and even the eyelids and ears. The breath is intolerably offensive. The disease always remains unilateral. The constitutional symptoms are necessarily severe. There is great prostration, fever (103° to 104°), rapid pulse, delirium, and diarrhœa. Septic pneumonia (by inhalation) is common, and gangrene of the external genitalia (in female children) and colitis may occur.

The duration of the disease varies from 7 to 14 days, rarely longer, and it almost invariably has a fatal termination.

VI. **Mercurial Stomatitis.**—Owing to personal idiosyncrasies this may follow the use of repeated minute doses of a mercurial; or it may be an occupation poisoning.

Symptoms.—The early symptoms are tenderness of the teeth on snapping the jaws sharply together, a metallic taste in the mouth, and a fœtid breath. The gums become swollen, red, spongy, and sore. There is profuse salivation, the tongue is swollen, ulcers may form, the teeth become loose and, rarely, necrosis of the jaw ensues. There is a disinclination to take food, and diarrhœa may be present.

The duration of the disease varies from 2 to 4 weeks and recovery is the rule.

VII. **Subvarieties of Stomatitis.**—In the newborn there may be small ulcers of the hard palate, symmetrically placed on either side of the median line, which may involve the bone (PARROT). Similar ulcers on the hard palate may be caused in marasmic children by the irritation of a rubber nipple (BEDNAR). Jacobi has described a chronic recurring herpetic eruption of the buccal cavity in neurotic persons, sometimes coexisting with erythema multiforme.

II. DISEASES OF THE TONGUE

(*See also pages 242 to 250.*)

I. **Glossitis.**—*Causes.*—*Acute parenchymatous inflammation* of the substance of the tongue (rare) is due to injuries (biting, burns, stings); sometimes to local infection or mercurial stomatitis.

Symptoms.—The tongue is painful and greatly swollen, so much so that it may protrude beyond the teeth. Chewing, swallowing, and talking are difficult or impossible; there is salivation, the tongue may be dry and cracked, and obstructive dyspnœa, sometimes to a dangerous degree, may develop. The inflammation may proceed to suppuration, generally unilateral. The cervical glands are swollen, and there is fever, usually in proportion to the severity of the local manifestations. The duration of the disease is about 1 week, and recovery is the rule.

There is a *chronic superficial glossitis*, due to the persistent and excessive use of tobacco, spirits, and highly spiced foods. The dorsum of the tongue is reddened and fissured, and there may be smooth glazed patches bounded by deep furrows. A *glossitis desiccans* is also described, which is characterized by the slow formation of a number of deep fissures and indentations, in the depths of which there are ulcers and excoriations. The tongue has a ragged and uneven appearance.

II. **Eczema or Psoriasis of the Tongue.**—Rounded patches resulting from thickening and desquamation of the superficial epithelium, healing in the centre while spreading at the periphery, and coalescing with each other to give a maplike appearance—the geographical tongue. The patches may burn and itch. The condition may be transient, but is usually chronic or recurrent.

III. **Leucoplakia Buccalis.**—Smooth, white or pearly patches of thickened epidermis appear on the sides of the tongue, and similar spots may be found on the mucous membrane of the cheeks. They do not ulcerate, but may furnish the starting point of an epithelioma. The disease is chronic and intractable.

III. DISEASES OF THE SALIVARY GLANDS

I. **Ptyalism or Salivation.**—See page 238.

II. **Xerostomia.**—The salivary and buccal secretions cease, the mucous membrane of the buccal cavity, palate, and tongue becomes dry, red, and glazed, and cracks may be visible on the surface of the tongue. Chewing, swallowing, and speaking are difficult. "Dry mouth" occurs usually in neurotic or hysterical women, or follows a shock or fright, or in some instances may be due to a central lesion. Usually recovered from, but may prove intractable.

III. **Symptomatic** (*Suppurative*) **Parotitis.**—Acute swelling and inflammation tending to suppuration, and occurring in the course of certain acute specific infections, especially typhoid fever, less frequently scarlet fever, typhus fever, pneumonia, pyæmia, erysipelas, and secondary syphilis. More rarely it arises in connection with various diseases or injuries of the abdomen and pelvis. When occurring in febrile diseases it constitutes a bad prognostic omen.

IV. **Chronic Parotitis.**—A persistent, perhaps tender, enlargement of the parotid gland may follow mumps, or succeed mercurial or lead poisoning, or occur in the course of chronic nephritis and syphilis.

V. **Epidemic Parotitis.**—See page 757.

VI. **Gaseous Tumours** of the parotid gland or its duct.—A tumour in the course of Steno's duct, which may contain air, saliva, and pus, and reach the size of a walnut or an egg; or, very rarely, multiple small, crepitating tumours of the parotid gland may be encountered in players on wind instruments and in glass-blowers.

IV. DISEASES OF THE PHARYNX

(*See also pages 252 to 254.*)

I. **Disturbances of the Circulation.**—Hyperæmia, evidenced by a dusky reddened tint of the mucosa, with unusual visibility of the veins, is frequent in smokers, and is a part of naso-pharyngeal catarrh. Distended veins may be due also to cardiac valvular disease, and to pressure upon the superior vena cava (tumour, aneurism). Rupture may occur, simulating hæmoptysis. Œdema of the uvula and soft palate may occur in nephritis, or grave anæmia. The capillary pulse and carotid throbbing of aortic insufficiency may be visible in the pharynx, the strong pulsation of the artery simulating an aneurism.

II. **Acute Pharyngitis.**—The most common *causes* are exposure to cold, digestive disorders, rheumatism, or gout. The *symptoms* are dryness and soreness of the throat, with dysphagia, slight chilliness, and fever. There is a constant desire to clear the throat, the neck is stiff, the cervical glands may be slightly enlarged and painful, and the inflammation may extend to the larynx (hoarseness), or to the Eustachian tubes (slight deafness).

On inspection, there is a general dry, red, and congested condition of the mucous membrane, perhaps with œdema of the uvula. It is often a part of an acute catarrh of the nasopharynx and larynx. Recovery occurs within a week.

III. **Membranous Pharyngitis.**—False membrane may form in the pharynx (diphtheroid sore throat) due to the streptococcus. It can be distinguished from diphtheria only by culture.

IV. **Chronic Pharyngitis.**—"Clergyman's sore throat" may result from repeated acute attacks, and often occurs in smokers and persons who make much and vigorous use of the voice, or it may be a part of a chronic naso-pharyngeal catarrh.

The *symptoms* are a constant hawking, with a dropping of mucus from the upper pharynx. The mucous membrane, especially of the posterior pharyngeal wall, is duskily hyperæmic and studded with a variable number of projecting rounded bodies (enlarged mucous follicles). Occasionally the mucous membrane is dry and glistening (*pharyngitis sicca*).

V. **Ulcers in the Pharynx.**—See page 253.

VI. **Acute Infectious Phlegmon of the Pharynx.**—The symptoms are soreness of the throat, dysphagia, hoarseness, swelling and œdema of the pharyngeal mucosa, with rapid suppuration, swelling of the neck, interference with respiration, high fever, and prostration.

VII. **Retropharyngeal Abscess.**—As a rule occurs in healthy children under 2 years of age, but may be a sequel of infectious fevers, especially diphtheria and scarlet fever, or a result of caries of the bodies of the cervical vertebræ. The *symptoms* are pain, dysphagia, difficult and impeded breathing, sometimes cough, hoarseness, and stiffness of the neck. The *diagnosis* is made by inspection and palpation of the pharynx, by which is discovered a fluctuating tumour projecting from the posterior pharyngeal wall. The *prognosis* is good if operated early.

VIII. **Angina Ludovici.**—This is a rare disease, not so much of the pharynx as of the floor of the mouth and the cellular tissues of the neck. It is a streptococcus inflammation, occurring either idiopathically or as a result of the specific infections, especially diphtheria and scarlet fever.

The symptoms are: Swelling beginning in the submaxillary region of the side, and spreading to the floor of the mouth and the front of the neck. There is much pain, with dysphagia and difficult mastication and articulation. Grave dyspnœa may supervene from compression of the larynx or œdema of the glottis.

V. DISEASES OF THE TONSILS

(*See also page 252.*)

I. **Follicular** (*Lacunar*) **Tonsilitis.**—(*a*) *Causes.*—Not seldom precedes an attack of rheumatic fever, and is often caused by exposure to cold, fatigue, and sewer-gas poisoning. The streptococcus is commonly present. It occurs mainly between the ages of 10 and 25 years. It is often contagious, and repeated attacks in the same individual are very common.

(*b*) *Symptoms.*—The onset is usually abrupt, with chilliness or a chill, fever (102° to 105°), headache, backache, and general aching, which may be very severe, and occasionally initial vomiting. The tonsils are reddened, more or less swollen, and studded with punctate whitish spots corresponding to the distended lacunæ. The spots may coalesce and form patches nearly or quite covering the tonsils. Both tonsils are usually affected, although one often starts a day or two previous to the other. The cervical glands are enlarged and tender. There is dysphagia, furred tongue, foul breath, nasal voice, and scanty and high-coloured urine, sometimes with a trace of albumin. The pulse is rapid, and in children the respiration is accelerated. The constitutional symptoms are often out of all proportion to the local signs. The fever not infrequently subsides by crisis on the 3d or 4th day, but may be protracted for 8 or 9 days. Among the occasional *sequelæ* which occur are pneumonia, rheumatic fever, acute nephritis, endocarditis, pericarditis, and otitis media.

(*c*) *Diagnosis.*—There is often so little initial soreness of the throat that the patient considers the routine examination of the buccal cavity, which should always be made in acute febrile cases, to be quite unnecessary. It may be confounded with diphtheria (p. 771), but a history of repeated attacks is in favour of tonsilitis.

II. **Phlegmonous or Suppurative Tonsilitis.**—Quinsy occurs mainly between 15 and 35 years of age, and there is a strong tendency to repetition of the attacks in certain individuals. The causes are similar to those of follicular tonsilitis.

Symptoms.—The throat becomes sore and dry, and is excessively painful. One or both tonsils are seen to be swollen, sometimes meeting in the median line, reddened, and at first hard. The uvula, soft palate, and surrounding parts are reddened and œdematous. There is salivation and the secretion of a tenacious mucus, which is removed with difficulty by hawking. Swallowing is excessively painful, and perhaps impossible. The lower jaw scarcely can be moved, articulation is difficult, and the voice has a nasal quality. The cervical glands are swollen. In from 2 to 6 days suppuration occurs and fluctuation can be felt (if the finger can be introduced) usually in the tissues of the soft palate above and anterior to the tonsil, rather than in the gland itself. If the pus is not evacuated by incision the abscess bursts anteriorly, or more rarely toward the pharynx, with immediate relief of the symptoms. The constitutional disturbance may be severe; fever (104° to 105°), with a rapid pulse, occasional nocturnal delirium, and exhaustion.

The inflammation sometimes undergoes resolution, and may at times be apparently aborted by treatment (guaiacum, salicylates). In children under 15 years of age resolution is common within 3 to 5 days; in adults suppuration is frequent, usually unilateral, and the disease runs its course in from 8 to 10 days. The *prognosis* is favourable except in rare instances in which ulceration of the internal carotid or internal maxillary arteries with fatal hæmorrhage takes place, or a large abscess ruptures and the pus enters the larynx, causing fatal suffocation. Œdema of the larynx is seldom seen. Chronic enlargement of the tonsils may result from repeated acute attacks.

III. **Chronic Tonsilitis, and Adenoids of the Pharynx.**—Enlarged tonsils and adenoid hypertrophy are usually associated, but the latter may be present alone. Occurs mainly between the 5th and 15th years of age, and is most frequently the result of repeated naso-pharyngeal inflammations, or a sequel of the exanthemata and diphtheria.

(*a*) *Symptoms.*—The cardinal symptom is mouth-breathing, especially during sleep. The child is restless, wakes frequently, sometimes with dyspnœa or night terrors, the respiration is irregular, noisy, stertorous or snoring, and the breath is often fœtid. Cough, frequent hawking, occasional croupiness or asthmatic attacks, some dysphagia, more or less deafness and ringing in the ears from involvement of the Eustachian orifice, and impairment of taste and smell, may occur.

If the obstruction is allowed to continue the face assumes a dull and stupid expression, the mouth remains permanently open, the nose becomes small and its orifices contracted, the lips thicken, the jaws project, the hard palate is arched and narrowed, and the voice has a nasal quality. The chest may become barrel-shaped in consequence of repeated asthmatic attacks, or more commonly presents the deformity known as pigeon breast or funnel chest. Headache is frequent, wetting the bed is a not uncommon occurrence, and habit-spasm may coexist. The child becomes listless, stupid, and inattentive (aprosexia), forgets readily, and is a poor student. The digestion is impaired, there is a tendency to follicular tonsilitis and a marked liability to diphtheria.

(*b*) *Diagnosis.*—In a well-marked case the facies is characteristic. The enlarged faucial tonsils can be readily seen. T. R. French says that, in children, their presence is always a proof of the existence of pharyngeal adenoids. The flat or grapelike vegetations may, of course, be palpated.

VI. DISEASES OF THE ESOPHAGUS

(*For methods of examination, see pages 480, 481.*)

I. **Acute Esophagitis.**—(*a*) *Causes.*—Corrosive poisons, hot fluids, foreign bodies; occurs rarely in typhoid fever, pneumonia, smallpox, diphtheria, pyæmia, and thrush, and in connection with cancer of, or near, the esophagus.

(*b*) *Symptoms.*—The main indication is substernal pain, often intense, on swallowing. The pain may be dull and continuous. If an obstructing foreign body is present there may be spasm, with regurgitation of food, blood, and pus. The passage of a sound is painful.

II. **Ulceration of the Esophagus.**—This may occur in cachectic states, typhoid fever, and cancerous disease. Perforation of an ulcer in the upper portion of the esophagus causes a brawny swelling of the tissues at the root of the neck and in the supraclavicular space.

III. **Spasm of the Esophagus.**—Occurs in hysteria, hypochondriasis, epilepsy, chorea, and above all hydrophobia.

It is indicated by an inability to swallow solid food, although with rare exceptions fluids can be taken. That the obstruction is spasmodic, and not organic, may be inferred from its occurrence in neurotic young persons or elderly hypochondriacs, and confirmed by the passage of the tube, which may be temporarily arrested at the site of spasm, but after a moment of waiting will slip past the apparent obstruction. In middle-aged or old people great caution should be exercised in excluding cancer as a cause of the dysphagia.

IV. **Cancer of the Esophagus.**—May occur at any point in the tube, causing stenosis (unless the ulceration and destruction of tissue is extensive), with dilatation and hypertrophy above the seat of the disease.

(*a*) *Symptoms.*—Increasing dysphagia, progressive emaciation, pain, either constant or upon swallowing, and enlargement of the cervical lymph glands. Regurgitation of food or fluid, perhaps containing blood and tumour particles, occurs at once or from 10 to 15 minutes or longer after taking food, depending largely upon the site of the growth. Perforation may occur into the trachea, or a bronchus, or the lung, causing pulmonary gangrene or an inhalation pneumonia; or into the mediastinum; or into the aorta (fatal hæmorrhage); or pericardium (fatal pericarditis). Erosion of the vertebræ with compression of the cord has been noted; also laryngeal paralysis from pressure upon the recurrent laryngeal nerve. The *duration* of the disease varies from a few months to a year and a half, death usually occurring from inanition or perforation.

(*b*) *Diagnosis.*—The cardinal symptoms are progressive dysphagia and rapid emaciation occurring in patients over 50 years of age. It is necessary to eliminate other causes of stenosis: foreign bodies and cicatricial contraction by the history; aneurism (*q. v.*) or mediastinal tumour (*q. v.*) by the absence of pressure and other symptoms which characterize them. The stomach tube is to be passed without violence, and tumour particles may be brought up by it, an examination of which will confirm the diagnosis.

V. **Stricture of the Esophagus.**—(*a*) *Causes.*—This may be *congenital,* but is more commonly a *cicatricial* stricture caused by contraction of the scar tissue resulting from ulceration due to corrosive poisons. More rarely it is due to typhoid ulcers, gumma, or tuberculosis. *Pressure* stricture, the next most frequent, is due to aneurism, enlarged thyroid, mediastinal tumour or enlarged mediastinal glands, vertebral abscess, and sometimes pericardial effusion. Finally, cancer or polypoid tumour of the esophagus may be responsible for the stricture.

(*b*) *Symptoms.*—Dysphagia with emaciation and debility, varying with the completeness of the obstruction. Regurgitation of food, or fluid of alkaline reaction, occurs, at once if the obstruction is high up, in 3 or 4 hours if it is low down, and the esophagus above the stricture is dilated. Auscultation of the esophagus may possibly be of some service in determining the presence and locality of the stricture, but the use of the sound or tube will prove of much more value.

(*c*) *Differential Diagnosis.*—It is evident that, having determined the presence of esophageal stenosis as distinguished from spasm (III preceding), it is next requisite to ascertain the cause of the narrowing in the individual case. The characteristics of *cancerous stenosis* have been stated in IV, preceding. *Pressure stricture* is to be eliminated by a careful examination for thoracic aneurism (*q. v.*), mediastinal tumour or abscess (*q. v.*), thyroid enlargement (*q. v.*), disease of the cervical vertebræ, and pericardial effusion (*q. v.*). *Cicatricial stricture* usually causes more pain in swallowing, and a history of injury by a foreign body or of ingestion of a corrosive poison is usually obtainable. If past syphilitic or tuberculous disease is responsible for the cicatricial tissue, a corroborative history or characteristic lesions elsewhere may be found.

VI. **Dilatation of the Esophagus.**—This occurs in all cases of esophageal stenosis, the walls of that part of the tube above the obstruction becoming hypertrophied, and the tube expanding. Very rarely it is a congenital condition. The symptoms are chronic dysphagia and habitual regurgitation. If the sound passes readily into the stomach stenosis may be eliminated.

VII. **Diverticula of the Esophagus.**—A lateral sac or circumscribed dilatation of the esophagus may be due to pressure from within or traction from without.

(*a*) *Pressure Diverticula.*—These may occur in rapid eaters, usually men, who gorge large masses of food. The sac is most commonly situated on the posterior wall at the junction of the pharynx and esophagus. The muscular coat is weakest at this point, and giving way allows the mucous membrane to bulge outward, hernialike, opposite to and on a level with the cricoid cartilage. Once started it continually enlarges. The patient is conscious that at least a part of the food lodges too high up, and at intervals, when endeavouring to swallow, he retches and regurgitates the contents (which may be offensive) of the sac. The diverticulum may be so large that it forms a visible tumour or swelling in the neck, which when compressed causes the passage of its contents into the mouth. If the existence of a small and not palpable sac is suspected, a sound slightly bent at the end may be employed, the point being turned posteriorly during introduction, when it will enter the cavity.

(*b*) *Traction Diverticula.*—These are small conical depressions occurring usually in children, situated on the anterior wall of the esophagus on a level with the bifurcation of the trachea, and result from the adhesion of inflamed bronchial glands to the wall with subsequent shrinking and outward traction. They do not give rise to clinical symptoms.

VIII. **Rupture of the Esophagus.**—May occur as an effect of prolonged vomiting, generally when intoxicated, after a large meal. The clinical evidence of the accident is severe pain, emphysema of the neck and chest, collapse, and death.

VII. DISEASES OF THE STOMACH

(For the physical examination of the stomach, see pages 483 to 492. For the examination of the stomach contents, see pages 658 to 674.)

I. **Acute Catarrhal Gastritis.**—(*a*) *Causes.*—Usually due to dietetic imprudence (unsuitable, irritating, or decomposing food, or too much good food), or to the abuse of alcohol; also to gout, fevers, and anæmia. There is a recognised liability to "dyspepsia" in certain persons or families, and it occurs at all ages.

(*b*) *Symptoms.*—In *mild* cases, especially after taking food, there is headache and depression of spirits, followed by epigastric fulness, weight, and distress, and perhaps dull pain. Eructations, nausea, and vomiting ensue, and in children diarrhœa and spasmodic abdominal pain as well. The vomitus contains undigested food, mucus, and finally bile. The tongue is furred, the breath unpleasant, and the saliva increased. The attack lasts about 24 hours, usually being relieved by the vomiting.

In *severe* cases there may be a chill, fever (102° to 103°), severe headache, delirium (occasional in children), vomiting, diarrhœa (frequent), or constipation (seldom), with slight abdominal distention and epigastric tenderness. There is a bitter taste in the mouth, herpes on the lips (frequent), a heavily furred tongue, and bad breath. The urine is scanty, high coloured, and loaded with urates. An examination of the vomitus shows diminished or absent HCl, and the presence of lactic and fatty acids and a quantity of mucus. If the duodenum and bile ducts share in the catarrhal inflammation jaundice ensues. An erythematous rash may be present in children. The attack lasts from 2 to 4 days, perhaps longer.

(*c*) *Differential Diagnosis.*—The diagnosis of the mild form is usually easy, but the severer cases, with sudden onset, chill, and fever, may require observation for a day or two in order to discriminate them from the acute infectious diseases—viz., from abortive *typhoid fever* by the absence of prodromata, of bronchitis, of splenic enlargement, rose spots, and the characteristic temperature curve; from *meningitis*, which the cases with severe headache and delirium may suggest, by the history and subsequent course; from the crises of *locomotor ataxia* by the presence of the patellar reflexes and the absence of the Argyll-Robertson pupil; from *scarlet fever*, which the occasional erythema may simulate, by the absence of the sore throat, swollen cervical glands, extraordinarily rapid pulse, and typical strawberry tongue of the specific infection.

II. **Phlegmonous** (*Suppurative*) **Gastritis.**—(*a*) *Causes.*—Diffuse or circumscribed suppuration of the submucous coat of the stomach is extremely rare, and occurs in connection with pyæmia, puerperal fever, cancer of the stomach, peritonitis, or injury.

(*b*) *Symptoms.*—Fever, dry tongue, delirium, vomiting, diarrhœa, weak and rapid pulse, with epigastric soreness, abdominal pain, and meteorism, followed by coma, collapse, and death. In more chronic cases there are fever, recurring chills, and abdominal pain.

(*c*) *Diagnosis.*—Rarely if ever made *ante-mortem*. The circumscribed abscess has been felt externally. Even if a quantity of pus is vomited, owing to rupture of a large submucous abscess, it can not be separated from the perforation of an outside pus collection into the stomach.

III. **Toxic Gastritis.**—(*a*) *Causes.*—Ingestion of corrosive poisons.

(*b*) *Symptoms.*—Intense burning pain in mouth, throat, and stomach, severe epigastric pain, salivation, unremitting vomiting, intense thirst, and dysphagia. The vomitus contains blood, and perhaps fragments of mucous membrane. Later there are abdominal pain, tenderness, and distention, with diarrhœa. There may be petechiæ, hæmaturia, and albuminuria. In severe cases the symptoms of collapse (cold, wet skin, lowered temperature, small rapid pulse) may supervene.

(*c*) *Sequelæ.*—Cicatricial stricture of the esophagus, or an ultimately fatal chronic atrophy of the gastric mucosa.

(*d*) *Diagnosis.*—The history, the inspection of the mouth and tongue (page 243), and an examination of the vomitus will usually enable a diagnosis.

IV. **Diphtheritic Gastritis.**—The membranous form of gastritis may be due to the infection of diphtheria, but is usually secondary to pneumonia, pyæmia, variola, typhus and typhoid fever. It can not be recognised *ante-mortem.*

V. **Chronic Catarrhal Gastritis.**—(*a*) Causes.—Dietetic errors (very frequent) and the excessive use of alcohol (common). It may be secondary to diabetes, gout, nephritis, tuberculosis, anæmia, or chlorosis; or to ulcer, cancer, and dilatation of the stomach; or to chronic pulmonary disease, chronic heart disease, and cirrhosis of the liver, all of which cause a persistent passive congestion of the gastric mucosa. Pathologically two forms of the disease are recognised, a simple *catarrhal*, and a *sclerotic*, gastritis. The latter results in atrophy of the secretory mucous membrane, either with thinning of the walls of the stomach, or with thickening, contraction, and a diminution in size of the whole organ.

(*b*) **Symptoms.**—These are varied. Headache is common, vertigo not infrequent, disturbed or dreaming sleep, depression of spirits, drowsiness, and a feeling of languor are almost always present. The appetite is fickle, sometimes abnormally great. After eating there is epigastric oppression, fulness, distress, or burning pain (*heartburn*, when substernal), with tenderness. There may be frequent eructations of bitter fluid, belching of gas, and well-marked tympanitic distention of the abdomen. Vomiting after meals is not very common, but in chronic alcoholism "dry retching," or vomiting of watery mucus, or nausea alone, occurs in the morning with great frequency. Constipation is usual, but may alternate with diarrhœa. The tongue is often swollen and indented, its tip and margin red, there is a bad taste in the mouth, and salivation often occurs. The urine is frequently high-coloured, and throws down a heavy uratic deposit. Palpitation is not uncommon, and "stomach" cough due to a chronic catarrhal pharyngitis may be present.

The symptoms of atrophy of the mucous membrane, the final result of prolonged and severe simple gastritis, resemble, in the majority of cases, those of gastric cancer—i. e., pain, vomiting, and progressive loss of flesh and strength; less frequently the symptoms of pernicious anæmia, resulting from the destruction of the gastric mucosa, announce the causative condition.

According to the results of an examination of the stomach contents three forms of chronic gastritis are recognised, viz.:

Simple Gastritis.—After Ewald's meal, HCl diminished or absent, lactic and acetic acids present (the latter two absent after Boas's meal), pepsin and rennin always present; fasting stomach contains a little slimy fluid.

Mucous Gastritis.—As in simple gastritis, except that a large amount of mucus is found.

Atrophic Gastritis.—HCl, pepsin, and rennin absent; fasting stomach empty.

(*c*) **Diagnosis.**—The presence of chronic gastritis as an indirect symptom of chronic disease of the heart or lung, or of a cirrhotic liver should be borne in mind. The severe forms of chronic gastritis require to be differentiated from cancer (*q. v.*), and the distinction, in the absence of tumour, may be extremely difficult, unless the history or prolonged observation shows that the symptoms cover a longer period than the utmost duration of cancer. In the cirrhotic form of gastritis the contracted stomach may in rare cases form a palpable tumour.

VI. **Gastric** (*Peptic*) **Ulcer.**—(*a*) **Causes.**—Occurs at all ages, but is most common in women, especially between 20 and 30 years of age. Less frequently it is found in men, usually between 30 and 40 years of age. Is often associated with overwork, poor food, anæmia, chlorosis, and amenorrhœa, and is especially common in servant maids, and in those whose work involves pressure on the epigastrium (tailors, cobblers, weavers). Hepatic and cardiac disease and arteriosclerosis predispose.

(*b*) **Symptoms.**—The disease may be entirely latent and discovered only at autopsy. More commonly the initial symptoms are those of dyspepsia or chronic gastritis—viz., anorexia, epigastric fulness or oppression, eructations, and pyrosis. Eventually in well-marked cases symptoms more or less characteristic of ulcer supervene. These are:

Pain, which may be dull and oppressive. More significant is an attack of sharp, boring, or burning pain, excited by taking food, and occurring either immediately or from 1 to 2 hours after eating. In many cases there are two points at which the pain is most intense: anteriorly in the epigastrium, posteriorly at the level of the 10th dorsal vertebra. It is relieved if vomiting takes place. Such paroxysms of pain (gastralgia) may, however, be independent of the ingestion of food, and are often of indescribable severity. Pressure may either aggravate or relieve the pain of gastric ulcer. It is doubtful whether, as often alleged, the sooner the pain comes after eating the nearer is the ulcer to the cardia; so also with reference to the relief afforded by posture, an amelioration of the pain by lying on the face indicating that the ulcer is situated on the posterior wall of the stomach, and *per contra*.

Tenderness, which is a common symptom, but only of diagnostic value when strictly circumscribed in the epigastrium so that a finger tip almost covers it, or when a tender spot is found to the left of the 11th or 12th dorsal vertebræ.

Hæmatemesis, a most significant event when a considerable quantity of unaltered blood is vomited, but occurring in only 50 per cent of the cases. It may be slight (coffee-ground), but ordinarily is large, and sometimes so excessive as to be immediately fatal. Recurrent attacks at intervals of hours or days are not uncommon. After a gastric hemorrhage tarry blood is found in the stools, and may be the only evidence of the slighter bleedings. Syncope or convulsions may attend, or hemiplegia and severe anæmia follow, large hemorrhages.

Hyperchlorhydria, often with hypersecretion, is usually present. On palpation in old ulcers a distinct induration, resulting from inflammatory changes, may at times be felt near the pylorus. Anæmia is usually present and may be severe (1,000,000, or less, reds to the cu. mm.). Loss of flesh is customary.

The *course* of the disease is usually chronic, rarely acute, and recurrences (new ulcers or renewal of old ulceration) are not uncommon.

(*c*) **Complications and Sequelæ.**—Of these the most important is perforation, occurring in about 6 per cent of all cases. It may be the first evidence of hitherto latent ulcer. Perforation of the anterior wall may take place into the general peritoneal cavity, in which case the symptoms are those of an acute perforation peritonitis; ulcers of the upper and posterior walls may perforate and cause a subphrenic abscess, or, if the lesser peritoneal cavity, and subsequently the pleura, is entered, subphrenic pyopneumothorax. Rarely, by adhesion and ulceration, fistulous communications may be established with the duodenum, colon (most common), or the external surface in the epigastric region. Cicatricial stenosis of the pylorus, with resulting dilatation or hour-glass contraction of the stomach, may follow the healing of a large ulcer. Cancer may develop on the site of an old ulcer.

(*d*) **Differential Diagnosis.**—The cardinal symptoms are gastralgia and hæmatemesis, usually with dyspeptic symptoms, and in cases presenting this triad the diagnosis is readily made. If hæmatemesis does not occur it may be very difficult to arrive at a positive conclusion. The following conditions require discrimination:

(1) *Gastralgia.*—Gastralgia is not only associated with ulcer, but also constitutes an independent form or symptom of nervous dyspepsia, and with the exception of hemorrhage may exactly simulate gastric ulcer. The discrimination can not always be made. In gastralgia of the neurotic form, as contrasted with ulcer, the attacks occur more frequently when the stomach is empty, pressure and the taking of food afford relief, vomiting of blood is absent, hyperchlorhydria is not constant, circumscribed tenderness is absent, gastric dilatation and pyloric hardening (from healed ulcer) are never present, ordinarily there are no dyspeptic symptoms between the paroxysms, there is less emaciation and loss of strength, and, as a rule, symptoms of hysteria or neurasthenia are found, or there are neuralgias in other parts of the body.

(2) *Gastric Crises.*—The attacks of pain and vomiting which

occur in certain spinal-cord diseases, especially tabes, may simulate gastric ulcer, but the absent knee-jerks, lightning pains in the legs, and Argyll-Robertson pupil will announce the true character of the attacks.

(3) *Hepatic Colic.*—In this affection, swelling and tenderness of the liver, a palpable gall bladder, the abrupt onset, longer continuance and sudden cessation of the painful paroxysm, and perhaps the subsequent occurrence of jaundice, will prevent its confusion with gastric ulcer.

(4) *Cirrhosis of the Liver.*—The vomiting of blood which occurs in some cases of this malady may suggest ulcer, but the usual alcoholic history, the ascites, the hardened and palpable liver, will declare the true cause of the hæmatemesis.

(5) *Chronic Gastritis.*—Very rarely is there hæmatemesis, the pain is not severe, the tenderness is diffuse, vomiting is not so frequent and when it occurs is not so painful, and HCl is diminished or absent.

(6) *Cancer of the Stomach.*—As a rule this is not mistaken for ulcer. Comparing the two, in cancer there is often a tumour, lactic acid is usually present, and HCl is deficient or absent; blood when vomited is "coffee-ground" or mixed in small quantity with mucus; there is usually great emaciation with cachexia, and the subject is more frequently middle-aged, almost invariably past 30. If, however, there is cicatricial contraction about the pylorus, forming a palpable tumour, a differential diagnosis may be impossible, especially when it is borne in mind that cancer may develop upon the site of an old ulcer, and that in such cases there may be hyperchlorhydria as in ulcer, instead of the absence of HCl as is usual in cancer.

VII. **Duodenal Ulcer.**—In the vast majority of cases the symptoms of duodenal ulcer are identical with those of gastric ulcer. In favour of duodenal ulcer are pain in the right hypochondriac region 2 or 3 hours after meals, sudden and recurring intestinal hemorrhages (tarry or bright-red stools) sufficient to produce anæmia in an otherwise well person, occasional jaundice, and violent gastralgic attacks, often followed by melæna.

VIII. **Cancer of the Stomach.**—Next to the uterus the stomach is the most frequent seat of primary carcinoma. Rarely it is secondary to mammary cancer. Gastric cancer is rather more common (5 to 4) in men, and three fourths of the cases occur between 40 and 70 years of age, increasing with each decade. It is seated in the pylorus in 8 out of every 13 cases. Alcoholism, previous ulcer, worry, and a family history of cancer or tuberculosis predispose.

lung, pleura, kidneys, and spleen. Perforation may occur, usually but not always causing diffuse peritonitis; or a fistulous communication with the colon, or more rarely the small intestine, may be established.

Duration.—The average duration of gastric cancer is from 1 year to 18 months; more rarely life is prolonged for from 2½ to 3 years. It is always fatal.

Differential Diagnosis.—The *cardinal symptoms* are progressive emaciation, pain, tumour, coffee-ground vomiting, dilated stomach, constant absence of HCl, and presence of lactic acid after a Boas meal, in a person between 40 and 70 years of age.

In the absence of tumour such a set of symptoms will warrant a quite positive diagnosis of cancer. The disease should be suspected if, in elderly persons, the ordinary symptoms of chronic gastric catarrh obstinately persist and a cachexia out of proportion to the apparent cause develops, especially when hyaline casts are found in the urine. The cases of cancer which present severe anæmia or gastric symptoms, without tumour, may simulate very closely and be mistaken for pernicious anæmia or chronic gastritis. Ulcer of the stomach, with cicatricial thickening at the pylorus, may exactly resemble cancer. Enlargement of the supraclavicular or inguinal glands, or a nodule at or in the neighborhood of the navel, may be of great value as a clew to the existence of gastric cancer.

The following diseases may require to be differentiated. As Osler well says, "there are cases of cancer of the stomach in which a positive diagnosis can not be reached for weeks or months."

(1) *Chronic Gastritis.*—This, as contrasted with cancer, occurs at any age, pain and tenderness are less marked, vomiting is not common, hæmorrhage is rare, or when present consists simply of blood streaks (not coffee-grounds), there is no fever, loss of flesh is not marked, and there is no cachexia, no tumour, and no œdema. Moreover, its duration may be much longer than that of cancer, and, on the other hand, it is susceptible of cure. No lactic acid is found after a Boas meal.

(2) *Gastric Ulcer.*—As compared with cancer, ulcer of the stomach occurs most frequently in early adult age and in women; the pain is more apt to be gastralgic and to be excited by the taking of food, and may be entirely lacking for days or even weeks; tenderness is strictly circumscribed in the epigastrium, or posteriorly at the level of the 5th dorsal vertebra; the ordinary symptoms of indigestion may be slight; vomiting is by no means a frequent symptom; tumour (cicatricial) is very rare; there is no œdema; bleeding from the stomach is common, and as a rule characteristically profuse; there is no fever. Gastric ulcer may recover or continue for several

years; no lactic acid is found after a Boas meal, and there is usually an excess of HCl.

(3) *Gastric Ulcer with Cicatricial Pyloric Thickening and Stenosis.*—This condition, which, because of the presence of tumour and dilatation of the stomach, exactly resembles cancer of the pylorus, may perhaps be differentiated from the latter by the comparative youth of the patient, the history, the abundant hæmatemesis, the hyperchlorhydria, and the longer duration.

(4) *Pernicious Anæmia.*—In this disease, as compared with gastric cancer, the number of reds is very frequently below 2,000,000 to the cu. mm.; indeed, if 1,000,000 or below, it points strongly to anæmia rather than cancer (OSLER); megaloblasts are present, leucocytosis is less frequent, the colour index is higher, and there is rarely the loss of flesh that occurs in malignant tumour of the stomach.

(5) *Cancer of the Pancreas.*—In this disease when a tumour is palpable it is immovable, jaundice often exists, dilatation of the stomach and coffee-ground vomiting are absent, HCl is present, fat may be found in the stools, and lipuria and glycosuria may exist.

(6) *Occasional Causes of Error.*—Cancer of the transverse colon lacks coffee-ground vomiting and anachlorhydria, and later often presents symptoms of intestinal obstruction. In duodenal cancer HCl is usually absent, but jaundice may be present. Impacted fæces in the colon disappear by treatment. Movable kidney is recognised by its shape and the practicability of reduction. Cancer of the liver or gall bladder lacks the gastric pain and frequent coffee-ground vomiting of gastric cancer. Tumour of the abdominal wall is unattended by gastric disorders. Tumours of the omentum are nodular and irregular, and subsequently lead to peritoneal effusion. The rare and remarkable tumour-like mass of swallowed hair in hysterical women is a clinical curiosity, and is usually recognised by operation, but the history may give rise to a suspicion.

IX. **Hypertrophic Stenosis of the Pylorus.**—Non-malignant thickening of the pylorus, due to hypertrophy of the muscular and submucous tissues, may occur in adults, and, as a congenital condition, in infants of the average age of 5 months.

The *symptoms* are those of a dilated stomach.

X. **Dilatation of the Stomach.**—Causes.—In rare instances gastrectasia is acute and due to overstretching, as in the taking of huge quantities of food and drink, or sudden weakness of the muscular coats as in shock, or rapid degenerative processes as in the specific fevers. Chronic gastrectasia, according to its origin, may be divided into two classes, the *stenotic* and the *atonic*.

The first class is due to pyloric or duodenal stenosis, either an actual narrowing caused by cancer, cicatricial contraction from ulcer or corrosive poison, or simple hypertrophic stenosis; or narrowing by outside pressure from cancer of the liver, gall bladder, pancreas, and omentum, or by large gallstones; or a floating right kidney, or kinking of the pylorus from adhesions to the gall bladder and liver.

The second class is due to atony of the muscular coats incident to chronic gastritis, the degenerative changes of anæmia, tuberculosis and like diseases, habitual overdistention of the organ as in beer-drinkers and diabetics, congenital weakness, and deficient innervation.

Symptoms.—Epigastric fulness or distress, flatulence, eructations, and vomiting. There may be anorexia, but not uncommonly the appetite is particularly good and there is great thirst. The skin is dry, constipation is present, and the urine is scanty. Great loss of flesh, anæmia, and debility commonly ensue. There may be cramps of the calf muscles, or occasionally tetany.

The characteristic symptoms are the manner of vomiting and the composition of the vomitus. The stomach empties itself at intervals of several days, ejecting large quantities (1 to 3 gallons) of stagnant fluid and remnants of partially digested or undigested food. The vomitus is acid from the presence of lactic, butyric, and acetic acids. Hydrochloric acid may be normal, diminished, absent, or increased. Offensive odours may be due to the presence of sulphuretted or phosphuretted hydrogen. The fluid contains the *Sarcina ventriculi*, yeast fungus, and many bacteria. On standing, it separates into a sediment of undigested food, and a supernatant grayish turbid fluid capped by a brownish froth.

The *physical signs* are, in brief, as follows: The outline of the distended organ (Fig. 173, page 491), together with its peristaltic waves, may often be seen; if not, it is to be inflated. Pyloric tumour may be felt, and the peculiar resiliency of the dilated stomach and its peristalsis may be perceived by palpation. Splashing or succussion sounds may be elicited, either by the hand of the examiner or upon deep and rapid breathing by the patient. Percussion after drinking water may enable the lower border of the organ to be recognised. Auscultatory percussion is of much service in determining the outlines of the stomach.

Differential Diagnosis.—The cardinal symptoms are the characteristic vomiting and the results of a physical examination of the stomach, especially inspection of the inflated organ or auscultatory percussion. The finding of a pyloric tumour will decide against the atonic nature of the dilatation, and in the majority of cases the tumour is due to cancer of the stomach (page 849).

Gastrectasia is to be distinguished from gastroptosis or prolapse of the stomach (page 492). An unusually large stomach (megalogastria) may be found, but unless evidence of stagnation of the gastric contents—i. e., the finding of a quantity of food or fluid in the stomach 7 hours after a full meal, or periodic vomiting of large amounts—is present, the condition can not be considered abnormal.

Experience teaches proper humility, but it is difficult to conceive of confounding ascites or ovarian tumour with a dilated stomach, yet both these errors have occurred.

.**Prognosis.**—This depends upon the cause. Atonic cases often recover; cancerous stenosis is, of course, fatal; simple stenosis may be greatly benefited. Modern surgery (by gastro-enterostomy, gastroplication, or stretching of the pylorus) may afford material help.

XI. **Hæmatemesis.**—See page 132.

XII. **Neuroses of the Stomach.**—These consist of various more or less serious functional disturbances of the stomach—i. e., without discoverable local lesions. Rarely these disturbances arise reflexly from organic disease in other parts or organs of the body. Almost invariably they form a part of the protean manifestations of a congenital neurotic diathesis, or of an acquired neurasthenia or hysteria. In a certain proportion of cases the neuropathic basis of the dyspeptic symptoms may elude recognition without careful inquiry and prolonged observation.

The term *nervous dyspepsia* is in reality a generic term covering the various gastric neuroses—sensory, motor, or secretory—about to be described. The individual patient rarely suffers from a single one of these separate disturbances. Usually two or more, in varying or complex combinations, are found to be associated. Leube, however, limits the meaning of the term to cases in which several neuroses, mainly sensory and of a mild grade, coexist, and are manifested only during the act of digestion; while any one of the neuroses, if severe and extreme and perhaps occurring alone, is considered as a separate and distinct disease—e. g., nervous vomiting; or hyperchlorhydria, either periodic or continuous. It is best, therefore, to describe first the ordinary form of nervous dyspepsia, and subsequently the special gastric neuroses. Investigation will show that a very large proportion of the cases of so-called nervous dyspepsia are in reality due to local disease, which is demonstrable by sufficiently careful examination.

A. NERVOUS DYSPEPSIA.—(*a*) *Symptoms.*—There is anorexia, less frequently bulimia. After eating there is a sense of epigastric distress and oppression, eructations, belching, pyrosis, acidity of the mouth, rumbling of gas, and sometimes nausea or vomiting. There may be headache, vertigo, cephalic pressure-sensations, numbness, cold hands and feet, bad taste in the mouth, and palpitation. Constipation may be present. As a rule there are marked depression of spirits, anxious forebodings, and perhaps a well-defined hypochondriacal condition. In spite of these symptoms the stomach is found to be empty 6 or 7 hours after a full meal, showing that the food is digested and that there is no stagnation. The gastric juice is usually of normal composition, although the HCl may be either increased or diminished, varying, often in the same person, from time to time.

(*b*) *Diagnosis.*—As the symptoms of nervous dyspepsia closely resemble those of chronic gastric catarrh it is necessary to distinguish between them. Contrasting the two, in nervous dyspepsia relief may follow the taking of food, there is less epigastric tenderness, and the symptoms may disappear at irregular intervals. The stomach contents usually contain little or no mucus or undigested food, and as a rule (with a number of exceptions) the HCl is in normal amount. The tongue is not so apt to be flabby, indented, and furred as in gastric catarrh. In certain cases the prominence of gastralgia or the presence of emaciation may suggest ulcer or cancer. After all, it is upon the existence of a neuropathic diathesis or temperament, to be discovered by careful and often necessarily prolonged observation of the patient and his neurotic peculiarities, that the diagnosis of the nervous character of the dyspepsia depends. Moreover, there are no obvious organic changes.

B. SECRETORY NEUROSES.—(I) **Hyperchlorhydria.**—The secretion of gastric juice containing an undue amount of hydrochloric acid (over 70 degrees) *only during digestion* is encountered mainly in adults, but is common in young chlorotic women. Worry, grief, and mental overwork are important antecedent causes. The liberal use of condiments and alcoholic beverages will predispose.

Symptoms.—Ordinarily the onset is gradual. There is at first simply a feeling of discomfort about 2 or 3 hours after a meal, but in course of time it develops into a burning pain, with weight and pressure, in the epigastrium, often with acid eructations, regurgitation, and pyrosis, sometimes followed by nausea and vomiting. Severe headache and attacks of vertigo are common, the urine may be scanty, and constipation exist. Loss of weight does not usually occur. The pain lasts for from 1 to 3 hours, and is relieved by vomiting, or more characteristically by taking albuminous food (milk, meat, white of egg) or alkalies (soda, magnesia). The physical examination may reveal a moderate diffuse epigastric tenderness, and occasionally evidence of slight gastrectasia. The examination of the stomach contents 1 hour after eating Ewald's meal shows an excess of HCl; so also 3 or 4 hours after a Leube-Rigel meal, while in the latter the meat is quite digested and the starches practically unchanged.

Hyperchlorhydria may be, and at first usually is, remittent, occurring daily for several days or many weeks, and then, having ceased for a variable period, returning with or without obvious causes, such as grief or mental worry or overwork. Finally, it may become a continuous condition. Ordinarily the prognosis is favourable.

Differential Diagnosis.—The cardinal symptoms are burning pain, occurring 2 to 3 hours after eating, and immediately relieved by alkalies or nitrogenous food, no marked emaciation or anæmia, and (the only positive evidence) a constant excess of HCl 1 hour after Ewald's meal. It is necessary to exclude the following diseases:

(1) *Gallstone Colic.*—In this the pain is felt in the right hypochondriac and the corresponding half of the epigastric regions, rather than in the middle of the latter; it occurs 4 or 5 hours after a meal, and is not promptly relieved by alkalies or food. Confusion can arise only in the absence of jaundice or a swollen gall bladder.

(2) *Gastric Ulcer.*—This condition is accompanied by hyperchlorhydria, but in addition to the other symptoms of gastric ulcer (page 829) the pain is not completely abolished by alkalies, and is aggravated by albuminous food.

(3) *Chronic Hypersecretion.*—In this, vomiting is more common, the paroxysms of pain usually occur during the night, and from the *fasting* stomach large amounts of gastric juice may be obtained.

(II) **Hypersecretion** (*Gastrosuccorrhœa*).—An excessive secretion of hyperchlorhydric gastric juice in the *fasting* stomach. Two forms are recognised, the periodic or intermittent (gastroxynsis, ROSSBACH), and the continuous or chronic (REICHMANN).

(*a*) *Periodic Hypersecretion.*—Into a state of health a sensation of epigastric uneasiness intrudes, deepening into pain, and shortly followed by nausea and the vomiting of a large quantity of very acid gastric juice, which ultimately may be greenish with bile. The nausea and retching are persistent, and at intervals of a few hours other large quantities of gastric juice are ejected. The throat may become raw and sore. The paroxysms often occur in the night or in the early morning hours. The attack as a whole lasts from 1 to 3 days, terminating with characteristic abruptness, but tending to recur at intervals varying from a few weeks to a year or longer. When recurrences tread one upon the heels of the other the condition merges into the continuous or chronic form. The pain may be severe, intense headache may be present, and pallor, coldness of the extremities, and constipation are common. Before this disease can be considered a pure neurosis it is necessary to exclude gastric ulcer (page 847) and the gastric crisis of locomotor ataxia (*q. v.*).

(*b*) *Continuous Hypersecretion.*—The early symptoms are those of hyperchlorhydria (page 855) or periodic hypersecretion. Epigastric pain becomes habitual after meals, and vomiting of an acid fluid, at first occasional, occurs once or more daily, most commonly after breakfast, rarely at night. From the *fasting* stomach an abnormally large amount of acid gastric juice free from fragments of food may be obtained. The prognosis is not bad.

As a pure neurosis this is rare. There is evidence to prove that it is due in the majority of cases, if not all, to gastric ulcer (page 847). It may be associated with pyloric stenosis and gastrectasia (page 852).

(III) **Hypochlorhydria.**—Diminution or absence of HCl is common in gastric cancer and in gastritis, especially of the atrophic form. As a not infrequent neurosis it may occur in hysteria, neurasthenia, and locomotor ataxia. Very rarely as a neurosis there is a total absence not only of HCl, but also of the ferments (achylia gastrica, EINHORN); but this is more commonly due to organic changes, particularly in atrophic gastritis. The *symptoms* of achylia of nervous origin may be negative; ordinarily they resemble those of chronic gastritis. The *diagnosis* is to be made by the result of the examination of the contents of the stomach. The total acidity is 4, HCl and ferments absent, mucus absent, and lactic acid absent except in traces. This condition is to be discriminated from the atrophic form of gastritis (page 846) and cancer of the stomach (page 849).

C. SENSORY NEUROSES.—(I) **Disturbances of the Hunger-sense.**—Elsewhere (page 122) have been considered, in general, bulimia, polyphagia, akoria, anorexia, and pica. Two special forms are the following:

(1) *Paroxysmal Bulimia.*—Sudden attacks of burning epigastric pain, faintness, headache, and excessive hunger, often occurring in the night, although the paroxysm may come on directly after a hearty meal. It is relieved by taking food. Frequent attacks may produce gastritis and dilatation. This condition is seen in hysteria, neurasthenia, exophthalmic goitre, migraine, epilepsy, and cerebral tumours.

(2) *Anorexia Nervosa.*—An absolute absence of appetite, which may be so extreme that the sight of food excites spasm. This condition is a manifestation of hysteria, and is commonly seen in girls between 15 and 20 years of age, more rarely as early as the 12th year. The patient is restless, but ultimately takes to bed; the emaciation may reach the last degree; the skin becomes dry and brawny, and contractures of the lower extremities may develop. Death has

been known to follow, but under proper treatment the prognosis is not unfavourable.

(II) **Gastric Hyperæsthesia.**—The symptoms are a sense of pressure, burning, fullness or weight, or gnawing pain, with tenderness in the epigastrium during the act of digestion. As these sensations may occur in organic disease of the stomach it is necessary to exclude gastritis (page 845) and gastric ulcer (page 847) by the absence of certain more or less distinctive symptoms, and by finding the gastric juice to be of normal composition and that the food is digested within the proper period.

(III) **Gastralgia.**—Paroxysmal gastric pain, which may be purely a neurosis, but occurs also, as a symptom, in cancer or ulcer of the stomach, or in locomotor ataxia (gastric crises).

(*a*) *Symptoms.*—The pain generally bears no relation to the taking of food. The attack is frequently preceded by slight nausea or epigastric pressure, salivation, faintness, vertigo, or headache. Shortly a severe and agonizing pain begins in the epigastrium, radiating through to the back and along the costal margins, especially toward the left, extending in some cases to the scapulæ and entire abdomen. The face is pale and anxious, the hands and feet are cold, and the skin cool and wet. The body is curved forward and the abdomen hollowed. There is slight epigastric tenderness, but pressure with the flat hand is often grateful. The attack may last from a few minutes to several hours. Recurrences may take place at intervals varying from a day to a year or more.

(*b*) *Differential Diagnosis.*—Before gastralgia is definitely decided to be of purely neurotic character it is necessary to exclude it as a symptom of organic nervous or gastric disease, and to separate it from other somewhat similar painful conditions.

As a *neurosis* it occurs mainly in women with menstrual irregularities, especially at the menopause; is most common in brunettes, and is favoured by worry, anæmia, and constipation. Healthy men are not exempt. It may be almost the only symptom of a neurasthenia (*q. v.*) or hysteria (*q. v.*), but more commonly is simply one manifestation of either of these conditions. The direct diagnosis of neurotic gastralgia is based principally upon the presence of neurasthenic or hysterical symptoms, together with the absence of signs of any causative organic lesion of the stomach or nervous system.

Without here going into the details of the different conditions which may be attended by or simulate gastralgic attacks, the following list will serve to point out the affections from which neurotic gastralgia must be discriminated by a careful consideration of the associated signs and symptoms, viz., cancer of the stomach, ulcer of the stomach, pyloric stenosis, the gastric crises of locomotor ataxia, hyperchlorhydria or hypersecretion, achylia gastrica, gallstone colic, renal colic, and flatulent colic.

D. MOTOR NEUROSES.—(I) **Peristaltic Unrest.**—This is seen especially after eating. The peristaltic movements are hyperactive, causing loud borborygmi, gurgling, and splashing, which may be audible at a distance. Emotion intensifies their activity. The hyperactivity may extend to the intestines. Peristaltic unrest is a frequent symptom of hysteria or neurasthenia. This mortifying condition has led to prolonged periods of seclusion.

(II) **Nervous Vomiting.**—This may occur either in children or adults, usually in hysterical women. Without preliminary nausea and without straining, the contents of the stomach are partly vomited, partly regurgitated. It usually, but not always, takes place shortly after eating. The attacks come on at irregular intervals. As a rule the general health is unimpaired, a point in favour of its hysterical origin in the absence of other neurotic manifestations. Primary periodical vomiting (LEYDEN) may occur as a neurosis in otherwise perfectly healthy persons, especially in women while menstruating.

(III) **Nervous Eructations.**—Loud belchings of air which has been swallowed or aspirated into the esophagus or stomach, either continuing from hours to days, or occurring in paroxysms which are excited by emotion. They may occur in hysterical women and children or in neurasthenic persons. In the young the attacks may be contagious by imitation. Anxiety, palpitation, epigastric fulness and distress may attend the paroxysms.

(IV) **Rumination or Merycism.**—In this infrequent condition the swallowed food is returned to the mouth and again chewed and swallowed. It occurs in hysterical, neurasthenic, or epileptic persons, and in idiots. It is sometimes hereditary.

(V) **Spasm of the Stomach.**—The spasmodic contraction may affect either the cardiac or the pyloric extremity of the stomach. If both are simultaneously present and the stomach is full of gas (*pneumatosis*), the upper abdomen may be enormously distended, the patient being unable to belch up the confined gas. It occurs in neurotic young persons.

(1) *Spasm of the Cardia.*—This may be painful, and is associated with a sensation as of a low-down obstruction opposite the lower end of the sternum. It is not a common condition, but may occur in hysteria, neurasthenia, tetanus, or with the rapid taking of very hot or cold food.

(2) *Spasm of the Pylorus.*—The symptoms are pain, perhaps a sense of resistance over the site of the pylorus, and increased gastric

peristalsis. It may be due to the irritation of highly spiced food, or of a hyperchlorhydria. If the latter is associated with long-continued hypersecretion, dilatation of the stomach may ensue.

(VI) **Relaxation of the Stomach.**—(1) *Atony of the Stomach or Atonic Dyspepsia.*—This is most commonly a symptom of chronic gastritis or general debility, but may infrequently occur as a neurosis. Organic disease must be excluded. The symptoms are epigastric discomfort, pressure, weight or distention, often with eructations. The physical signs are those of a moderate dilatation. The motility is impaired, as evidenced by the presence of food 3 hours after Ewald's, 7 hours after Leube-Rigel's, meal.

(2) *Pyloric Incompetency.*—Insufficiency of the pylorus, whereby the contents of the stomach are allowed to pass prematurely into the duodenum, or the contents of the latter to regurgitate into the stomach. It is recognised upon inflation of the stomach, when, if this condition exists, there is an immediate and visible passage of gas into the intestine.

E. GENERAL DIAGNOSIS OF GASTRIC NEUROSES.—In general the diagnosis of the purely neurotic character of the gastric disturbances just described is dependent upon two points: first, the exclusion of organic disease; second, and most important, the recognition of an abnormal weakness or excitability of the nervous system, as evidenced by the coexistence of one or more of the manifold symptoms of hysteria or neurasthenia (congenital or acquired), the characteristic exaggeration in the manner of describing the condition, and the precipitation of an attack or an exacerbation by worry, anxiety, and mental overwork or strong emotions.

VIII. DISEASES OF THE INTESTINES

(See also pages 123 to 155, 459 to 479, and 492 to 497.)

I. **Splanchnoptosis.**—"Glénard's disease," a general term applied to falling or dropping of certain abdominal viscera, embracing prolapse of the stomach (*gastroptosis*), of the kidney (*nephroptosis*), of the intestines (*enteroptosis*), and more rarely of the spleen (*splenoptosis*) and of the liver (*hepatoptosis*).

(*a*) *Causes.*—This condition is due to looseness of the attachments of the organs involved, to overstretching and consequent relaxation of the abdominal wall by repeated pregnancies or ascites, to tight lacing, and to a rapid loss of flesh.

(*b*) *Symptoms.*—In many instances one or more pronounced ptoses may be found, especially in multiparæ, without any symptoms whatever. With other cases varying symptoms are associated. There may e excessive flatulence, constipation alternating with diarrhœa, ucous "colic" or neurosis (the so-called membranous enteritis), astric disturbances or neuroses, throbbing of the abdominal aorta; ragging, aching, or weakness in the back; vasomotor instability or taxia; chronic tire, and other of the diverse symptoms of neurashenia or hysteria. The cases of abdominal ptosis with symptoms ccur more frequently in thin young women, especially those who ave rapidly lost flesh from any cause or undergone a long and xhausting nervous strain.

(*c*) *Diagnosis.*—The existence of the symptoms just rehearsed *lus* the finding of the prolapsed abdominal viscera makes the diagosis. Elsewhere will be found descriptions of the physical examnation required to determine the presence of displacement of the tomach (page 492), kidney (page 514), spleen (page 511), and liver page 504). Of the intestines the transverse colon is usually involved *coloptosis*). It sinks so as to form a V with acute bending or obtructive kinking at the hepatic and splenic flexures (Fig. 176, page 95). Glénard says that it may be palpated as a cordlike body at or below the level of the umbilicus, although others claim that the body felt is the pancreas uncovered by the prolapse of the stomach.

II. **Acute Catarrhal Enteritis.**—**Causes.**—Intestinal catarrh may be *primary*, and excited by hot weather, sudden falls of temperature, improper or decomposing food or spoiled milk, and irritant poisons; or *secondary* to infectious diseases, cachectic states, portal engorgement (disease of heart, lungs, liver), or by extension from inflammatory processes in the abdomen.

Symptoms.—The cardinal symptom is diarrhœa. In addition there may be colicky abdominal pain, moderate gaseous distention of the abdomen with rumbling and gurgling noises, and occasionally vomiting. Fever, if present at all, is slight (99.5° to 100.5°); there is a furred tongue, with thirst, anorexia, and scanty urine. These symptoms indicate the condition most commonly encountered, namely, an inflammation of both small and large intestine, or *ileo-colitis*.

In some cases the clinical symptoms are sufficiently distinctive to allow an inference that the chief seat of the inflammation is in certain definite portions of the intestine, viz., the duodenum, the small intestine, the colon, or the rectum.

(1) *Duodenum.*—If the duodenum alone is affected (duodenitis) there is constipation instead of diarrhœa, with some localized discomfort or slight pain and tenderness, but these symptoms are not at all definite. Usually gastritis coexists, with nausea, vomiting, and gastric pain (gastro-duodenitis), but the vague duodenal symptoms are obscured by those relating to the stomach. In a certain

proportion of cases, however, jaundice, due to involvement of the bile duct, appears, constituting the only positive proof of duodenitis. Otherwise the duodenal inflammation is clinically unrecognisable.

(2) *Small Intestine.*—That the small intestine (jejunum, ileum) is bearing the brunt of the inflammation and that the colon has escaped may be inferred by the absence of a marked diarrhœa and the presence of colicky pain and rumbling, with slight distention and tenderness over the area in which lies the bulk of the small intestines. The stools are flocculent, contain undigested food, unchanged bile, and small, scarcely visible masses of mucus—findings which are quite characteristic of a small-intestine catarrh.

(3) *Colon.*—Considerable pain, a marked diarrhœa, with tenderness along the course of the colon, and souplike stools containing mucus, perhaps in large and easily seen masses, indicate that the affection is in the main a colitis.

(4) *Rectum.*—Considerable quantities of mucus and pus, passed with painful tenesmus, indicate proctitis.

The duration of acute catarrhal enteritis varies from 3 days in mild cases to 7 or 10 days in severe cases. Prognosis usually good.

Diagnosis.—The symptoms and short duration of the disease generally enable a ready recognition of its character. Diarrhœa is the important symptom.

There is rarely any difficulty in distinguishing it from *typhoid fever* by its brief duration, slight and atypical fever, and absence of splenic swelling and rose spots. Occasionally one meets severe cases, which, because of unusually severe pain and marked tenderness, strongly suggest *peritonitis*, but the presence of diarrhœa in particular, and the absence of a high degree of meteorism and abdominal rigidity, will usually suffice to rule out peritoneal inflammation. Nevertheless in some cases it is impossible to resist the conviction that the peritoneum is sufficiently irritated and congested to cause "peritoneal" symptoms. During an epidemic of *cholera Asiatica* all diarrhœal cases are to be regarded and treated as cholerine (page 784) or possible beginning cases of the full-fledged disease. For the separation of diarrhœa from dysentery, see page 783.

III. **Chronic Catarrhal Enteritis.**—(*a*) *Causes.*—It may be a sequel of repeated acute attacks of enteritis or dysentery, or develop as a consequence of obstruction to the portal circulation by hepatic cirrhosis or chronic disease of the heart or lungs, or arise independently.

(*b*) *Symptoms.*—Mainly a chronic diarrhœa, which may alternate with constipation, particularly if the colon alone is affected. Colicky pain and abdominal tenderness are infrequent. The stools may be lienteric, containing undigested food, when the small intestine is chiefly involved, or fluid and mucous if the main seat of the disease is in the colon. In course of time anæmia and emaciation become manifest, and mental depression or hypochondriasis may develop. *Prognosis* is usually good as to life, but recovery is not common.

IV. **Enteritis in Infancy and Childhood.**—The greatest number of cases are found during the hot months, from May to September inclusive, in infants from 6 to 18 months of age, and with few exceptions in those who are fed artificially. Many varieties of bacteria are found in the diarrhœas of children, but no one form can be held responsible for the toxic products which develop in the intestine as the result of bacterial growth. Adopting Osler's classification, the following varieties may be recognised :

(I) **Acute Dyspeptic Diarrhœa.**—(*a*) *Symptoms.*—After drinking spoiled milk or eating unripe fruit or improper food the child becomes ill. The symptoms may develop slowly, with restlessness and perhaps slight fever, followed by diarrhœa with offensive, sometimes greenish, stools, varying from 4 to 20 in 24 hours, which contain milk curds, undigested food, and occasionally mucus. In the severer forms the onset is sudden, sometimes attended by an initial convulsion, with vomiting, thirst, scanty urine, colicky pain, abdominal tenderness, and a rapid rise of temperature to 104° or 105°.

(*b*) *Diagnosis.*—The small quantity of mucus in the stools will rule out entero-colitis. The serous stools and severe symptoms of cholera infantum are absent.

(*c*) *Duration and Prognosis.*—In well-nourished children under good hygienic surroundings and with proper care recovery occurs in a few days. Otherwise it may merge into an entero-colitis, or, in very weakly children, prove fatal.

(II) **Cholera Infantum.**—This occurs in but 2 or 3 per cent of the hot-weather diarrhœas (HOLT). It is generally preceded by some intestinal disturbance.

(*a*) *Symptoms.*—The disease usually begins with vomiting, which becomes excessive and persistent, at first containing bile, later becoming serous. At the same time purging sets in, with copious and frequent stools varying from 10 to 30 in 24 hours. Primarily the stools are offensive, fluid, and fæcal, but ultimately become serous, watery, odourless, and alkaline, and are voided with force. The temperature by rectum is 105° to 108°, by axilla 3 or more degrees lower. The tongue becomes red and dry, there is intense thirst, great restlessness, rapid, weak pulse, and scanty or absent urine. The prostration, which is present from the beginning, becomes extreme, the face is ashy, pallid, and pinched, the eyes are sunken and the eyelids but partly closed, the mouth is open, the fontanel depressed, and the extremities cold. The skin loses its elasticity, and if pinched remains in folds. Death may ensue within 24 hours with hyperpyrexia or collapse symptoms; or after a few days gradual improvement may take place, or the disease pass into an entero-colitis; or the symptoms of spurious hydrocephalus, the hydrencephaloid state, may supervene. In the latter there is an apathetic or semicomatose condition, with irregular or Cheyne-Stokes respiration, cervical retraction, clinching of the hands, sunken abdomen, and perhaps convulsions. Autopsies usually fail to reveal any organic changes in the brain.

(*b*) *Diagnosis.*—The cardinal symptoms are the incessant vomiting, the uncontrollable diarrhœa, the serous stools, and the collapse symptoms. This clinical picture renders a mistake in diagnosis almost impossible.

(*c*) *Duration and Prognosis.*—Death, convalescence, or a change to a less acute form of intestinal inflammation usually occurs in from 1 to 4 days. The prognosis in the majority of cases is unfavourable.

(III) **Acute Entero-colitis.**—Follicular or catarrhal ulceration, especially of the ileum and colon. It occurs during hot weather, or may follow dyspeptic diarrhœa, cholera infantum, or the specific fevers. See also Acute Specific Dysentery, page 780.

(*a*) *Symptoms.*—Usually with preliminary diarrhœal symptoms the temperature rises, perhaps to 104°, and the stools, which are passed without pain and are rarely offensive, become blood-streaked, contain much mucus, and vary from 15 to 30 in 24 hours. There is abdominal pain and distention, with tenderness along the course of the colon. Vomiting is not a prominent symptom.

(*b*) *Duration and Course.*—The severity and course of the disease are variable. After lasting for 2 or 3 weeks convalescence may begin; or the symptoms may moderate and the fever cease, but the diarrhœa continues and the child wastes away, until at the end of 5 or 6 weeks recovery begins or death occurs. In certain instances the disease may have a sudden onset with high fever, intense abdominal pain, vomiting, diarrhœa with frequent stools containing much blood and mucus, prostration, collapse, and death, within from 2 to 7 days.

(*c*) *Diagnosis.*—This disease presents higher fever, a more severe type of symptoms, and much larger quantities of mucus in the stools than the simple dyspeptic diarrhœa, as well as a more protracted course.

V. **The Cœliac Affection.**—This disease of children from 1 to 5 years old—of unknown origin and pathology except that in some cases the *Filaria sanguinis* has been found in the dejections—resembles the "hill diarrhœa" of the tropics affecting adults. The stools are large, gruel-like, frothy, fermenting, and offensive. Anæmia, debility, and wasting of the body gradually occur. The disease is protracted, with many relapses, and usually terminates fatally.

VI. **Phlegmonous Enteritis.**—A rare diffuse or circumscribed suppurative inflammation of the submucosa, occurring in connection with chronic intestinal obstruction, strangulated hernia, and intussusception; or, usually affecting the duodenum, as a complication of the severer types of the specific infections or pyæmia. The *symptoms* resemble those of peritonitis, with severe, sometimes fæcal, vomiting, high temperature perhaps with chills, intense pain, and tenesmus, the latter especially in cases of obstruction. A diagnosis is impossible, but the condition may be suspected when such symptoms occur in connection with intussusception or strangulated hernia.

VII. **Diphtheritic (Croupous) Enteritis.**—A membranous inflammation of the mucosa of the small intestine and colon may occur, most commonly as a result of pneumonia, scarlet fever, typhoid fever, and pyæmia; or from poisoning by arsenic, mercury, and lead; or as a terminal event in various cachexiæ, especially chronic nephritis, malignant disease, and hepatic cirrhosis. The disease is often latent and clinically unrecognisable. There may be in some, especially the toxic, cases, pain, diarrhœa, blood-stained mucous stools, and, more rarely, tenesmus, but these symptoms are not very distinctive.

VIII. **Ulceration of the Intestine.**—Ulcers in the intestine occur in dysentery, typhoid fever, chronic enteritis, tuberculosis, syphilis, and cancer; other ulcers are the duodenal, stercoral (due to the pressure of hardened fæcal masses), the solitary ulcer of cæcum or colon, the embolic ulcer (due to embolism of an intestinal artery in the course of valvular disease or pyæmic processes), and simple ulcerative colitis. Furthermore, ulceration and perforation of the bowel from without inward may result from the pressure and erosion of outside neoplasms, or more commonly from the localized abscesses of appendicitis or suppurative or gangrenous pancreatitis.

The symptoms of intestinal ulceration, apart from those of the causative condition, are not very distinctive. In general, intestinal hemorrhage, especially if profuse, manifested by bloody or tarry stools; the presence of pus in *moderate* quantity, large amounts indicating rather the perforation of a neighbouring abscess cavity (usually pericæcal or from the broad ligament); and the finding of tissue shreds or particles, constitute the most significant symptoms. Diarrhœa, colicky pain, and finally perforation followed by peritonitis may occur. All of these symptoms are most marked when the colon is involved, as ulcers of the small intestine tend to latency. The following forms require special but brief mention:

(1) *Simple Ulcerative Colitis.*—The ulceration of the colon may be very extensive. The disease is not infrequent, and usually occurs in men past middle life. The *symptoms* are a lienteric diarrhœa, often alternating with constipation, a protracted course with weakness and loss of flesh, and possibly perforation. The disease usually becomes chronic. The *differential diagnosis* is to be made from dysentery, to which it bears a close resemblance, by the presence of undigested food in the stools and the absence of blood and pus.

(2) *Stercoral Ulcers.*—In very chronic cases of constipation, small, hard, rounded scybalæ, perhaps covered by a deposit of lime salts (enteroliths), may be retained for so long a time as to cause distinct ulcers in the saccules of the colon in which they lie. There is often tenesmus, colicky pain which may occur in severe paroxysms, diarrhœa from the irritation of the hardened masses which nevertheless fail to be dislodged, and the stools may contain mucus, pus, and perhaps blood. The diagnosis is not easy, but the condition should be borne in mind as a possible explanation of the symptoms. The rectum should be digitally examined for the presence of hard scybalæ, and the abdomen palpated to discover, if possible, a cylindrical fæcal mass, or the circumscribed tenderness of an ulcer in the colon, both of which are rare findings.

IX. **Appendicitis.**—Pathologically there are three forms of appendicitis: *obliterative*, in which the appendix may become occluded at its proximal end and dilate into a cyst, or its lumen becomes entirely obliterated, or ulceration and perforation occur, or the process may terminate in resolution; *ulcerative*, in which the ulceration may heal with or without the formation of a stricture, or may perforate; and *interstitial*, in which gangrene, usually limited, occurs, followed by perforation and peritonitis. Fæcal concretions often, and foreign bodies more rarely, play an important part in initiating ulcerative or necrotic changes. The micro-organisms which are associated with the infective inflammatory, ulcerative, or necrotic processes are the *Bacterium coli communis* (most common), *Streptococcus pyogenes* (most virulent), *Staphylococcus pyogenes aureus*, *Bacillus pyocyaneus*, *proteus*, *Bacillus typhosus*, *Bacillus tuberculosis*, and *Actinomyces.*

The majority of cases occur between the 15th and 30th year of life, although no age is exempt, and about four fifths of the cases are in males. Important predisposing causes are blows, falls, excessive physical exertion, fatigue, improper food and digestive disturbances, fæcal concretions or foreign bodies, exposure to cold, rheumatism, influenza, and typhoid fever.

According to the intensity, frequency of occurrence, and duration of the disease three clinical varieties of the disease are recognised : *acute*, *chronic*, or *recurrent*.

(I) **Acute Appendicitis.**—(*a*) *Symptoms.*—In a large majority of cases the onset is sudden with abdominal pain, at first diffuse, later localized over the appendix ; nausea, vomiting, and frequently constipation ; circumscribed tenderness in the appendical region, and fever. *Pain* may be colicky and sharp, or dull and aching. It is often diffuse at the outset, or felt in any part of the abdomen, but in 24 or 48 hours settles in the site of the appendix, whence it may radiate into the testicle or down the right leg. It is usually increased by movement or coughing. *Gastro-intestinal symptoms* are usually present. Nausea and vomiting, which in mild cases are neither severe nor prolonged, are common at the outset. Constipation is usual and may be so obstinate as to simulate obstruction, but it must not be forgotten that there may be an initial diarrhœa, especially in children or in early adult life.

Palpation reveals *localized tenderness*, generally situated at McBurney's point (Fig. 174, page 493), although in exceptional instances of an unusual location of the appendix the "tender point" may be found in the right groin or deep in the pelvis, or in the left iliac, umbilical, right hypochondriac and right lumbar regions. *Rigidity of the right rectus*, another important early symptom, and which, because of its one-sided character, is determined with ease, usually coexists with the tenderness. *Tumour* may or may not be palpable after the first 24 or 48 hours. It may be quite absent in the severest cases. When present it may be either an indefinite induration or a circumscribed mass, and its extent and character are often obscured by the coincident tenderness and rigidity. Percussion gives uncertain results. The note may be dull, dull tympanitic, or tympanitic according to the relative amounts of gas, fæcal matter, and exudate. The induration is found about 1½ to 2 inches above Poupart's ligament, unless the appendix is in some one of the unusual situations previously mentioned.

Fever, an important diagnostic symptom, is commonly present, varying from 100° to 102°, or at the outset in children rising to 103° or over. When an intense diffuse peritonitis or a gangrenous appendix is present, or a circumscribed abscess has formed, the temperature may be normal or subnormal, but the associated symptoms are of too grave a character to be in keeping. A chill at the onset is very infrequent.

The *urine* is scanty, and usually contains an excess of indican, often also a trace of albumin. The bladder is often irritable at the onset, and the frequent micturition may suggest a cystitis. Later in the disease retention may occur. Leucocytosis is usually present.

When the *peritoneum* is involved the facies is often indicative of anxiety. The dorsal posture, with the right leg persistently drawn up, is common and often suggestive. The tongue is moist and furred, becoming later, in severe cases, dry and brown. The pulse corresponds at first with the temperature; subsequently, but not always, with the gravity of the local condition.

(*b*) *Possibilities and Terminations.*—In general there are three eventualities in every case of acute appendicitis: *resolution*, *abscess formation*, or *diffuse peritonitis*.

(1) Resolution.—In the majority of the milder cases the pain rapidly settles into the appendical region; the tenderness is moderate and steadily diminishes, requiring deep pressure to elicit it; the abdominal distention and rectus rigidity are not marked and steadily lessen; the initial nausea and vomiting, if present, soon subside; the fever continues for from 3 to 5 days and then departs. In a week or 10 days the patient is thoroughly convalescent. In some instances a slight elevation of temperature may continue for 2 or 3 weeks, and while apparent recovery takes place there remains some hardness or a small swelling in the appendical region, a condition ominous of recurrence.

(2) Circumscribed Suppuration.—Earlier in the severe cases, later in the mild, in both usually as the result of ulceration and perforation, less commonly from extension of the inflammation through the non-perforated appendical walls, a circumscribed abscess, generally intraperitoneal, may form.

As evidences of suppuration the fever persists, the pain increases, the tenderness covers a wider area from hour to hour, especially downward toward the pelvis, the constipation becomes more obstinate, and frequent urination or retention is common. A tumour, deep-seated or superficial, becomes manifest, or, if present, increases in size, and in either case is apt to be excessively tender. It is to be remembered that an amelioration of the general symptoms may occur after the abscess has become thoroughly walled off.

The abscess cavity may be small or large, symmetrical or irregular, and is limited by adherent coils of intestine or, partly, by various portions of the abdominal or pelvic walls. If the abscess is not surgically evacuated it may empty itself into the bladder, vagina, rectum, or cæcum, or enter the hip joint, or pass out through the obturator foramen. If it is retroperitoneal it may discharge externally in the groin, or form a large perinephric abscess, or, lying under the diaphragm, give rise to the symptoms of subphrenic abscess or (if it contains air) of pneumothorax, perhaps followed by perforation of the pleura and a resulting pleuro-fæcal fistula. Finally, a localized periappendicular abscess may rupture slowly or suddenly into the general peritoneal cavity with resulting diffuse peritonitis.

(3) Acute Diffuse Peritonitis.—Following perforation of the appendix, or rupture of an abscess either in mild or severe cases, a general peritonitis may ensue. The symptoms indicating the supervention of general peritonitis are extreme and diffuse pain, a rigid and distended abdomen, nausea and vomiting, rapid pulse, dry tongue, anxious countenance, dorsal posture with the knees drawn up, and often a normal or subnormal temperature.

This condition may develop rapidly from the outset in cases of early perforation in which an immediate general infection occurs without a limiting inflammation; or more gradually when the perforation has occurred later in the disease after a certain amount of protective adhesion has taken place, or when a periappendicular abscess has ruptured and its contents slowly infect the peritoneum. The symptoms of gangrene of the appendix may be latent and excessively deceptive.

(*c*) *Complications.*—Some of the events previously mentioned may be considered as complications. Under this head may be included suppurative pylephlebitis, causing multiple hepatic abscesses; thrombosis or compression of the right iliac vein; hemorrhage from perforation of the intestine or necrosis of the iliac arteries; subphrenic abscess, perhaps followed by perforation of the diaphragm, and pleurisy (serous or suppurative) or pericarditis; appendicitis in a hernial protrusion; and suppurative parotitis. The majority of these are of rare occurrence.

(*d*) *Diagnosis.*—The cardinal symptoms are acute pain in the right lower abdomen, localized tenderness with or without tumour, and fever, even if slight. As Fowler well says, "pronounced tenderness in the right iliac fossa is almost as pathognomonic of appendicitis as rusty sputum is of pneumonia." If to the symptoms just mentioned are added nausea, vomiting, right rectus rigidity, and tumour, the diagnosis is absolute.

Although a typical case of appendicitis is unmistakable, there are variations in the severity and manner of onset, and an occasional lack of correspondence between the symptoms and the actual local condition which may cause the most expert diagnostician to trip. For example, the disease may begin with diarrhœa, and be mistaken for catarrhal enteritis; mild cases, with slight symptoms and lasting only for a few hours, are often self-diagnosed as "biliousness" or "cold," and in others the symptoms may be insignificant and the patient may be up and around carrying an abscess; the tenderness may be in an unusual location; the severest cases may have a subnormal temperature; rectus rigidity may be lacking if the peritoneum is not involved; the symptoms may lessen and yet the patient get up with an abscess; a gangrenous appendix may be present with a most misleading mildness of symptoms; and, finally, a diffuse peritonitis may be present from the very outset of the disease.

Careful, complete, and repeated abdominal palpation in all cases, and examination by the rectum (emphasized by Delatour) and vagina in obscure or suspicious cases should never be omitted.

Suppuration is announced especially by an exacerbation of previous symptoms and a slow increase in the size of the tumour.

Perforation of the *appendix*, present or imminent, is usually indicated by severe pain, excessive tenderness, and marked right rectus rigidity. Imminent perforation of the *bladder* may be signified by painful, scanty, and frequent urination. Imminent perforation of the *rectum* may be heralded by tenesmus and the passage of thick blood-stained mucus. Multiple suppurations of the liver are announced by chills, irregular fever, and swelling with tenderness of the organ.

With reference to the *leucocyte count* in appendicitis Greenough concludes that a leucocytosis is a fairly constant symptom; that a count of 20,000 on the first or second day suggests general peritonitis; that a count of 10,000 or less after the first week indicates, if the symptoms are grave a general peritonitis, if the symptoms are severe a mild catarrhal inflammation or a subacute well-walled-off abscess; and that a count of 20,000 or over after the first week or ten days is significant of a local abscess.

(*e*) *Differential Diagnosis.*—The following conditions may require differentiation:

(1) Renal Colic.—There is no tumour, no fever, no "spot" of tenderness, hæmaturia is present, and the pain radiates into the groin and testicle.

(2) Acute Cholecystitis.—An inflamed and distended gall bladder presents pain, tenderness, tumour, and fever, but the tumour is rather above than below the level of the navel; it is pear-shaped, movable, and usually lies superficially, and jaundice is often present. Nevertheless mistakes will occur.

(3) Indigestion and Entero-colitis.—In these, nausea, vomiting, and epigastric or colicky abdominal pain may closely simulate appendicitis, but there is no circumscribed tenderness, no rigidity,

no tumour, and while diarrhœa may initiate an appendicitis it is exceptional.

(4) Acute Intestinal Obstruction.—There may be a more or less distinct tumour with localized tenderness, but this is not necessarily in the appendical region. If due to intussusception, there will be, especially in children, rectal tenesmus with the frequent passage of bloody mucus; if to internal strangulation, fæcal vomiting may be present, neither of which occur in appendicitis. Moreover, the onset is more gradual, fever, if present at all, is usually of late occurrence, and constipation is more apt to be complete. Nevertheless mistakes will be made.

(5) Typhoid Fever.—See page 743.

(6) Salpingitis, Ovaritis, or Ectopic Gestation.—An abscess of the right ovary or a right-side salpingitis may in some instances simulate very closely a low-down appendical abscess. Both cause fever and right-side pain and tenderness, but a history of previous or present menstrual irregularities, together with a pelvic examination which reveals a fixed uterus, an indurated pelvic exudate, or an abscess cavity joined to the uterus by the ridge of the broad ligament, will, as a rule, suffice for the discrimination. But in one case a distinct ovoid mass easily palpated in the right iliac region proved at operation to be an unusually high ovarian abscess. Extra-uterine pregnancy affords a rather characteristic previous history of morning nausea, breast signs, menstrual irregularities, and attacks of colicky pain with faintness, and the physical examination shows a movable mass lateral to the uterus. Fever is absent. If rupture has occurred collapse symptoms are superadded. A diseased right ovary is often associated with appendicitis, especially the chronic form of the latter.

(7) Dietle's Crises.—If the ureter of a right floating kidney becomes twisted, the resulting nausea, vomiting, pain, and tumour may simulate appendicitis, but the outline and mobility of the tumour, the occasional hæmaturia, the absence of fever, and the sudden relief of the symptoms (by spontaneous untwisting), point to the kidney as the offending organ.

(8) Perinephric Abscess.—As this in rare instances may arise from appendicitis, an absolutely positive differentiation can not be made previous to operation.

(9) Tuberculous Peritonitis.—In this the invasion is gradual, right iliac tumour is absent, and the course is more protracted. If a local peritonitis in the right iliac fossa occurs from tuberculous mesenteric glands, time or operation may be required to separate it from appendicitis. In both cases the existence of tuberculous disease elsewhere may offer a valuable clew.

(10) Hysteria and Mucous Colic.—In nervous or hypochondriacal women (or men) presenting mucous colic (*q. v.*), formerly termed membranous enteritis, as one of the evidences of the neuropathic constitution, a mistaken diagnosis is not uncommon. It is to be discriminated from appendicitis by the absence of fever or of strictly limited tenderness or tumour, and especially by the presence of some of the multiform neurotic symptoms of the condition in question.

(11) Appendicular Colic.—Recurring pain in the appendical region, sometimes of great severity, and explained theoretically as an effort on the part of the appendix to expel mucus or fæcal matter through a somewhat narrowed orifice, may be mistaken for appendicitis, but the total absence of fever will rule out the latter.

(12) Pericæcal Abscess from perforation of the intestine in the appendical region by a simple, typhoid, or carcinomatous ulcer can not be separated from appendicitis except by operation.

(13) Disease of Hip Joint.—In young children appendicitis may be mistaken for disease of the right hip joint (Gibney).

(14) Pneumonia.—The abdominal pain, tenderness, rigidity, and meteorism which may occur in pneumonia of the lower lobes (involvement of diaphragmatic pleura) has not seldom led to a misdiagnosis of appendicitis. This error can be avoided by routine examination of the chest. If the physical signs of pneumonia are present, it is possible, but most unlikely, that appendicitis coexists.

(*f*) *Prognosis.*—The mild types usually recover, those with localized abscesses generally get well if operated, the majority of the severe cases with diffuse peritonitis will die, operation or no operation. The prognosis in the individual case is uncertain. My own rule is to have a competent surgeon watch the case with me from the onset. The tendency of the practitioner of internal medicine to temporize is thus counterbalanced by the frequently opposite trend of the surgical mind, and the middle way of wisdom is more apt to be trodden.

(II) **Chronic and Recurrent Appendicitis.**—If relapses occur at comparatively short intervals, of weeks or months, the condition is termed *chronic* or *relapsing* appendicitis; if at longer intervals, of months or years, it is termed *recurrent* appendicitis. The frequency of relapse in all cases has been variously estimated at from 23 to 44 per cent. The symptoms of the relapse or recurrence are similar in all respects to those of the primary attack.

(1) In the *chronic* or *relapsing* form there may be no symptoms in the intervals between the attacks. More commonly the patient complains of a slight but constant sense of discomfort or moderate soreness in the appendical region. From time to time, as a result of

muscular effort, dietetic imprudences, digestive disturbances, or the collection of fæces in the cæcum, the uneasy sensation may develop into moderate pain with perhaps slight fever, the symptoms not being sufficiently acute to be entitled a relapse. Not infrequently the abiding consciousness of the nature of the ailment will, by causing persistent anxiety, lead to the development of neurasthenic or hypochondriacal symptoms with impairment of the general health.

The diagnosis of chronic appendicitis is not often doubtful. The presence of localized tenderness, possibly of tumour, perhaps with occasional slight fever, will serve to distinguish it from a mere "appendicular hypochondriasis" or phobia. Moreover, by suitable diversion of the patient's mind during the examination an apparent right iliac tenderness may be made to disappear. It is an open question whether the normal or thickened appendix itself can be so distinctly palpated and recognised as is claimed by some writers. Surgeons of my acquaintance of as wide experience and with quite as good a *tactus eruditus* deny it positively.

Malignant disease of the cæcum, by causing pain, tenderness, and tumour, may closely simulate chronic appendicitis, but a history of gradually increasing constipation, progressive loss of flesh and strength, the development of a cachexia, and the usual absence of febrile movement point toward carcinoma. Nevertheless, operation may be required for the discrimination.

(2) In *recurring* appendicitis the separate attacks may differ in severity, but during the intervals the patient is practically well.

X. **Intestinal Obstruction.**—(*a*) **Causes.**—Strangulation, intussusception, volvulus, fæcal impaction, enteroliths, gallstones, tumours, strictures, intestinal paralysis, and foreign bodies.

(*b*) **Symptoms.**—With reference to its suddenness of occurrence, two clinical forms of obstruction are recognised: *acute* and *chronic*.

(1) *Acute Obstruction.*—The cardinal symptoms are abdominal pain, vomiting, and obstinate constipation.

Pain, which may be in any part of the abdomen, is the earliest of these, and generally comes on during movement or some sudden exertion. It is at first colicky and intermittent because the obstruction is partial, but when the stenosis is complete it becomes intense and continuous. Abdominal tenderness, absent at first, may become excessive. Constant and distressing *vomiting* soon ensues, at first of the stomach contents, then of bile containing green fluid, finally of a dark fluid with a fæcal odour—a most important symptom. *Constipation* may be present from the outset, but very often the contents of the intestine below the point of stenosis are evacuated, after which no further fæcal stools are voided. Tympanites makes its appearance from the second to the sixth day. It is most marked when the obstruction is in the colon, and slight when the stenosis is high up in the small intestine. Tumour is rare, except in intussusception. Tenesmus is frequent if the obstruction is in the colon, especially if there is intussusception, less often with volvulus and stricture. Excessive peristalsis may be seen, accompanied by easily audible rumbling, gurgling, or splashing sounds.

The *general symptoms* are well marked and grave from the beginning of the disease. They are prostration, pallor, an anxious expression, cold sweating skin, and a normal or subnormal temperature, more rarely moderate fever (100° to 102°), usually late in the disease. Thirst is extreme, the tongue is dry, the respiration accelerated, and the pulse feeble and rapid. The urine is scanty, high coloured, and may be suppressed. The *duration* of acute obstruction is usually from 3 to 6 days, the patient becoming comatose or dying from exhaustion, or in very acute cases from rapid collapse.

(2) *Chronic Obstruction.*—The obstruction for a long time may be only partial, but with a gradually increasing degree of constipation. Occasional diarrhœa may occur, especially in fæcal impaction, from a colitis excited by the irritation of hardened scybalæ; or regular but inadequate stools may be passed by a way channelled through the retained mass. In stenosis due to tumour or cicatricial stricture the degree of constipation varies from time to time. There may be, at irregular intervals, passing attacks of vomiting, with colicky abdominal pain and distention. In chronic obstruction the stools may be small, hard, and scybalous, or ribbon-shaped, perhaps containing mucus and blood, especially if the colon is the seat of the stenosis. The general health is impaired, particularly in malignant disease, anæmia, emaciation, or cachexia occurring.

In any case of chronic stenosis the symptoms may become those of the acute form from the sudden completion of the occlusion, death occurring in from 10 to 12 days.

(*c*) **Complications.**—There may be catarrhal, diphtheritic, or suppurative inflammation of the mucous membrane, with localized peritonitis in the immediate neighbourhood of the obstruction; gangrene or ulceration, with perforation and a resulting diffuse peritonitis; or pyæmia.

(*d*) **Diagnosis.**—The existence of obstruction having been determined by the cardinal symptoms—acute and severe abdominal pain; vomiting, at first gastric, then bilious, later fæcal; early prostration; and obstinate constipation followed by tympanitic distention—it becomes desirable to ascertain, if possible, the site and the cause of the obstruction.

(1) THE SITE OF THE OBSTRUCTION.—Is it in the small intestine or in the colon?

In general, if in the *small intestine*, vomiting, soon becoming fæcal, is an early symptom, and the tympanitic distention is not prominent and may be slight. If the obstruction is high up, in the duodenum or jejunum, the meteorism is slight, fæcal vomiting may not be marked, indicanuria is common, collapse and suppression of urine occur early and rapidly. If in the ileum, the distention is greater, is manifest mainly in the mid-abdomen, and fæcal vomiting is early present.

In general, if the obstruction is in the *colon*, abdominal distention is marked, fæcal vomiting is much less frequent, and indicanuria is seldom found. If the obstruction is in the lower end of the ileum or in the cæcum, inspection may show the ladder pattern of tumidity in the lower mid-abdomen; if in the rectum or sigmoid flexure of the colon, the latter may stand out prominently, in a horseshoe shape, around the upper and lateral portions of the abdomen, and tenesmus with the passage of blood and mucus may be present.

Abdominal examination may reveal a tumour or swelling, but this is rare except in intussusception or fæcal accumulation. Digital examination of the rectum may enable the recognition of a tumour or stricture, or the emptiness or fulness of the cavity. Information may be gained by noting the amount of water which may be injected, under anæsthesia, by the fountain syringe, the patient lying upon the back with the hips elevated. Normally, in an adult, 6 quarts, in an infant, 1½ pints, under a pressure of 3 feet, will enter. Unless more than 3 pints will enter in an adult, obstruction at the sigmoid flexure can not be eliminated. Auscultation over the cæcum may be employed during the inflow, to recognise the arrival of fluid at this point (TREVES).

(2) THE CAUSE OF THE OBSTRUCTION.—If it is difficult to determine its site, it is still more difficult to decide the nature of the obstruction. The doctrine of probabilities does not apply to the individual case, but it is helpful to remember that, roughly, according to Fitz, of all cases, strangulation will be present in about 35 per cent, intussusception also in 35 per cent, volvulus in 15 per cent, gallstones in 8 per cent, and stricture or tumour in 6 per cent.

Obstruction in the *small intestine* is usually due to strangulation (72 per cent) or gallstones (14 per cent), less frequently to intussusception (8 per cent), volvulus (5 per cent), rarely to stricture or tumour; in the *colon* mainly to intussusception (51 per cent) and volvulus (30 per cent), less commonly to stricture or tumour (12 per cent).

Acute obstruction is usually due either to strangulation or intussusception, less commonly to volvulus; *chronic obstruction* mainly to strictures, tumours, or fæcal impaction.

The following points may be of some assistance in determining the cause of the obstruction:

Strangulation.—Most commonly due to adhesions from previous peritonitis or cœliotomy; next most commonly to Meckel's diverticulum (the remains of the omphalo-mesenteric duct) which springs from the ileum, about 1½ feet from the ileo-cæcal valve, as a finger-like projection 3 to 10 inches in length, the free end of which may become adherent to the abdominal wall near the umbilicus or to the mesentery. Strangulation may also occur as a result of the tip of the appendix or the Fallopian tube becoming adherent; or of the existence of omental and mesenteric slits, or peritoneal openings and pockets (foramen of Winslow, duodeno-jejunal fossa); or a pedunculated tumour; or rupture of the diaphragm with hernia. Through any of the rings, slits, or openings a loop of intestine may slip and become strangulated.

The evidence pointing toward strangulation is, the age of the patient, between 15 and 30 years; the sex, predominating in males; a previous history of peritonitis or cœliotomy; early fæcal vomiting; and usually acuteness of obstruction. A palpable tumour is rare.

Intussusception.—A portion of the intestine, because of irregular peristalsis, becomes invaginated into a lower portion, the invagination increasing downward. In most cases—75 per cent—the ileum enters the cæcum (*ileo-cæcal*) or the colon (*ileo-colic*); less frequently the ileum (*ileal*) or the colon or cæcum (*colic*) enters itself, or the colon enters the rectum (*colico-rectal*).

In intussusception there are no previous symptoms; the patient is usually an infant or child (56 per cent of all cases occurring from the 4th month to the 10th year) or a young adult; the onset is acute; tenesmus, mucous and bloody stools are present; and a palpable sausage-shaped tumour is generally to be felt (63 out of 93 cases) in the right iliac or umbilical regions, less frequently to be palpated by the rectum.

Volvulus.—A twist, usually axial and of the large intestine (87 per cent). In 50 per cent it is in the sigmoid flexure, next most commonly (33 per cent) about the cæcum.

The symptoms are not distinctive and the condition is seldom diagnosed. There is, in volvulus, often previous constipation and flatulence, the patient is rarely under 40 years of age, the onset is usually acute, the abdomen is greatly distended, tender, and often rigid. Because of the frequent occurrence of volvulus at the sigmoid flexure the injection of fluid may be of diagnostic value by proving that the usual amount will not enter.

Fæcal Obstruction.—The obstruction is usually chronic. There is a history of habitual constipation, becoming progressively more difficult to overcome; the patient is apt to be a woman, probably in middle or advanced life; abdominal pain, meteorism, nausea, and fæcal vomiting are not constant and appear late; and fæcal masses, dull on percussion, sometimes indentable, occasionally tender, can be felt along the distended colon, especially in the cæcum and sigmoid flexure, and by rectum. The diagnosis can usually be made, but the possible tunnelling of the mass, with the passage of fluid stools, is to be remembered. If the impaction is in the rectum, especially in old persons, there may be constant tenesmus.

Obstruction by Gallstones.—A large concretion which has ulcerated through into the intestine may cause obstruction. It may be suspected if in a person over 50 years of age there is a previous history of hepatic colic with or without jaundice; occasionally a hard movable nodule can be found by abdominal palpation. Pain and vomiting, usually fæcal, are early symptoms. I have watched the travels of a palpable gallstone through the intestine, causing intermittent obstruction as it lodged transiently from time to time.

Stricture or Tumour.—In strictures resulting from dysenteric, tuberculous, syphilitic, or malignant ulceration, or due to tumour, the obstruction is chronic. Abdominal examination may reveal a tumour, and rectal exploration a stricture or tumour.

Paresis of the Intestine.—As a result of sepsis, peritonitis, or defective innervation, or following a cœliotomy, the intestinal muscularis may lose its motor activity—a condition practically equivalent to obstruction. The history is of service in its recognition; the abdomen is uniformly rounded, smooth, and very tympanitic, and, most important, patient auscultation reveals an absence of the usual sounds.

The presence of *miscellaneous* and *rare* causes of obstruction, such as knots, foreign bodies (masses of roundworms, peanuts, glass, stones, false teeth, nails) or enteroliths (deposition of salts about bits of bone, clumps of hair, fruit stones, thread) can, as a rule, only be conjectured.

(*e*) **Differential Diagnosis.**—Acute intestinal obstruction, without reference to its site or cause, is to be distinguished from the following conditions:

(1) *External Hernia.*—A strangulated external hernia (inguinal, femoral, umbilical, etc.) must be excluded as a cause of obstruction by a careful examination of the usual sites of protrusion.

(2) *Acute Enteritis.*—Severe cases of this disease with pain, tympany, intestinal paresis, and vomiting, may simulate acute obstruction so closely that in certain instances a differential diagnosis is impossible. Ordinarily the presence of fever, and of diarrhœa with mucus and blood; and the absence of intractable constipation, fæcal vomiting, tumour, or marked meteorism, will declare against obstruction.

(3) *Acute Diffuse Peritonitis.*—Contrasting peritonitis with obstruction, there is in the former an initial and decided rise of temperature; constant and general abdominal distention, sometimes with effusion; and absence of tumour, of intestinal sounds on auscultation, of excessive visible peristalsis, and of fæcal vomiting. Inquiry may reveal some of the antecedent causes of peritonitis (*q. v.*). Nevertheless time is often requisite for a diagnosis, and mistakes will occur.

(4) Acute hemorrhagic *pancreatitis* (*q. v.*) may be readily mistaken for obstruction; *hepatic colic* may be eliminated by the site of the pain and perhaps the appearance of jaundice; *renal colic* by the site and radiation of the pain, and the presence of blood in the urine.

(*f*) **Prognosis.**—This is always grave, particularly in acute obstruction. It is more favourable in fæcal obstruction.

XI. **Carcinoma of the Intestine.**—As a result of carcinomatous or other tumour of the bowel the lumen of the intestine is usually, but not necessarily, obstructed. Carcinoma occurs more frequently in men, particularly after 50 years of age.

The growth is almost invariably found in the large intestine (95 per cent of all cases); in the rectum in 80 per cent, in the cæcum or colon in 15 per cent. In the colon it involves the flexures—hepatic, splenic, and sigmoid—in particular. The order of frequency in various parts of the intestine is, rectum, sigmoid flexure, transverse colon, opening of the common bile duct (papilla duodenalis), ascending colon, lower portion of ileum.

(*a*) **Symptoms.**—The early symptoms are not at all distinctive, consisting merely of an irregularity of the bowels, which may continue for a long time before the growth ulcerates or becomes of such a size and character as to cause narrowing or palpable tumour, although an associated and apparently disproportionate loss of flesh and strength is suggestive of malignancy. In time the symptoms usually become those of chronic obstruction (page 874). Marked constipation exists, sometimes alternating with diarrhœa. At irregular intervals there may be attacks of colicky pain with vomiting, obstinate constipation, and moderate abdominal distention; or beginning as a constant localized discomfort, the pain may become colicky and f daily occurrence, especially a few hours after eating, and there is noderate local tenderness. The fæces may be voided in pellets or arger scybalæ, or, if the narrowing is low down, as ribbon-shaped ieces. The presence of blood or bloody mucus, pus, or shreds of issue (which sometimes show a carcinomatous structure) may be aken as evidence of ulceration; when this occurs there may be a iarrhœa. At any time complete obstruction may take place with evere pain, meteorism, and fæcal vomiting. Important symptoms re anorexia, emaciation, and progressive pallor and yellowness of he skin, indicative of the cancerous cachexia.

If, as it may be, the growth is so situated that it does not cause tenosis of the bowel, the obstructive symptoms may be absent, the achexia and perhaps the finding of a tumour constituting the most ignificant evidence.

(*b*) **Duration, Prognosis, and Complications.**—Death usually occurs rom asthenia, perhaps with pulmonary œdema, in from 6 months to year (in rare instances 2 to 3 years) after the discovery of tumour, lceration, or obstruction. Surgical treatment, however, may prolong life, and in cancer of the rectum some excellent results have ollowed removal.

The complications are hydronephrosis and renal changes from nvolvement of the ureters; cancerous peritonitis; perforation of the ntestine and sequent diffuse peritonitis; recto-vaginal and recto-vesical fistula; embolism of pulmonary artery from a thrombus in an iliac vein; cellulitis; pyæmia; and, rarely, rupture of the intestine from distention due to accumulated fæces. Secondary deposits may occur in the liver and lungs, less commonly in more distant parts or organs.

(*c*) **Diagnosis.**—(1) *Of Carcinoma.*—The cardinal symptoms are the age of the patient (over 50), cachexia, the detection of a tumour, and, usually, the signs of chronic obstruction. All malignant tumours are included under this head. As a benign tumour may give rise to many of the symptoms mentioned, exploratory operation, or the progressively downward course of the case, or the appearance of secondary deposits, may afford the only positive evidence of malignancy.

(2) *Of its Site.*—If an *abdominal* tumour has been found and is judged to be malignant, it remains, if practicable, to determine its site. If the tumour is nodular and hard, and lies near the junction of the epigastric and umbilical regions, and there is obstinate jaundice of the obstructive type, it may be inferred that the disease is in the duodenum and involves the opening of the common bile duct (rare); if without jaundice the transverse colon. If the tumour is

low in the right iliac region, it may implicate the cæcum; if on a level with the umbilicus and to the right, the ascending colon or hepatic flexure; if in the left iliac region or higher, the sigmoid flexure, ascending colon, or splenic flexure. Inflation of the colon with air, or distention with water, may serve to determine the height of the stenosis, or make the relation of the tumour to the colon more distinct. Carcinoma of the *rectum* is often mistaken for chronic dysentery, because of the pain, tenesmus, and voiding of fœtid mucous and bloody stools, but a digital examination will reveal an ulcerated mass or annular hard infiltration.

(*d*) **Differential Diagnosis.**—Certain conditions may be mistaken for carcinoma intestinalis. The differentiation must be made by the presence of the signs and symptoms just described, a consideration of the characteristics of the possible confusing ailments, and, in not a few cases, by exploratory operation, a procedure which will not add to the gravity of carcinoma and in other cases may result in recovery. These conditions are: Fæcal impaction, which may also coexist with cancerous stenosis; the tumour of intussusception; pyloric tumour; cancer of the head of the pancreas, which is immovable and deep-seated; distended or carcinomatous gall bladder, or "lacing" liver; movable or enlarged kidney, and chronic appendicitis.

XII. **Dilatation of the Colon.**—This may occur as a result of obstruction (tumour, volvulus) in some part of the colon; as an acute condition due to twisting of the meso-colon, or acute and temporary from gaseous distention; as a consequence of habitual constipation and coprostasis; or it may be idiopathic, due perhaps to a low-down congenital narrowing of the colon. The *symptoms* are usually those of obstinate or habitual constipation, often with extreme abdominal distention and meteorism, which may be sufficient to cause upward displacement of the liver, spleen, diaphragm, heart, and lungs.

XIII. **Diseases of the Mesentery.**—The diseases of clinical interest are cysts and infarctions.

(*a*) *Mesenteric Cysts.*—These may be chylous, dermoid, hydatid, serous, or sanguineous; of varying size, in rare instances filling the abdomen; and frequently becoming adherent to the liver, spleen, sigmoid flexure, and uterus. The *symptoms* are those of a steadily growing abdominal tumour, usually in the epigastric region, occasionally with colicky pain and constipation. The *diagnosis*, usually made at autopsy, is extremely doubtful, and may be suggested by the chronic course (10 to 20 years), the tympanitic note due to the presence of intestine over the growth, and the situation of the tumour in the middle line. Commonly it is supposed to be an ovarian tumour, or an omental or hydronephrotic cyst, or a movable kidney.

(*b*) *Intestinal Infarction.*—The superior mesenteric artery may be occluded by emboli or thrombi derived from diseased heart valves or aneurism of the abdominal aorta, with resulting hemorrhagic infarction of a portion of the jejunum or ileum. The *symptoms* comprise violent and colicky abdominal pain; diarrhœa, perhaps with blood-stained stools; vomiting, which may be fæcal; and abdominal distention. As the clinical picture is almost exactly that of acute obstruction, a diagnosis is rarely possible prior to operation, unless the presence of the causative conditions may suggest the true nature of the case.

XIV. **Intestinal Neuroses.**—These may be *motor*, *sensory*, or *secretory*.

(I) MOTOR NEUROSES.—(1) **Nervous Constipation.**—In neurasthenia, hysteria, and various forms of nervous disease, constipation, often associated with slow or sudden gaseous distention, is due to deficient innervation and muscular atony of the intestinal muscularis. Paralysis of the voluntary control of the sphincter ani, the act of defecation becoming reflex and involuntary, is met with not only as a result of organic or functional central nervous disease, but also of local inflammatory processes.

(2) **Nervous Diarrhœa.**—This occurs mainly in neurasthenic, hysterical, or "nervous" persons, and is due to an unusual irritability of the motor nerves of the intestine, which results in excessive peristalsis. In such persons anxiety, worry, fright, or any strong emotion will produce or aggravate a diarrhœa. It may also occur as a symptom of organic disease of the nervous system, especially tabes.

The stools are soft or watery and do not contain blood or mucus (except in mucous colic), and vary from 2 to 20 per day. Characteristically, the diarrhœa may occur only in the morning (morning diarrhœa, DELAFIELD), may come suddenly and after lasting several days suddenly stop, or the condition may persist continuously for months or years. Aside from the nervous symptoms the general health may remain good. Inflammatory or other organic disease of the intestine must be excluded before this diagnosis is made.

(3) **Enterospasm.**—Excessive contraction of the muscular fibres of the intestine is usually associated with enteralgia (page 882), but may exist without pain. It may produce transient severe constipation or even obstruction, but this diagnosis can seldom be made because of the extreme difficulty in positively excluding the organic and other causes of constipation or stenosis. Spasm of the rectum and sphincter (*proctospasm*) is not uncommon as a neurasthenic or hysterical neurosis, or as a symptom of hemorrhoids or anal fissure.

It is usually discovered on attempting a digital examination of the rectum, but may be responsible for ribbon-shaped stools.

(II) SENSORY NEUROSES.—These comprise *enteralgia*, *neuralgia of the rectum*, and *diminished intestinal sensibility.*

(1) **Enteralgia.**—Neuralgia of the intestines, which, when associated with spasm of the muscularis, is termed intestinal colic, may be a pure or a reflex neurosis, or may be symptomatic of organic disease. As a *neurosis* it occurs particularly in anæmic, neurasthenic, or hypochondriacal persons, because of the increased sensibility of the sensory nerves, and may be excited by sudden, especially unpleasant, emotions. As a *reflex neurosis* it arises from exposure to cold, or from gout; from foreign bodies and the irritation of worms, accumulated fæces, enteroliths, indigestible food, or an excessive amount of gas (flatulent colic) in the intestine. As a symptom of *organic disease* it occurs in locomotor ataxia ("crisis"); in lead-poisoning, probably by direct action of the toxic agent upon the nerves; and in various abdominal diseases, such as intestinal obstruction, appendicitis, peritonitis, enteritis, and the like.

The *symptoms* comprise pain, usually diffuse, sometimes localized, which may be extremely severe. It is often, but not always, of sudden onset, may be relieved by pressure or forward doubling of the body, and commonly lasts for a few hours, rarely days, and may terminate gradually or suddenly. There is often a free discharge of flatus, after which the attack tends to subside. Recurrences at varying intervals are common. In the *diagnosis* of a neurotic or reflex enteralgia it is necessary to exclude the colicky pain which is symptomatic of inflammatory abdominal disease by the absence of fever and the relief afforded by pressure; lead colic and tabes by the associated symptoms. It is usually easy to separate enteralgia from renal or hepatic colic and rheumatism of the abdominal walls. As a matter of fact, a purely neurotic enteralgia is not common.

(2) **Rectal or Pubic Neuralgia.**—This occurs in neurasthenic, hysterical, or tabic individuals, sometimes in connection with hemorrhoids. There is an undue sensibility of the hemorrhoidal plexus of nerves, manifested by a feeling of pain or discomfort in the pubic region, sacrum, and perinæum, often extending to the thighs, with an aching tenesmic desire to evacuate the rectum, although the latter may be empty.

(3) **Diminished Intestinal Sensibility.**—There may be anæsthesia of the intestine, so that the normal indication of the need of defecation is absent and constipation ensues. It is a common symptom of the spinal paralyses. Unless motor paralysis is present purposive defecation still takes place.

(III) SECRETORY NEUROSES.—**Mucous Colic.**—The only clinically interesting secretory intestinal neurosis is the mucous neurosis (membranous enteritis, mucous colic) affecting the follicles of the colon. It is properly considered to be one of the manifestations of hysteria, neurasthenia, or the neuropathic constitution. The great majority of cases occur in women, and it is most frequent between the 30th and 40th years, but has been observed in children of from 3 to 12 years of age.

(*a*) *Symptoms.*—The disease generally begins in a subacute manner. Whether its onset is acute or subacute, its subsequent course is chronic. There are more or less persistent symptoms of gastro-intestinal derangement, which differ little from those of ordinary occurrence. The characteristic events are the painful paroxysmal passage of membrane and a peculiar train of phenomena referable to the nervous system. The paroxysms may occur daily, or at intervals of a month, or at any intermediate period. The pain begins lightly, is referred to the lower abdomen, increases in severity, reaches its acme, and in many cases is relieved by the passage of membrane, after which it gradually declines. The paroxysms may last for a day or a week. The pain itself is colicky, tenesmic, and of a peculiarly sickening character, producing a facies like that which accompanies pressure on a tender ovary. There is almost invariable abdominal tenderness, sometimes so great and general as to simulate peritonitis. It is usually circumscribed in either iliac fossa, especially the left. This abdominal tenderness may be persistent in varying degrees during the continuance of the disease. There may be vesical and uterine tenesmus, and mucous discharges from these organs. The membranes may be shreddy, ribbon-shaped, cordlike, or may constitute perfect cylindrical casts of the intestine. The quantity ranges from a very small amount up to three kilogrammes in one paroxysm. It may be passed with fæcal matter or alone. By stirring and decantation with water separation is readily effected. Chemically the membrane is composed of dense transformed mucus (OSLER). The temperature is always normal or subnormal. The general nutrition usually, but not invariably, suffers. Emaciation, anæmia, and loss of strength may occur in varying degrees. Diarrhœa and constipation, hemorrhoids, rectal prolapse, jaundice, polydipsia, aphthous stomatitis, and furuncles may coexist with the disease.

The nervous phenomena are peculiar and striking in the extreme. Some of these are practically of invariable occurrence in this disease. Among them are hysteria and hysterical stigmata of all kinds: hysterical coma, convulsions, and aphasia; neurasthenia, vertigo, attacks of blue nails and lips, tingling and numbness of hands and feet, acute neuralgias of all parts of the body, pain in the external ear, tender scalp, tinnitus aurium, hyperæsthesia, paræsthesia, anæsthesia, temporary defects of vision, morbid alterations of taste, irregular muscular tremors, paresis, paralyses, chorea, catalepsy, amnesic aphasia, mental depression, poor memory, hypochondriasis, and melancholia. Many if not all of these are transient and, in the absence of definitely ascertained lesions, largely functional in character. Finally, to these may be added the peculiar paroxysmal pain and tenderness.

When occurring in children it is found, after eliminating simple intestinal catarrh, that the subjects are from parents whose nervous systems are diseased, or who have suffered from convulsions, hysteria, neuralgia, rheumatism, or insanity. The children themselves have shown convulsions, passionateness, morbid timidity, chorea, or rheumatism (EDWARDS).

(*b*) *Diagnosis.*—I am persuaded that the existence of this disease is not infrequently overlooked. Da Costa's rule is good and practical—to suspect this disease "in every case of anomalous nervous symptoms, particularly hysterical, in which there is abdominal pain." Membrane, if found, must be discriminated from ascaris lumbricoides and the varieties of tænia, fatty discharges, undigested portions of vegetable food, arteries, ligaments, fibrous and elastic tissues of meat, sausage skins, necrosed mucous membrane, fibrinous and diphtheritic shreds, and anal fissure with hypersecretion.

(*c*) *Prognosis.*—The outlook for permanent recovery is not good. Nevertheless, the prognosis, with appropriate and judicious treatment, is not so gloomy as it is usually stated to be.

IX. DISEASES OF THE LIVER, GALL BLADDER, AND BILE DUCTS

(*See Jaundice, pages 86 to 89; and Physical Examination of the Liver, pages 497 to 506*)

I. **Abnormal Form.**—Aside from the alterations in shape which are caused by disease of the organ or by deformities or tumour of the ribs, the only malformation of clinical interest is the "lacing" or "corset" liver. In this the anterior portion of the right lobe is divided from the body of the organ by a transverse groove made by the ribs, the tongue or lappet of liver extending down to or even below the horizontal umbilical line. In rare cases the laced-off portion may be connected with the liver simply by a thin membranous isthmus or band.

Subjective *symptoms* are usually absent, but there may be sensations of weight and pressure; rarely swelling and pain in the lappet as a consequence of interference with the venous outflow. The *diagnosis* is important mainly because of the likelihood of mistaking the separated portion for amyloid disease, passive congestion, or tumour of the liver, abdominal tumour, or movable kidney. The difficulty in discrimination is increased when the lappet is swollen, or the uniting band is thin, or the transverse furrow is so deep as to permit the colon or a loop of small intestine to lie in it, thus causing a line of tympanitic percussion between the isolated lappet and the liver and giving an impression that the two are entirely separate. The lappet, however, usually moves with respiration, and as a rule its edge is found by careful palpation to be continuous with the left lobe of the liver.

II. **Abnormal Position.**—The various causes of a misplaced liver have been described (page 504). Brief reference is here made to the movable or floating liver—a rare condition. It occurs mainly in middle-aged or elderly women with relaxed abdominal walls. There may be a sense of weight and dragging, with referred discomfort or pain in the right shoulder. The condition is to be diagnosed by finding the normal liver dulness absent; the liver, recognised by its shape and notch, prolapsed; and the practicability of replacing the organ in its normal site by position or manipulation.

III. **Acute Catarrh of the Bile Ducts.**—A simple angiocholitis not caused by gallstones. It is in the great majority of cases an extension of a gastro-duodenitis (page 861) to the intestinal portion of the common duct.

(*a*) *Causes.*—It may arise from dietetic errors (most common), exposure to cold, mental or physical fatigue, malaria, influenza, pneumonia, typhoid fever, the passive congestion of chronic valvular disease, and chronic nephritis. It may be epidemic. As a rule it occurs in young persons as a result of indigestion.

(*b*) *Symptoms.*—Occasionally icterus is the only symptom present. Ordinarily there is anorexia; moderate nausea and vomiting, which may persist for 3 or 4 days; mild epigastric or right hypochondriac tenderness; constipation, seldom diarrhœa; fever, if present at all, is moderate (101° to 102°); and often slight swelling of the liver and perhaps of the spleen. The gall bladder is usually not palpable. The pulse and respiration are either normal or, as happens, may be much diminished in frequency. Pruritus is not so common as in the grave form of jaundice due to chronic obstruction. The other signs of icterus simplex (page 88) are present. The onset may be with chill, headache, and severe vomiting, especially in the epidemic variety. The disease usually lasts from 4 to 8 weeks, seldom but 2 weeks or as long as 3 months.

(*c*) *Diagnosis.*—The cardinal symptom is jaundice of a moderate grade occurring in previously healthy young persons, without pain and with or without digestive disturbances. The form due to infectious diseases, nephritis, or cardiac disease is, of course, preceded and accompanied by the symptoms of the causative illness. If the icterus persists longer than 3 months it may be suspected to be due to a graver ailment than simple acute catarrhal inflammation, and the patient should be watched and examined with reference to progressive emaciation, or the evidences of impacted gallstones, cirrhosis, or carcinoma.

IV. **Chronic Catarrh of the Bile Ducts.**—Catarrhal cholangitis occurs in obstruction of the common duct, usually by gallstones, less commonly by malignant disease, stricture, or outside pressure. Osler recognises two groups:

(*a*) *Complete* obstruction of the common duct, the patient presenting chronic and intense jaundice without fever. A history of previous attacks of hepatic colic and slight or absent enlargement of the gall bladder point toward obstruction by gallstones rather than by a neoplasm.

(*b*) *Incomplete* obstruction of the common duct, when due to gallstones, presents recurrent paroxysms of pain, accompanied by chills, fever (103° to 105°), and sweats, the hepatic intermittent fever of Charcot. The chills, which are separated by an apyrexial interval, may be very severe and in their periodicity resemble quotidian, tertian, or quartan ague. Jaundice may be intense and persistent, or varying and intermittent, according to the degree of obstruction or the ball-valve action of the stone, and usually intensifies after each paroxysm. The attack may be accompanied by nausea and vomiting. Bile may be found in the stools from time to time; the liver and gall bladder are slightly or not at all enlarged; and ascites is absent. Steady failure of the health is not common. Each series of attacks may continue for days or weeks, and there may be a recurrence of a series at varying intervals, covering a period of 3 or 4 years. Recovery may occur. It is probable that the repeated attacks take place especially when there is a "ball-valve" stone in the ampulla of Vater, the dilatation in the wall of the duodenum into which the common duct empties. The condition is doubtless due to repeated infection by micro-organisms contained in the bile, the infective process not being sufficiently intense to cause suppurative inflammation (see V following), but the latter may occur as a sequel.

The *diagnosis* requires the exclusion of malaria by the blood examination. As previously stated, the presence of a dilated gall bladder indicates occlusion from other causes than an impacted gallstone in the *common* duct.

V. **Suppurative Inflammation of the Bile Ducts.**—Suppurative cholangitis of both larger and smaller ducts is usually due to gallstones, occasionally to cancer of the duct, worms, foreign bodies, or extension from suppurative pylephlebitis. The *symptoms* are frequent paroxysms such as described in IV preceding, except that the fever is septic and remittent rather than intermittent; jaundice, mild, or not severe; a swollen and tender liver and a moderately enlarged gall bladder; leucocytosis; progressive loss of strength, and emaciation. Pain may be slight. The general symptoms assume a rapid septic or pyæmic type, and death is inevitable.

VI. **Icterus Neonatorum.**—There are two forms, physiological and pathological.

(*a*) *Mild* or *physiological* icterus occurs in about one third of the newborn. The skin and conjunctivæ are yellow, but, as a rule, the urine does not contain bile, and the fæces are of normal colour. It disappears in from 4 to 14 days.

(*b*) *Severe* or *pathological* icterus may be due to congenital obliteration or absence of the common or hepatic duct, syphilitic hepatitis, or a septic phlebitis of the umbilical vein. The jaundice is marked, the urine bile-stained, the stools clay coloured, and hemorrhages from the cord may occur. It is an often fatal disease.

VII. **Stenosis and Obstruction of the Bile Ducts.**—Aside from obstruction by gallstones, the common duct may become occluded from ulceration and adhesion (rare) due to the previous passage of a calculus; or the presence of parasites (lumbricoid worms, echinococcus, distoma hepaticum) or foreign bodies (seeds) in the duct. It may also be occluded by pressure from outside by carcinoma of the pylorus or pancreas; abdominal tumours or aneurism; enlarged glands in the hepatic fissure secondary to malignant disease of the stomach or other abdominal viscera; and cicatricial contraction due to perihepatitis, syphilis of the liver, or ulcer of the duodenum.

The *symptoms* are practically those of a chronic obstructive jaundice (pages 87 and 88). The icterus varies in intensity, but usually increases; the liver is enlarged, except in cases of long standing, when it becomes somewhat cirrhotic and shrunken; the gall bladder is enlarged if the common duct is obstructed, except in the case of gallstones; there is hepatic and referred right-shoulder pain; and hepatic fever—chill, fever, and sweat, with gastric disturbances—especially if the obstruction is due to calculus.

The *diagnosis* as to the exact cause of the obstruction is often extremely difficult. It depends on a careful study of the symptoms (*q. v.*) of each of the possible causative conditions, especially gallstones; carcinoma of the head of the pancreas, or of the pylorus, or the hepatic lymph glands, perhaps secondary to malignant disease of the breast, stomach, rectum, colon, or pelvic organs, not omitting an examination of the clavicular and other glands for confirmatory evidence; and abdominal tumour or aneurism.

VIII. **Acute Infectious Inflammation of the Gall Bladder.**—Acute cholecystitis is due to infection by a variety of micro-organisms, most commonly the *Bacillus coli communis*, *Bacillus typhosus*, *Pneumococcus*, *Staphylococcus*, and *Streptococcus*.

While it is usually associated with the presence of gallstones, it is not so generally recognised that infection may take place without cholelithiasis. One may recognise then a *calculous*, and a *non-calculous* or idiopathic, form. The inflammation may be catarrhal, suppurative (empyema of the gall bladder), or phlegmonous; resulting, in the severer forms, in gangrene, perforation, localized peritonitis and abscess, or a general peritonitis.

(*a*) *Symptoms.*—Acute paroxysmal pain usually in the right hypochondrium, less commonly in the epigastrium or right iliac (appendical) region, is the earliest evidence of the disease. It is shortly followed by nausea, vomiting, abdominal distention, rigidity, and tenderness. The tenderness is at first diffused, becoming localized, but not always over the site of the gall bladder. Prostration is usually well marked or severe. There may be obstinate constipation or even an apparently complete intestinal obstruction, neither flatus nor fæces passing. In the form due to gallstones, jaundice is common; in the non-calculous variety it is seldom present. There may be comparatively mild and recurring attacks of acute cholecystitis without the presence of gallstones.

(*b*) *Diagnosis.*—In the form due to calculi there is usually a history of hepatic colic, followed by jaundice in a certain proportion of cases; in the non-calculous variety, the fact that the patient is convalescing from pneumonia or typhoid fever, or has had previous symptoms referable to the gall bladder, is very suggestive.

The diagnosis is often extremely difficult. The symptoms may exactly simulate those of appendicitis or acute intestinal obstruction, as the local pain and tenderness may be elsewhere than over the gall bladder. The finding of a distended gall bladder and the presence of jaundice, together with a suggestive history, constitute the most distinctive evidence of cholecystitis. In a certain proportion of cases exploratory operation will be required to settle the question.

Extremely severe symptoms, with evidences of local or general peritonitis, point to a suppurative, phlegmonous, or gangrenous cholecystitis, perhaps with perforation of the gall bladder; but such cases, which are fatal without surgical aid, may be attended by deceptively mild manifestations.

IX. **Carcinoma of the Gall Bladder.**—This may be primary, and, if so, is associated with gallstones in about 90 per cent of all cases; or secondary to disease in the liver or neighbouring organs. It usually starts in the fundus of the bladder.

The *symptoms* are chronic jaundice, occurring in about 70 per cent of the cases; persistent pain and tenderness, subject to severe exacerbations; occasionally vomiting, hæmatemesis, melæna, ascites, and fever; in about two thirds of the cases the presence of a firm, tender, and uneven tumour, which, unless adherent, moves with respiration, and extends downward and toward the umbilicus from the usual site of the gall bladder; and the development of cachexia.

Carcinoma may be primary in the ducts, especially the common duct, but this is not common. There is severe jaundice and enlargement of the gall bladder, but the diagnosis is rarely made except by exploratory operation.

X. **Gallstones.**—Cholelithiasis occurs mainly in women (75 per cent), especially those who have borne children. The patient is usually between 40 and 60, rarely under 25, years of age. Other predisposing causes are excessive eating, sedentary occupation, constipation, tight lacing, enteroptosis, and nephroptosis. While small concretions may form in the liver itself, the great majority of gallstones which cause symptoms originate in the gall bladder.

Calculi may, and usually do, remain in the gall bladder for an indefinite period without giving rise to symptoms. If a calculus leaves the gall bladder and enters the ducts, the symptoms of hepatic colic usually arise; if it becomes permanently impacted in the cystic or common duct, the evidences of chronic obstruction appear. Subsequently ulceration and perforation may occur with the formation of a biliary fistula, or the calculus may ulcerate through into the intestine and, if of sufficient size, cause intestinal obstruction. The symptoms of these various events are as follows:

(I) **Hepatic Colic.**—(*a*) *Symptoms.*—The attack is sudden, with excruciating cutting pain, usually localized in the right hypochondrium, whence it may spread over the abdomen and lower thorax, and in some cases be referred to the right shoulder and arm. There are often vomiting, drenching sweats, a feeble and rapid pulse, and occasionally syncope. Rather frequently there is a chill with fever (101° to 103°). The liver may become enlarged and noticeably tender, the gall bladder swollen, tender, and palpable, and the spleen also may swell moderately. Jaundice appears in about one half of the cases, usually within 24 hours after the beginning of the attack and while the stone is passing through the common duct. Ordinarily it is slight and of brief duration, but may be either absent or intense, depending respectively on the freedom of the passage or the degree of temporary impaction of the stone. The urine may contain bile pigment and albumin. Palpitation, præcordial oppression, and an acute mitral murmur have been noted.

The *duration* of the attack is variable, lasting from a few hours to a week, or even longer, with remissions and exacerbations, until the stone is finally expelled. Possible but rare accidents are convulsions, fatal syncope, and rupture of the duct followed by a lethal peritonitis.

(*b*) *Differential Diagnosis.*—If colic of the type just described, and jaundice, even if but a trace, are present, the diagnosis is practically certain. A history of previous attacks is very suggestive. If the character and location of the pain are not distinctive and icterus is absent, a positive diagnosis may be extremely difficult. It is of great diagnostic importance to examine the stools for several days after the attack, in order, by finding the stone, to confirm beyond doubt the nature of the attack. The stool is placed in a fine-meshed sieve and water allowed to run through until the soluble portions have been washed away. It is to be discriminated from the following diseases:

(1) Acute Non-calculous Cholecystitis.—The symptoms of this disease (page 888) are so similar to those of hepatic colic that in certain cases they can not be differentiated except by operation which discloses the absence of calculi.

(2) Renal Colic.—Compared with gallstone colic the pain of right-side renal colic is in the lower abdomen, starting in the lateral or posterior lumbar region and radiating downward into the groin, inner aspect of thigh, and the testicle. Frequent and painful urination may be present, and the urine contains red blood cells. The calculus may be voided by way of the urethra.

(3) Gastralgia.—In this the pain begins near the middle line in the epigastrium, there is rarely chill or fever, jaundice is absent, and no calculus is found in the stools. The patient is usually neurotic.

(4) Enteralgia.—The pain is in mid-abdomen, is relieved by firm pressure and the passage of flatus, and a history of dietetic errors is usually obtained.

(5) Nervous Hepatic Colic.—In nervous women there may be a pseudo-biliary colic precipitated or aggravated by fatigue and

anxiety or other depressing emotions. The liver may be tender, the pain come in paroxysms or be continuous with exacerbations, but the gall bladder is not swollen, jaundice is absent, there is no fever, and no calculus in the stools.

(II) Impacted Gallstones in the Cystic Duct.—When a calculus engages in the cystic duct and becomes impacted certain results may ensue, as follows:

(1) *Dropsy of the Gall Bladder.*—The distended organ can usually be felt below the costal margin as an elastic gourd-shaped, ovoid, or rounded tumour, ordinarily of moderate size, occasionally as large as a fœtal head, rarely of such dimensions as to be mistaken for an ovarian tumour. It may not be sufficiently tense to be palpable. It moves with respiration. Gallstone crepitus may be perceived. Jaundice is not present in obstruction of the cystic duct alone. If the obstruction is recent the gall bladder contains bile, mucus, and perhaps some pus; if of long standing, simply a clear, thin mucous fluid. It may require discrimination from movable kidney (*q. v.*) and carcinoma of the gall bladder (page 889).

(2) *Acute Infectious Inflammation of the Gall Bladder.*—Acute cholecystitis, either catarrhal, suppurative (empyema), or phlegmonous, with or without perforation and subsequent abscess or peritonitis, may arise as a consequence of the impaction in the cystic duct. For the symptoms see Cholecystitis (page 888).

(III) Impacted Gallstones in the Common Duct.—Commonly the stone lodges near the end of the common duct, or there may be a series of stones along the duct.

(*a*) *Symptoms.*—The distinctive signs of a stone impacted in the common duct (NAUNYN) are the presence of jaundice, of variable intensity, for more than one year, with the persistent or intermittent presence of bile in the stools; fever; enlargement of the spleen; absent or slight enlargement of the liver or distention of the gall bladder; and the absence of ascites.

(*b*) *Results.*—The presence of the stone causes a chronic catarrhal inflammation of the bile ducts (cholangitis) which may eventuate in a suppurative cholangitis. Indeed, the symptoms of impacted stone are largely those of the cholangitis excited by its presence. According to Osler, three groups of cases are recognisable, which, with their distinctive symptoms, are as follows:

(1) Complete obstruction with mild catarrhal cholangitis (see IV (*a*), page 886); (2) incomplete obstruction with a severer grade of catarrhal cholangitis (see IV (*b*), page 886), in which recovery is possible; (3) incomplete obstruction with suppurative cholangitis (see V, page 887), in which death is inevitable.

(IV) **Other Sequelæ and Complications of Gallstones.**—The stone may ulcerate, with the formation of a biliary fistula, through the gall bladder or common or cystic ducts into the stomach (rare); duodenum (more common); colon (not uncommon); abdominal cavity (not uncommon); bladder (occasional); lungs (not uncommon), in which case bile may be coughed up; or an external communication (most common) may be established in the right hypochondriac or epigastric regions, by which the stone escapes.

Large gallstones which enter the bowel, and are either discharged or cause obstruction, have usually ulcerated into the duodenum or colon, but in exceptional cases calculi of very considerable size have passed through a greatly dilated common duct.

XI. **Hyperæmia of the Liver.**—This may be *active* or *passive.* The latter is by far of the most clinical importance.

(I) **Active Hyperæmia.**—The *causes* are overeating of rich food, alcoholism, acute infections like malaria or typhoid fevers, amenorrhœa, or the sudden arrest of habitual bleeding from hemorrhoids. The *symptoms* are vague, consisting of a feeling of distress and fulness in the right hypochondrium, especially after an overhearty meal, perhaps with slight tenderness from upward pressure under the costal margin.

(II) **Passive Congestion.**—(1) *Causes.*—This arises from any condition which produces an obstruction to the flow of blood through the right heart, especially chronic valvular disease, less commonly pulmonary emphysema or cirrhosis; or through the inferior cava, as with pressure by intrathoracic tumours. The ultimate result is a "nutmeg" liver.

(2) The *symptoms* are enlargement of the liver, which may be very considerable, the lower border extending as low down as the navel. If due to valvular disease and the tricuspid valve is incompetent, the whole organ may pulsate. The swelling may increase or diminish more or less rapidly. There may be a feeling of weight and discomfort, and the swollen liver is usually tender. Slight jaundice is not uncommon, with clay-coloured stools and bile-tinged scanty urine of high specific gravity. There may be an enlarged spleen; occasionally hæmatemesis; and in advanced cases ascites, followed perhaps by general œdema. Gastro-intestinal disturbances are usually present.

XII. **Thrombosis of the Portal Vein.**—In rare instances clotting may occur in the portal vein as a result of hepatic cirrhosis or syphilis; carcinoma involving the vein; circumscribed peritonitis; perforation of the vein by a gallstone (rare); or pressure by tumours. The *diagnosis* is uncertain and seldom made. An *extremely abrupt* onset of vomiting of blood, intestinal hemorrhage, ascites, and swelling of the spleen will justify a strong suspicion of portal thrombosis, especially if one of the causative conditions be present.

XIII. **Abscess of the Liver.**—**Causes.**—Hepatic abscess is single or multiple, always the result of infection. The infective material may reach the liver by way of the portal vein from lesions in the portal territory due variously to dysenteric, typhoid, or gastric ulcers, appendicitis, amœbic dysentery, disease of the rectum or neck of the bladder, pelvic abscess, abscess of the spleen, or phlebitis of the umbilical vein in the newborn. These usually cause a suppurative pylephlebitis from which septic emboli arise, and, entering the liver, initiate abscesses, usually multiple. The septic foci may lie outside of the portal area and the infective material be carried to the right heart by way of the superior or inferior cava, traverse the lungs and left heart, and enter the liver by way of the hepatic artery, as in general pyæmia, suppurative diseases of the bone, and suppurating wounds of the scalp; or the infection may come from the heart itself as in ulcerative endocarditis. Rarely it enters, against the blood stream, by the hepatic veins. Finally, infection may be caused by a cholangitis, due to gallstones or parasites (distoma, echinococcus, roundworms).

In the great majority of cases abscess of the liver is due to dysentery, next most frequently to appendicitis, less often to suppurating hemorrhoids, gastric ulcer, or osteomyelitis. Except when of amœbic or dysenteric origin the suppuration is usually multiple.

Symptoms.—Multiple hepatic abscesses occurring in the course of a general pyæmia may present no distinctive symptoms except an enlarged and tender liver with slight jaundice.

In general the symptoms of liver abscess comprise the following: Fever, which is often high at the outset (103° to 105°), but may begin insidiously, soon becoming irregular, intermittent, or hectic in type, and interrupted by periods of normal temperature. Chills may precede, and sweats often follow, the exacerbations of the fever. If the case becomes chronic, fever may be absent. There is usually hepatic and right-shoulder pain of a dull aching character, increased and dragging when the patient lies upon his left side. There is jaundice, seldom more than a moderate muddy yellowness of the skin and conjunctivæ. Gastric disturbances, often with alternating diarrhœa and constipation, or constipation alone, are present. Ascites is rare. If the abscess is sufficiently large the pressure upon the lung through the diaphragm may cause pleuritic symptoms. There is a progressive loss of flesh and strength.

Upon examination the liver is found to be enlarged and tender, and the enlargement, especially if a single abscess of considerable size exists in the right lobe of the liver, is upward and to the right, contrary to the findings in other swellings of the liver. The upper limit of liver dulness in such cases may lie as high as the fifth instead of the eighth rib in the midaxillary line, and at the level of the angle of the scapula posteriorly. It may project below the costal margin anteriorly as much as 4 inches, but in multiple abscesses palpable swelling may be absent. If the liver is accessible, it is found to be smooth and tender, and, rarely, fluctuation is obtained. A friction rub over the hepatic area may be heard upon deep inspiration if the perihepatic peritoneum is inflamed. In fatal cases the typhoid status usually develops.

Complications and Sequelæ.—The suppurative inflammation may involve the pleural cavity with or without perforation of the diaphragm, and invade the lung. In this case there will be a severe paroxysmal cough, with dulness, weak bronchial respiration, and increased fremitus at the right base, with the expectoration of a characteristic reddish-brown sputum, perhaps containing the *Amœba coli*. The abscess may finally rupture into a bronchus; or into the stomach or intestine, with the passage of a quantity of pus in the stools; or into the peritoneal cavity with sequent peritonitis; or very rarely into the pericardium; or point and discharge externally (after adhesions have formed) below the costal margin or in the epigastric region.

Differential Diagnosis.—The cardinal symptoms are: irregular fever with chills and sweats, hepatic pain, enlargement and tenderness of the liver, and slight icterus. In doubtful cases aspiration of the liver should be done, which, if successful, affords a reddish-brown, gray, or creamy pus containing liver cells, bile pigment, amœbæ, or cocci. Leucocytosis is always present. Except in tropical climates, hepatic abscess is almost invariably secondary, and inquiry should be made for the causative affections previously mentioned. The following conditions require to be differentiated:

(1) *Intermittent Malarial Fever.*—Liver abscess in temperate or malarial climates is usually diagnosed as malaria because of the ague-like paroxysms attending it; but the absence of the plasmodium and the futility of quinine in arresting the fever will rule out malaria. Moreover, there is no splenic enlargement in abscess, and there is usually a history of dysentery or other cause of intestinal ulceration.

(2) *Intermittent Fever of Hepatic Colic or Catarrhal Cholangitis.*—In this there is a history of previous attacks; the jaundice is more intense, and usually deepens after each paroxysm of chill, fever, and

sweat; the duration is much longer, with entire absence of fever between the paroxysms; and there is no serious impairment of the general health.

(3) *Typhoid Fever.*—The diarrhœa, delirium, rapid pulse, and other symptoms of the typhoid status which often attend the later stages of liver abscess may closely simulate typhoid fever, but chills, sweats, irregular fever, meteorism, and bronchitis are less common; rose spots and a positive Widal reaction are absent; and there is usually a preceding history of dysentery. Nevertheless, hepatic abscess may complicate typhoid fever.

(4) *Suppurative Pleurisy.*—In the cases where an empyema is due to and coexists with hepatic abscess, a correct appreciation of the true condition is impossible, unless the characteristic brownish-red sputa are present, or an examination of aspirated pus from the pleural cavity reveals liver cells and bile pigment, thus proving the hepatic origin of the empyema. The *Amœba coli* may be found in the sputum or pus. A large right-side empyema, by which the liver is pushed down and apparently enlarged, may so closely simulate liver abscess that a differential diagnosis is extremely difficult. The upper line of flatness in empyema lies at a higher point than in abscess and may change position with movement; bulging of the affected side is common; the cough and dyspnœa are more marked; there is a history of previous tuberculous disease, or a pneumonia or serous pleurisy; and the earliest symptoms are pulmonary rather than intestinal or abdominal.

Prognosis.—The mortality is from 50 to 60 per cent. Death usually occurs in from 6 weeks to 3 months. In solitary abscesses operation may give favourable results.

XIV. **Cirrhosis of the Liver.**—CAUSES.—Chronic alcoholism (50 per cent of all cases), syphilis, chronic heart or lung diseases causing passive hepatic congestion, chronic inflammation of the bile ducts or obstruction by gallstones, and other but minor causes.

VARIETIES.—Two clinical forms are recognised, the *atrophic* (or alcoholic) and the *hypertrophic.*

(I) **Atrophic (Alcoholic) Cirrhosis.**—*Symptoms.*—Occurs mainly in men, about 40 years of age or over. So long as the collateral circulation compensates for the obstruction caused by the contracting connective tissue, to the portal circulation, the disease may be latent. When passive portal congestion occurs the early symptoms are usually those of a chronic gastric catarrh, anorexia, furred tongue, nausea, and vomiting. Later, sometimes early, there may be epistaxis, hæmatemesis, or bleeding from dilated veins of the esophagus, intestinal hemorrhage, or bleeding hemorrhoids. Ascites, often becoming enormous, usually ensues, with sequent great œdema of the legs and genitals. Jaundice, if present at all, is slight. The urine is scanty and high coloured, of high specific gravity, loaded with urates, and often contains bile pigment. Fever is uncommon, but may be present (100° to 102°) during advanced stages.

The face, often ruddy at first, becomes pallid, sallow, and pinched. There is usually progressive loss of flesh, and the emaciation of the thorax and upper extremities contrasts strongly with the distended abdomen and œdematous legs. The presence of the caput Medusæ and other distended veins of the abdomen (mammary, epigastric) may be noted. The liver is at first somewhat enlarged, but later becomes smaller, although—a fact to be noted—the reduction in size may be slight. Its firm lower edge may be felt under the costal margin if the ascites will permit, and its surface is hard, sometimes finely granular. The spleen is usually enlarged and palpable. After tapping, the area of liver dulness is found to be diminished in its vertical diameter. In any stage of the disease intense headache, amaurosis, noisy delirium, convulsions, stupor, or coma may develop, closely simulating and usually supposed to be the cerebral symptoms of uræmia, but due to a toxic agent as yet unknown.

The *duration* of the disease may cover many years.

Diagnosis.—The cardinal symptoms are a history of alcoholism, the presence of ascites, and the detection of a firm, perhaps a small, liver. The occurrence of hæmatemesis or melæna and enlargement of the spleen will tend to confirm the diagnosis. It may be impossible to distinguish the initial enlargement of a cirrhotic liver from a fatty liver; and in rare instances cancer and cirrhosis are associated, as proved at autopsy. The syphilitic origin of the disease may be suspected if there is an indubitable history or unmistakable signs of previous syphilis, or if the liver is irregular in shape.

(II) **Hypertrophic Cirrhosis.**—This form occurs mainly in men under 40 years of age; occasionally in children; and there is not usually an alcoholic history (OSLER). Its duration varies from 4 to 10 years. The cause of the disease is practically unknown.

Symptoms.—There is jaundice, usually slight. The liver is uniformly increased in size and the enlargement is often visible; its edge is smooth, firm, hard, and may extend downward to the level of the umbilicus, and its surface is smooth; the spleen is enlarged, hard, and readily palpated. There are attacks of pain in the region of the liver, slight or severe, sometimes accompanied by nausea and vomiting and followed by an increase of the jaundice, but the gall bladder is not swollen. The urine contains bile pigment, but the stools remain dark. There may be slight fever, and a marked leucocytosis is not uncommon. In children the enlargement of the spleen may be very considerable. There may be hemorrhages, purpura, and itching or bronzing of the skin. At any time during the course of the disease delirium and high fever with a grave form of jaundice may develop. In contradistinction to atrophic cirrhosis, ascites and dilated abdominal veins do not occur.

Diagnosis.—In amyloid liver jaundice is absent. In carcinoma of the liver the spleen is not enlarged, there may be ascites, the patient is over 40, and the liver is nodular and irregular.

XV. **Fatty Liver.**—Two varieties are recognised, *fatty infiltration* and *fatty degeneration.*

(I) **Fatty Infiltration.**—This may be present as a part of a general obesity; or, on the other hand, because of interference with oxidation, may supervene in the course of chronic wasting diseases like pulmonary tuberculosis, carcinoma, grave anæmia, malarial cachexia, or syphilis. A fatty cirrhotic liver may be found as the result of chronic alcoholism. Aside from the evidence derived from physical examination, the general symptoms are not distinctive, being commonly those of the causative disease or condition. There is no jaundice whatever, although the stools may be pale, and ascites does not occur. The liver is found to be greatly enlarged, smooth, and painless, often reaching as far down as the umbilicus. The spleen remains of normal size.

The presence of general obesity suggests but may interfere with the physical detection of this condition. It is easily recognised in thin subjects. By comparison the amyloid liver is of firmer consistence, and usually associated with enlargement of the spleen and albuminuria. The physical characteristics of the lucæmic liver resemble those of the fatty liver, but a blood examination will readily make the discrimination.

(II) **Fatty Degeneration.**—May be due to alcoholism, carcinoma, phthisis, chronic dysentery, profound anæmia, acute specific infections, poisoning by phosphorus, arsenic, or chloroform, and forms an essential feature of acute yellow atrophy. The slighter degrees of fatty degeneration are clinically unrecognisable. The symptoms of the grave forms are those of acute yellow atrophy (page 900) or phosphorus poisoning.

XVI. **Amyloid Liver.**—Occurs most frequently as a result of chronic suppuration in tuberculous disease of the bones, especially of the hip joint or vertebræ; next most commonly it is due to syphilis, particularly syphilitic ulceration of the rectum and disease of the bones. Less often it is associated with rachitis, carcinoma, and infectious fevers. The *symptoms* are not distinctive. The liver is uniformly and greatly enlarged, smooth, solid, firm, and not tender. The margin is usually rounded, but may be sharp and firm. The spleen also is enlarged from associated amyloid disease. Jaundice is absent, the stools may be light coloured but contain bile, and there is no ascites or other evidence of portal obstruction. As an amyloid kidney usually coexists, the urine often contains serum albumin, and also serum globulin, with abundant casts.

The *diagnosis*, except in the rare instances when the amyloid liver is not enlarged, is readily made. The cardinal symptoms comprise a great and steady enlargement of the organ, in conjunction with chronic suppuration, syphilis, or chronic phthisis.

XVII. **Carcinoma of the Liver.**—This occurs most commonly in men, seldom under 40 years of age, very rarely in children. It is next in frequency to carcinoma of the stomach and uterus; usually secondary, mainly to primary disease in the portal territory, especially of the stomach and rectum, or to mammary carcinoma. Carcinoma of the gall bladder has already been described (page 889).

(*a*) *Symptoms.*—The disease may be latent except for an indefinite ill health. Progressive emaciation and loss of strength may be the earliest symptoms. There are usually anorexia, nausea, and vomiting. Jaundice, ordinarily slight, sometimes intense, is present in 50 per cent of cases. Dull pain or uneasiness in the right hypochondrium or shoulder is usually present, but may be lacking. Ascites is not common, occurring in but a small proportion of cases. The spleen is seldom enlarged. Fever, usually moderate (100° to 102°), is not infrequent during the later stages; rarely chills and high intermittent fever may occur. A marked cachexia with anæmic œdema of the feet and legs almost invariably appears toward the end of the disease; and at the same period toxic symptoms, headache, delirium, stupor or coma, may supervene.

Physical examination shows enlargement of the superficial veins and distention of the upper portion of the abdomen. If the emaciation is marked, the nodular character of the enlarged liver may be evident by inspection. The organ is found to be greatly increased in size, its margin lying perhaps below the navel, and moving with respiration. Usually the liver is hard, irregular, and nodulated, each nodule sometimes presenting a characteristic depression or umbilication in its centre. When the growth is mainly in the left lobe the latter may resemble a distinct epigastric tumour. In rare cases the liver is uniformly enlarged and smooth, lacking nodulation. The *duration* of the disease is from 3 to 15 months, rarely 2 years.

(*b*) *Differential Diagnosis.*—The cardinal symptom of hepatic carcinoma is the enlarged nodular liver with cachexia. Previous or present carcinoma of other organs (stomach, intestine, rectum, mammary gland), with jaundice and perhaps ascites, confirm the diagnosis. Age and heredity are suggestive.

(1) If the liver is diffusely carcinomatous and the enlargement is smooth and uniform, a variety sometimes encountered, it may require differentiation from amyloid or fatty liver. The presence of jaundice, the rapid increase in the size of the organ, and the development of a marked cachexia will pronounce for malignant disease.

(2) The large nodulated hydatid liver may resemble that of carcinoma, but in the former the duration of the disease is much longer, the wasting and anæmia are much less marked, the nodules are softer, jaundice is more common, aspiration may enable the finding of hooklets, and finally it is of rare occurrence.

(3) The large syphilitic amyloid liver containing irregular or rounded projecting gummata may be difficult to separate, as the jaundice may be marked and the organ greatly enlarged. But the history or evidences of syphilis, the longer duration of the disease, and the slighter degree of impairment of the general health will negative malignant disease.

(4) Hypertrophic cirrhosis in the early stages can not always be distinguished from carcinoma. Reliance must be placed upon smooth and painless enlargement of the liver, the presence of an enlarged spleen, and the non-appearance of the cancerous cachexia.

(5) Whether the malignant disease of the liver is sarcoma rather than carcinoma, as sometimes happens, can not be decided unless there is a primary sarcoma elsewhere. The primary growth is usually a melano-sarcoma of the eye, lymph glands, or skin, and if secondary deposits occur in the liver there may be melanuria, which, with the presence of the original tumour and the very rapid increase in the size of the liver, may enable a diagnosis.

It is hardly possible to determine positively whether carcinoma of the liver is primary or, as usual, secondary, unless the primary growth can be discovered.

XVIII. **Perihepatitis.**—Reference is here made to a chronic inflammation, with great thickening (capsulitis) of the fibrous envelope (Glisson's capsule) of the liver. Acute inflammation of the peritoneal covering of the liver, including subphrenic abscess, is described under diseases of the peritoneum (*q. v.*).

Chronic capsulitis (Glissonian cirrhosis) is divided (OSLER) into two groups: one, in adults, with recurring ascites and evidences of interstitial nephritis, without jaundice, and which can not be distinguished from atrophic cirrhosis of the liver; the other forming a part of a widespread fibroid process (multiple serositis) which includes perihepatitis, perisplenitis, proliferative peritonitis, adherent pericardium, and indurative mediastinitis. The liver may be rounded and smooth, resembling the spleen, and there is persistent ascites.

XIX. **Acute Yellow Atrophy.**—A rare disease attended by a rapid necrosis of the liver cells. It occurs with the greatest frequency in pregnant women between 20 and 30 years of age. The condition is almost exactly that which is produced by phosphorus poisoning.

Symptoms.—The initial symptoms are malaise, headache, anorexia, nausea, and vomiting, followed in a few days by jaundice, the condition resembling a simple gastro-duodenal catarrh. In from 1 to 3 weeks from the onset cerebral symptoms, delirium, muscular trembling, perhaps convulsions, drowsiness, coma, and persistent vomiting become manifest. At this time the jaundice deepens. There may be subcutaneous or mucous-membrane hemorrhages. There may or may not be moderate fever. There is no bile in the clay-coloured stools; the urine is bile-stained, contains albumin, fatty casts, and quite frequently leucin and tyrosin. The typhoid status usually develops with dry tongue, rapid pulse, coma, and death. Physical examination shows a progressive decrease in the area of hepatic dulness, and its replacement by tympanicity.

Diagnosis.—The cardinal symptoms are jaundice, vomiting, delirium, hemorrhages, with leucin and tyrosin in the urine, and atrophy of the liver. It is to be remembered that severe cerebral symptoms may occur during the course of any grave jaundice.

In *phosphorus poisoning*, which yellow atrophy resembles, the onset is sudden, the gastric symptoms more prominent, the nervous symptoms are less marked and appear at a later period, and the urine does not contain leucin or tyrosin. Moreover, there may be a history of the ingestion of rat paste or match heads.

The symptoms of *hypertrophic cirrhosis* may, in some respects, exactly simulate those of yellow atrophy, but enlargement instead of shrinkage of the liver, the absence of leucin and tyrosin, and the frequent pyrexia will speak for the former.

XX. **Syphilis of the Liver.**—See page 822.

XXI. **Leucæmic Liver.**—See Index.

XXII. **Hydatids of the Liver.**—See Index.

X. DISEASES OF THE PANCREAS

(For the examination of the pancreas, see pages 506 and 507)

I. **Acute Pancreatitis.**—Of this 3 forms are recognised: *hemorrhagic*, *suppurative*, and *gangrenous*.

(I) **Hemorrhagic Pancreatitis.**—Occurs mainly in adult males. Predisposing causes are injury, alcoholism, gallstones in the diverticulum of Vater, severe chronic gastro-duodenitis, and chronic mercurialism.

(*a*) *Symptoms.*—The onset is sudden, with deep-seated violent, paroxysmal pain in the upper abdomen, followed by persistent vomiting, constipation, and abdominal distention, perhaps limited to the epigastrium. The temperature may be subnormal at first, later there is moderate fever, perhaps beginning with a chill. Deep pressure over the upper abdomen may reveal circumscribed resistance, and there is well-marked tenderness between the ensiform and umbilicus. Tender points (fat necrosis) may be found scattered over the abdominal wall. Delirium, dyspnœa, cyanosis, hiccough, fatty diarrhœa, and albuminuria may be present. The fat-spitting ferment may be found in the urine. Collapse rapidly supervenes, and death occurs from the 2d to the 4th day of the disease.

(*b*) *Differential Diagnosis.*—The cardinal symptoms (FITZ) comprise a sudden, violent, deep-seated pain in the epigastrium, followed by vomiting and collapse, and in 24 hours by a circumscribed epigastric swelling, tympanitic or resistant, with slight fever, constipation, tenderness over the course of the pancreas, and tender spots in the abdomen. Few correct diagnoses are made *intra vitam.* Commonly the disease is mistaken for acute intestinal obstruction, or a perforation-peritonitis. Symptoms pointing to obstruction are general abdominal distention, visible peristalsis of the intestinal coils, obstipation, and fæcal vomiting, although obstruction manifesting itself by local signs in the epigastrium is not common. Ulcer of the stomach or duodenum occurs usually in younger persons, with a history of anæmia, pain after eating, and hæmatemesis or melæna. In gallstone colic there is seldom severe collapse or prostration, the pain is in the right hypochondrium, and jaundice is often present; but gallstones and pancreatitis may coexist. A ruptured phlegmonous cholecystitis will present a history of a previous tumefaction beneath the 9th right costal cartilage. In fulminating appendicitis, while the pain often begins in the epigastrium, the physical signs are in the right iliac fossa. The *prognosis* is always bad.

(II) **Suppurative Pancreatitis.**—Occurs mainly in men. The disease may set in acutely with severe epigastric pain, hiccough, vomiting, chills and irregular fever, tympanites, constipation, slight jaundice, and splenic swelling, ending fatally within a week; or it may continue for 3 or 4 weeks with irregular chills and fever, and steady loss of flesh and strength, finally ending in death; or it may become chronic, lasting several months or a year, with progressive weakness and emaciation, and either continuous moderate fever or occasional attacks of pain and vomiting, followed by fever and delirium. There may be fatty diarrhœa, jaundice, and glycosuria. The abscess may perforate into the peritoneal cavity, stomach, or duodenum, or cause portal thrombosis. The *diagnosis* of pancreatic abscess can not be made unless it forms a palpable, deep-seated mass in the epigastrium, the presence of which, in association with the symptoms described, may, in rare instances, suggest the diagnosis.

(III) **Gangrenous Pancreatitis.**—This is usually a sequel of hemorrhage (see II, following), or of (I) and (II) just described, the pancreas becoming necrotic. The symptoms are the same as those of hemorrhagic pancreatitis, except that chills and fever are usual, jaundice may occur, and the duration is from 10 days to 3 weeks. Death is the ordinary termination, although the necrotic pancreas has been discharged by way of the rectum with subsequent recovery.

II. **Hemorrhage into the Pancreas.**—Occurs mainly in adults over 40 years of age. The *symptoms* comprise a sudden onset, during perfect health, of severe, sharp, or colicky pain in the upper abdomen, accompanied by nausea and obstinate vomiting. The patient rapidly becomes depressed, restless, and anxious, with a cold, sweating skin. The pulse is small and rapid, becoming later running and imperceptible. The temperature is normal or subnormal, the abdomen becomes distended and tender, especially over its upper portion. Collapse, syncope, and death follow within 24 hours.

III. **Chronic Pancreatitis.**—The organ becomes hard, and often contracted, as a result of interstitial fibrous overgrowth. The most frequent cause is an extension into the pancreatic duct of a chronic gastro-duodenitis or catarrh of the bile passages; next most frequently it is a result of alcoholism and syphilis. It is often associated with diabetes.

The *symptoms* are not distinctive. There may be evidences of chronic catarrhal gastritis with occasional attacks of deep-seated pain in the epigastric region, faintness, anxiety, and moderate fever. Jaundice, due to pressure upon the common bile duct by the fibroid changes in the head of the pancreas, may be present, so also fatty diarrhœa, and fat and sugar in the urine. A sense of resistance over the epigastrium has been observed. Nevertheless an *ante-mortem* diagnosis is rarely, if ever, possible without exploratory operation.

IV. **Carcinoma of the Pancreas.**—This occurs principally in men over 40 years of age; is usually primary; and most commonly involves the head of the organ. It is not a frequent disease.

Symptoms.—The symptoms are not distinctive. There is continuous dull, occasionally paroxysmal and radiating, pain in the epigastrium. Nausea, vomiting, and dyspeptic symptoms are common.

From pressure by the enlarged head of the pancreas upon the end of the common duct intense and permanent jaundice may arise, with swelling of the liver and gall bladder; upon the portal vein, ascites; upon the thoracic duct, chylous ascites; upon the pylorus, gastrectasia; upon the inferior cava, œdema of the lower half of the body; upon the left ureter, hydronephrosis. The stools are clay coloured, and fatty diarrhœa and diabetes may be present but are not common. There is very rapid emaciation and cachexia. In about one third of the cases a deep-seated epigastric tumour can be felt, which lying, as it does, directly upon the aorta, may present a distinctly transmitted pulsation, perhaps a *bruit*. The *prognosis* is almost invariably unfavourable, but cases have recovered after operation.

Diagnosis.—This is difficult and not often made. The significant symptoms are epigastric pain, rapid emaciation, the late onset of intense and permanent jaundice with dilatation of the gall bladder, and the presence of a deep-seated, immovable, and hard epigastric tumour. It requires to be separated from the following:

(1) *Carcinoma of the Pylorus.*—Compared to carcinoma of the pancreas, a pyloric growth is readily movable; the stomach is usually dilated; there may be coffee-ground vomitus and melæna; jaundice, ascites, glycosuria, and fatty diarrhœa are generally absent; HCl is absent, lactic acid present, in the stomach contents; and, finally, the duration is much longer than that of carcinoma of the pancreas.

(2) *Carcinoma of the Transverse Colon.*—In this the tumour is not so deep-seated, it is movable, evidences of intestinal obstruction are generally present, and there is no jaundice.

(3) *Aneurism of the Abdominal Aorta.*—The history, the expansile pulsation, and the absence of cachexia, will vouch for aneurism rather than carcinoma.

V. **Pancreatic Cysts.**—(*a*) *Causes.*—These cysts are usually either traumatic, inflammatory, or retention cysts; the latter due to plugging of the duct of Wirsung by calculi, or occlusion of the smaller ducts by the contraction of a chronic interstitial pancreatitis.

(*b*) *Symptoms.*—In the traumatic cases the onset may be sudden, with pain, vomiting, and peritonitic symptoms; in inflammatory cases either gradual after dyspeptic attacks with colicky pain suggestive of gallstones, or more rapid with symptoms of intestinal obstruction. The more chronic retention cysts may give rise to no symptom until they attain a very considerable size.

Frequently there are attacks of colicky pain, sometimes referred to the left hypochondrium and left shoulder, with nausea, vomiting, and steady enlargement of the abdomen. Fatty diarrhœa and salivation are rare, glycosuria and albuminuria not infrequent, and from the pressure of large tumours jaundice, ascites, and dyspnœa may become manifest. A decided loss of weight has been noted in a number of cases. Constipation is common, and there may be recurring intestinal hemorrhages.

The elastic, perhaps fluctuating, smooth, or lobulated cystic tumour is found in the upper abdomen, usually in the middle line. In the majority of cases it reaches the abdominal wall between the stomach and the colon, the former having been thrust upward. More rarely it lies above the stomach or below the colon; still more infrequently, when springing from the tail of the pancreas, it is discovered in the left hypochondrium. It is fixed, moves slightly, if at all, with respiration, and may be so large as to fill the abdomen. The cyst may develop rapidly, but as a rule the growth is slow and chronic, and it may exist for many years, in the majority for from 2 to 4 years.

(*c*) *Diagnosis.*—This rests upon the finding of a tumour with the characteristics just described. The cyst may be aspirated and the contents examined. Inflation of the stomach and the colon will aid in determining the exact location of the cyst. It requires differentiation from hydronephrosis (*q. v.*) or renal cysts (*q. v.*), a greatly dilated gall bladder (page 503), or, if very large, from an ovarian cyst. The *prognosis*, if the condition is correctly diagnosed and operated, is good.

VI. **Pancreatic Calculi.**—These rare formations may lead by their presence to chronic pancreatitis, cysts, suppurative pancreatitis, or carcinoma. A *diagnosis* may be attempted if, without jaundice, there are attacks of colic, due to the passage of the calculus through the main duct, the pain extending along the left costal margin and through to the back, with glycosuria and fatty diarrhœa. If calculi composed of calcium carbonate or phosphate can be recovered from the stools subsequent to the attack the diagnosis is confirmed. Ordinarily a diagnosis of gallstone colic is made.

XI. DISEASES OF THE PERITONEUM

I. **Acute Diffuse Peritonitis.**—**Causes.**—Rarely this is primary or idiopathic, occurring as a terminal event in chronic nephritis, arteriosclerosis, or gout. Ordinarily it is secondary, arising by extension from an inflamed organ covered by peritoneum, or from perforation of a similarly covered hollow organ, or from rupture of an abscess into the peritoneal cavity. Peritonitis from *perforation* may be due to typhoid, cancerous, tuberculous, simple, or stercoral ulcer of the stomach or intestine, or to perforation of the appendix or gall bladder; from *rupture of a purulent collection*, to appendical abscess, pyosalpinx, ovarian abscess, retroperitoneal abscess, empye ma of the gall bladder, and abscess of the liver, spleen, or pancreas. It may arise *by extension* from any of the inflammations just mentioned, without perforation or rupture. It may form a part of septicæmia or pyæmia, or be due to cancer or tuberculosis of the abdominal viscera. The infective organisms are various, but those of most frequent occurrence are the *Bacillus coli communis* (in peritonitis from perforation of the intestinal tract), the *Staphylococcus aureus* (post-operative and puerperal), and the *Streptococcus pyogenes* (idiopathic or terminal).

Symptoms.—If the patient is already gravely ill, particularly when a stuporous condition is present, or when a localized peritoneal abscess causes a slow-spreading general peritonitis, the onset may be gradual and insidious, the symptoms of the primary disease overshadowing those of the peritoneal involvement.

Ordinarily the initial symptom is a chilly feeling or a marked rigour, with a rise of temperature, vomiting, and intense abdominal pain. The pain may at first be local and correspond to the seat of the primary lesion, but soon becomes diffused and general. Except when due to perforation of a gastric ulcer, when it is referred to the chest, back, or shoulder, the greatest pain is below the navel. The abdomen is excessively tender, soon becomes distended and tympanitic, in rare cases remaining flat and rigid, and the abdominal muscles are firmly contracted. As the pain is increased by pressure or movement, the patient lies upon the back with the knees drawn up, in order to minimize the tension of the abdominal walls; the respiration is costal, and talking, coughing, vomiting, and straining to empty the bladder or bowel are avoided, so far as possible, because contraction of the diaphragm is painful. The vomiting is usually persistent, first of the stomach contents, then of yellowish bile-containing fluid, finally, of a greenish fluid. Very seldom the vomitus is brownish black, with an odour suggestive of a fæcal origin. Constipation is usual, but a brief initial diarrhœa may occur. The pulse is rapid (110 to 160), small, often wiry; the respirations are accelerated (30 to 40). In extremely severe cases the temperature may be normal or subnormal; ordinarily it is moderately elevated; exceptionally, and especially at the onset, it may be high (104° to 105°), with a cool surface. The urine is scanty, high coloured, and contains a large excess of indican. There may be frequent urination, less commonly retention. More or less marked symptoms of collapse often become manifest at an early period. The gray face bears an expression of anxiety, the nose is pinched, the eyes sunken, the cheeks collapsed, and the lips cyanotic. The skin is cool and clammy, the hands and feet cold and wet.

Physical examination reveals in the majority of cases a rigid, motionless, greatly distended abdomen, universally tympanitic on percussion. In rare instances, particularly if the abdominal muscles are strong and well-developed, there may be a flat and rigid abdomen throughout the course of the disease. Prolonged auscultation shows, in consequence of intestinal paresis, an absence of the usual gurgling or splashing sounds, and possibly the presence of friction sounds during respiration. If the meteorism is sufficiently great the splenic dulness is obliterated; so also with hepatic dulness, except in the midaxillary line, at which point it may still be found. If the remaining hepatic dulness disappears when the patient is turned on the left side the presence of free air or gas in the peritoneal cavity (pneumo-peritoneum) may be inferred. Owing to the upward pressure upon the diaphragm the apex beat may be found in the 4th interspace, farther to the left than normal. If the patient lives long enough the signs of peritoneal effusion become manifest.

Duration and Prognosis.—Usually terminates in death from exhaustion, the duration varying, according to acuteness and severity, from 2 to 10 days. Cardiac paralysis is occasionally responsible for a sudden lethal ending.

Diagnosis.—The cardinal symptoms of a classical case are sudden severe abdominal pain, increasing abdominal distention, tenderness, and gradual effusion, with persistent vomiting, fever, and symptoms of collapse. Always suspect and look for evidences of appendicitis, especially in young adults; for puerperal infection, gonorrhœa, salpingitis, ovarian or pelvic abscess, in women; for perforated gastric ulcer; and for walking typhoid fever with perforation, especially in young and vigorous persons.

The diagnosis is at times extremely difficult if, as may happen in typhoid fever, or when the patient is stuporous or comatose, the symptoms are insidious and not very distinctive. There may be simply an increase of the already existing meteorism, a more marked tenderness, and an intensification of the evidences of collapse or prostration, with perhaps a higher level of temperature. Occasional errors, both positive and negative, are inevitable. The following diseases may require differentiation:

(1) *Acute Entero-colitis.*—See page 862.

(2) *Intestinal Obstruction.*—See page 878.

(3) *Embolism of the Superior Mesenteric Artery.*—See page 881.

(4) *Acute Hemorrhagic Pancreatitis.*—See page 901.

(5) *Rupture of an Ectopic Gestation.*—See page 871.

(6) "*Hysterical Peritonitis.*"—This neurosis, which in several personal cases has been one of the varying features of mucous colic

(page 883), may exactly simulate true diffuse peritonitis in its abrupt onset with severe abdominal pain and rigidity, great tenderness, and marked meteorism, with vomiting, diarrhœa, frequent urination, and even evidences of collapse. It is said that fever may also be present. If characteristic hysterical symptoms are associated the diagnosis is ordinarily clear. Otherwise the usual absence of fever, the exaggerated intensity of the local signs in comparison with the general condition, and a final recovery, will suggest the neurotic nature of the affection. Recurrences may take place. Nevertheless mistakes will occur, at least in the first attack.

II. **Acute Localized Peritonitis.**—The symptoms of circumscribed peritonitis resemble those of the diffuse form except in degree. The pain and tenderness is limited to the neighbourhood of that part of the peritoneum which is involved; the vomiting and meteorism may be slight or absent; constipation is usually present, but is easily overcome; and the collapse symptoms are moderate or slight. If, as often happens, the inflammatory focus is walled off from the general peritoneal cavity by protective adhesions, the constitutional symptoms may disappear and the local signs be reduced to those indicative of adhesions or of a thick-walled pus sac—an encapsulated abscess. The three varieties of localized peritonitis which are of especial interest to the student of internal medicine are appendicular abscess (page 868), pelvic peritonitis from salpingitis or ovaritis (page 871), and *subphrenic peritonitis.*

Subphrenic Peritonitis.—An inflammation, usually suppurative, of the peritoneum covering the right and left lobes of the liver, or of the lesser cavity of the peritoneum, together with that of the adjacent portions of the diaphragm.

(*a*) *Causes.*—The majority of subphrenic abscesses are due to perforation of a gastric ulcer, next most commonly to the upward extension of an appendical inflammation, then to perforation of a duodenal ulcer. Less frequently the origin is from extension of a pneumonic infection or perforation of an empyema through the diaphragm, malignant disease of the stomach or liver, rupture of an hepatic, perinephritic, or pancreatic abscess, diseases of the gall bladder, or trauma. The majority of abscesses due to perforation of a gastric or duodenal ulcer contain air, forming a subphrenic pyopneumothorax.

(*b*) *Symptoms.*—As the great majority of cases are due to perforation of a gastric ulcer the onset is usually abrupt, with severe epigastric or hypochondriac pain and tenderness; vomiting usually of bile-stained, sometimes of bloody, fluid; and rapid, embarrassed, or painful respiration. Soon afterward the symptoms indicative of suppuration become manifest—chills, sweats, irregular fever, and loss of flesh and strength. At a later period the abscess may perforate the diaphragm into the pleural cavity and establish a communication with a bronchus, an event indicated by severe and paroxysmal cough and profuse purulent expectoration.

The *physical signs* are very often perplexing. Ordinarily they simulate those of an empyema. When the abscess is on the *right side*, lying between the liver and diaphragm, there may be visible bulging and deficient mobility of the right hypochondriac, and sometimes of the epigastric, region. The liver is pushed downward, its lower edge reaching even to the level of the navel. If the abscess does not contain air there is an apparent vertical and upward increase of hepatic dulness, perhaps to the 4th rib, above which is normal or slightly tympanitic pulmonary resonance. If the abscess contains air there will be a zone of tympanitic percussion between the area of dulness (or flatness) and the area of pulmonary resonance; a change in the posture of the patient will alter the position of the line of flatness; and a succussion sound, limited to the subdiaphragmatic area (JANEWAY), may be heard upon shaking the thorax. There is an absence of respiratory murmur, voice sounds, and vocal fremitus over the area of dulness or of tympanicity; while over the lung, which is compressed by pressure transmitted through the diaphragm, the respiration may be normal, broncho-vesicular, or even bronchial in quality. Friction sounds, due to an associated dry or fibrinous pleurisy, may be heard over the complementary pleura. These physical signs may lie at and be limited to a lower level of the thorax than one would expect in an empyema, a finding which, if present, is a somewhat suggestive fact from a diagnostic point of view; but if there is a large amount of air in the abscess the diaphragm may be pushed up to the 3d or even to the 2d rib, with physical signs exactly like those of pneumothorax (*q. v.*).

When the abscess is contained in the lesser peritoneal cavity and lies between the diaphragm above and the spleen, stomach, and left lobe of the liver below, which is the case in the large majority of subphrenic abscesses caused by perforation of the stomach or duodenum, the physical signs are upon the *left side*. If the abscess contains air the signs are exactly those of a left pneumothorax.

If the lesser cavity contains a large quantity of pus (or other fluid), a tumour may be formed in the left hypochondriac, epigastric, and umbilical regions. The colon invariably lies below, never above or in front of the tumour. The tumour apparently changes in size and shape, according to the character of the contents of the stomach, which latter viscus lies between the fluid collection and the anterior abdominal wall. If the stomach contains fluid, the size and area of dulness of the tumour are apparently increased; if it is distended with gas, the dulness is replaced by a tympanitic percussion sound and the tumour may elude palpation.

Occasionally milder, fibrinous and non-suppurative, perihepatic peritonitis may be met with, occurring in the course of acute or chronic inflammations of the liver, or a pleurisy, or following a blow, indicated by localized moderate pain and tenderness, with friction sounds over the epigastrium or in the right hypochondrium, corresponding to the exposed area of hepatic dulness.

(*c*) *Diagnosis.*—The condition is obscure and often escapes recognition. It is usually diagnosed as an empyema, pneumothorax, or pyopneumothorax. Effusion into the lesser peritoneum is frequently judged to be a cyst of the pancreas.

Information of value may be gleaned from the history. If the *earliest* symptoms are (upper) abdominal in location and character (severe pain, vomiting), the presumption leans toward a subphrenic inflammation; if thoracic (cough, pleuritic pain) it points toward an empyema. The latter, however, may cause the former by perforation, or *per contra.* In suspected cases of subphrenic abscess aspiration should be done in the 7th or 8th space in the midaxillary line. It is stated that if the fluid flows more freely during inspiration it is indicative of its location below the diaphragm, the descent of which during inspiration increases the intra-abdominal pressure while producing a negative intrathoracic pressure.

(*d*) *Terminations and Prognosis.*—The prognosis of subphrenic abscess is very grave, but depends largely upon an early recognition of the condition and prompt operation. In rare cases the pus discharges by way of the abdominal wall or the lungs, or still more infrequently undergoes absorptive and other changes.

III. **Chronic Peritonitis.**—Of this there are several varieties: *adhesive*; *proliferative*, in which the peritoneum is greatly thickened, with but little adhesion; *cancerous*; *tuberculous*, and *hemorrhagic.* The process may be local or general.

(I) **Chronic Local Peritonitis.**—This is usually of the adhesive variety, occurring as a result of acute or chronic inflammation of the abdominal viscera, mainly of the spleen, liver, intestines (especially the appendix), or mesentery; or of the pelvic organs. The points of attachment of the adhesions, which may be short, or long and bandlike, vary according to the locality and the viscus affected. Thus the spleen and liver may be adherent to the diaphragm, and coils of intestine to each other, to the abdominal wall, or to the mesentery. In the greater proportion of cases no *symptoms* arise. Intestinal adhesions may, however, cause internal strangulation and obstruction, or give rise to more or less constant and severe colicky pain. The latter condition, if correctly diagnosed, usually by exploratory operation, may be relieved by separation of the adhesions.

(II) **Chronic Diffuse Peritonitis.**—*Causes.*—Most commonly tuberculous; less often a sequel of acute simple inflammation; rarely cancerous; or may arise from chronic cardiac, hepatic, or intestinal disease. The proliferative form is most frequently associated with chronic alcoholism, occasionally with chronic nephritis.

Symptoms.—For those of tuberculous peritonitis, see page 812.

The disease may be entirely latent. The symptoms are often obscure and indefinite. There may be vague abdominal discomfort, burning sensations, or actual colicky pain, either with constipation or diarrhœa. There may be irregular slight fever, with loss of flesh and strength. More or less ascites, or one or more collections of encysted fluid, may be present; and occasionally there is jaundice from pressure upon the common bile duct. As in acute peritonitis, but in lesser degree, there may be abdominal distention with rigidity. Sacs of fluid and adherent intestinal coils may cause tumourlike masses; and the omentum may be rolled and puckered into a transverse cylindrical mass between the stomach and the colon. The physical signs are obviously variable, and the exact significance of the dull or tympanitic, ill-defined swellings, which may be found, is often difficult to determine.

IV. **Carcinoma of the Peritoneum.**—This is usually secondary to malignant disease of the liver, stomach, or pelvic viscera; much less frequently it is primary.

Symptoms.—These are a persistent ascites with loss of flesh and cachexia. Fever may or may not be present. If the abdominal effusion is moderate or is removed by tapping, multiple, somewhat large, irregular nodules may be found by palpation; so also the transverse roll of puckered omentum, although this occurs as well in tuberculous and proliferative peritonitis. Secondary umbilical nodules or hardening may be present, and the inguinal glands may be implicated. The fluid may be hemorrhagic and contain significant cells or cell groups.

Diagnosis.—If there is primary malignant disease of the stomach, liver, uterus, ovaries, or rectum (for which search should always be made), the nature of the peritoneal involvement is evident. If, however, the peritoneum is primarily affected, and no antecedent local carcinoma can be found, the diagnosis becomes difficult or impossible, as the physical signs previously described are common to chronic peritonitis, whether carcinomatous, tuberculous, or proliferative, or to hydatids of the peritoneum. *Carcinomatous disease* occurs usually in persons past middle life; there is a marked cachexia; and nodules or indurations about the navel, or enlargement and induration of the inguinal glands, point to carcinoma. *Tuberculous peritonitis* occurs mainly in children or before middle life; the cachexia is not marked, suppurative inflammation of the navel, with a fistulous opening, is more common than induration, and evidences of tuberculous disease may be found elsewhere. *Hydatids of the peritoneum* may exactly simulate the numerous nodules of carcinoma, and, unless hydatid fremitus can be obtained or the aspirated fluid contains hooklets, the diagnosis may be difficult or impossible.

V. **Ascites.**—*Causes.*—Pressure upon the end branches of the portal vein within the liver, as in hepatic cirrhosis, syphilis, carcinoma, or chronic passive congestion; or upon the vein, before it enters the liver, by new growths, carcinoma, abscess, or chronic peritonitis involving the transverse fissure or gastro-hepatic omentum; or pressure by aneurism, abdominal or ovarian tumours, or the enlarged spleen of leucæmia or malaria; and chronic simple, tuberculous, or carcinomatous peritonitis. Ascites may be a part of a general œdema due to chronic cardiac disease, pulmonary cirrhosis or emphysema, nephritis, anæmia, or the malarial, cancerous, or syphilitic cachexiæ. It is extremely rare with uterine fibroids.

Character of the Fluid.—The fluid may be hemorrhagic in tuberculosis and carcinoma of the peritoneum, occasionally in cirrhosis. In non-inflammatory ascites the fluid usually has a sp. g. of 1.015 or below, with 2.5 per cent or less of albumin; in peritonitis, 1.018 or over, with 4.5 per cent or more of albumin. Chylous or chyloid ascites (page 715) occurs in carcinoma, perforation of the thoracic duct, filariasis, and perhaps under an exclusive milk diet (OSLER).

Diagnosis.—See page 465.

VI. **Retroperitoneal Sarcoma.**—According to the summary by Steele, Lobstein's cancer occurs more frequently in males (6 to 4), either during the first 10 years of life or between 40 and 50 years of age. In 90 per cent of the cases the tumour (rarely larger than a man's head) originates in the lumbar region or the central portion of the posterior abdominal wall at the attachment of the mesentery.

(*a*) *Symptoms.*—These at first consist of indefinite digestive disturbances, such as constipation, diarrhœa, nausea, anorexia, colicky pains, or dragging sensations in the abdomen. Later there are œdema and neuralgic pains in the legs, genitalia, abdominal walls, and lumbar region (at first unilateral), and due to pressure upon the iliac veins and the sacral and lumbar plexuses. Finally, cachexia and the evidences of partial or complete obstruction of the small intestine become manifest.

Physical Signs.—At first it is simply possible to recognise the presence of a deep-seated tumour, but as the growth increases in size the colon (inflated, if necessary) will be recognised to lie upon its anterior surface. The tumour may be central, lying in mid-abdomen, or be found to the right or left of the median line, and may fluctuate and be movable or move with respiration, but is ordinarily immovable and solid. In central tumours there is a dull area surrounded by a zone of tympany.

(*b*) *Diagnosis.*—Œdema of the legs and neuralgic pain in the legs and lumbar region, evidences of intestinal obstruction, the finding of a tumour at or to one side of the navel, and the presence of the colon over the anterior surface of the neoplasm, indicate the retroperitoneal origin of the latter. The rapid growth of the tumour (average duration between 8 and 9 months) and the appearance of cachexia indicate its malignant nature. It is often impossible, especially in the later stages, to distinguish retroperitoneal sarcoma from tumours of the kidney and suprarenal capsules or other growths lying behind the peritoneum, except by an exploratory incision.

SECTION III

DISEASES OF THE RESPIRATORY SYSTEM

(See, in Part I, Larynx (pages 255 to 260); Voice (pages 260 to 263); Cough (pages 273 to 276); Sputum (pages 276 to 280 and pages 650 to 657); Physical Examination of Lungs and Pleuræ (pages 411 to 454)

I. DISEASES OF THE NOSE

(See also pages 228 to 235)

I. **Acute Nasal Catarrh.**—(*a*) *Causes.*—An acute coryza or rhinitis may be an initial symptom of an infection like influenza or measles; more commonly it is primary—a "cold in the head." Its epidemic and contagious character is so marked that it probably depends upon germ infection. The chief predisposing causes are exposure to cold, variable weather, and inhalation of irritating vapours.

(*b*) *Symptoms.*—There is chilliness, headache, slight fever (100° to 101°), and sneezing, with quickened pulse, dry skin, and thirst. Some backache and general aching are not uncommon. The nasal mucous membrane swells so that mouth-breathing is imperative, and there is a thin acrid discharge from the nostrils. The eyes water, the senses of smell and taste are impaired, the pharynx is reddened, the throat is sore and the neck stiff, and slight dysphagia may be present. Herpes of the nose and lips is common. The larynx may be involved, causing hoarseness; the trachea and bronchi, cough; the Eustachian tubes, slight deafness. In a day or two the nasal discharge increases, becomes thicker and muco-purulent, and in 5 or 6 days the swelling of the mucosa and the associated symptoms subside. The coryzal discharge persists for a week or two longer.

(*c*) *Diagnosis.*—Ordinarily easy, but the possibility that it is the initial coryza of measles or influenza is to be borne in mind.

II. **Chronic Nasal Catarrh.**—Caused by recurring attacks of acute coryza, syphilis, rarely tuberculosis. Three forms may be recognised: simple, hypertrophic, and atrophic.

Symptoms.—(*a*) *Simple Chronic Catarrh.*—There is a special liability to "catching cold," the mucous membranes readily become congested and swollen, with consequent occasional stenosis, and there is an overabundant thick secretion. If this condition persists it becomes:

(*b*) *Hypertrophic Rhinitis.*—The lower turbinals are swollen and enlarged, there is constant hawking to remove the thick secretion from the upper pharynx, and the patient becomes a mouth-breather to a varying extent. In the majority of cases the pharyngeal mucosa and adenoid tissues are coincidently affected, constituting a chronic naso-pharyngeal catarrh. The voice becomes nasal, and varying degrees of deafness are common. (See also Mouth-Breathing, page 178; Adenoids, page 840; and Chronic Pharyngitis, page 838.

(*c*) *Atrophic Rhinitis.*—This may be, but is by no means necessarily, a sequence of the hypertrophic form. The horrible and disgusting odour (*ozena*), which is the principal symptom of the disease, is met with also as an evidence of syphilis, disease of the nasal bones, glanders, and foreign bodies. The sense of smell is abolished. On inspection, the nasal mucosa is seen to be shrunken and atrophied, with a resultant unusual roominess of the nasal chambers. The thick purulent secretion coating the membrane dries into yellowish-green adherent crusts, which emit the offensive odour.

III. **Hay Fever.**—(*a*) *Causes.*—A neurotic idiosyncrasy must be predicated, together with an unusually irritable nasal mucosa. The exciting causes are many, comprising the pollen of various plants, dusts, emanations from feathers, etc.; and occasionally a strong suggestion appears to be a causative factor. According to season two forms have been recognised: the "June" or "rose" cold in the spring, and the "hay asthma" or "hay fever" which occurs during August and September.

(*b*) *Symptoms.*—The disease begins suddenly, and often shows a curious punctuality in the date of its annual recurrences. The symptoms are those of a severe coryza with a profuse watery, rarely muco-purulent, discharge. The eyes are reddened and watery, with itching lids. The senses of taste, smell, and hearing are much impaired. Cough, sometimes very severe, and excited by a tickling sensation in the larynx and pharynx, is a frequent concomitant. There may be slight chilliness, fever, disturbed sleep, poor appetite, and a sense of weakness. Not infrequently attacks occur which are identical with bronchial asthma; or the asthmatic attack may alternate with the coryza. The symptoms vary from day to day in severity, and the entire attack usually covers from 4 to 6 weeks.

IV. **Epistaxis.**—See page 232.

II. DISEASES OF THE LARYNX

(See also pages 255 to 260, 260 to 264, and 273 to 276)

I. **Acute Catarrhal Laryngitis.**—(*a*) *Causes.*—Cold, excessive use of the voice, inhalation of irritating vapours, injury, foreign bodies, swallowing of corrosive poisons or very hot fluids. May be primary, more commonly associated with an acute naso-pharyngeal catarrh.

(*b*) *Symptoms.*—The voice is hoarse, husky, or completely lost; there is a sensation of tickling in the larynx, with a frequent dry cough; and there may be a feeling of constriction with moderate dysphagia, and slight tenderness on grasping the larynx. Examination shows a reddened and swollen laryngeal mucosa, affecting particularly the ary-epiglottic folds. The vocal cords are pinkish, swollen, and lack their normal mobility. A moderate mucous secretion coats the affected portions. In rare instances œdema of the glottis may supervene. As a rule there is but slight fever. Very severe cases may present marked dysphagia, incessant and distressing cough, and intense dyspnœa.

(*c*) *Diagnosis.*—Ordinarily the diagnosis is easy, particularly if the patient is of an age to permit a laryngoscopic examination. The latter will rule out nervous aphonia and glottic œdema.

II. **Acute Laryngitis with Spasm of the Glottis.**—In the vast majority of instances it is this condition which in children constitutes "spasmodic croup," the laryngitis occurring alone or as a part of an acute naso-pharyngeal catarrh. The spasmodic attack almost always occurs after the first sleep, and in many cases is preceded by the symptoms of a slight "cold." Usually between 10 and

12 P. M. the child awakes with a brazen, croupy cough, husky or whispering voice, dyspnœa, stridulous respiration, congested face, and great restlessness. Under treatment the spasmodic element subsides, the child falls asleep and wakes the next morning either well, or, as is more commonly the case, still with a croupy cough and other evidences of a mild catarrh of the larynx. The attacks may recur with diminishing intensity for 3 or 4 subsequent nights.

It seldom happens that laryngismus stridulus is mistaken for spasmodic croup, as in the former there is an absence of fever, coryza, and antecedent hoarseness; but in the absence of patches on the pharynx, swollen cervical glands, and a satisfactory inspection (difficult or impossible in infants and very young children), it may be hard to exclude membranous or diphtheritic laryngitis except by the lapse of time, which in the latter case will disclose the signs of a progressive laryngeal stenosis. I have seen a case of scarlet fever requiring intubation, and another of severe catarrhal laryngitis requiring tracheotomy, but in neither of which was there œdema or false membrane.

III. **Chronic Laryngitis.**—(*a*) *Causes.*—Results from repeated acute attacks, especially in those who speak much in public or in the open air; too much smoking; and chronic alcoholism.

(*b*) *Symptoms.*—The voice is husky, hoarse, or rough, and in severe cases an almost complete aphonia may occur. Cough is usually present, either slight or severe and paroxysmal, and is due to a frequent sensation of irritation or tickling in the larynx. Rarely is there pain. It is often associated with chronic pharyngitis, and may be caused by nasal stenosis. Laryngoscopic examination will show a slightly swollen and but moderately reddened mucous membrane, with distention of the mucous glands of the epiglottis and ventricles. Superficially eroded spots are occasionally seen.

IV. **Œdema of the Larynx.**—(*a*) *Causes.*—Very rarely it follows acute laryngitis. It occurs in connection with erysipelas, diphtheria, scarlet fever, typhus and typhoid fevers, and acute phlegmonous inflammations of the pharynx, neck, and floor of the mouth; syphilitic or tuberculous laryngitis; with the general œdema of acute or chronic nephritis and chronic heart disease; or from pressure by intra-thoracic growth or aneurism.

(*b*) *Symptoms.*—The chief symptom is a rapidly developing dyspnœa, with increasing huskiness and final extinction of the voice. The respiration may be stridulous.

(*c*) *Diagnosis.*—In addition to the presence of one of the causative affections and the quick oncoming of dyspnœa, the laryngoscope, or palpation, or even simple inspection with the tongue fully depressed, will reveal great swelling of the epiglottis and ary-epiglottic folds. The condition is very fatal in the absence of prompt surgical treatment.

V. **Tuberculous Laryngitis.**—In the great majority of cases this is secondary to pulmonary tuberculosis, with which it occurs as a complication in from 18 to 30 per cent.

(*a*) *Symptoms.*—The earliest symptom is a huskiness or hoarseness of the voice, which may eventuate in aphonia and mere whispering. Sooner or later there is dysphagia, which may be so marked that any attempt to swallow causes excruciating pain, perhaps cough and suffocation. Cough is frequent, ineffectual, and painful. The severer symptoms are seen particularly when there is extensive ulceration and destruction of the epiglottis and ulceration in the pharynx. In the early stage the mucous membrane of the larynx is thickened and pale; later, the broad, gray, ill-defined tuberculous ulcers appear, particularly on the posterior surface of the epiglottis, the ary-epiglottic folds, the false and the true cords. The latter are thickened and eroded.

(*b*) *Diagnosis.*—In the presence of ascertained pulmonary tuberculosis the diagnosis of tuberculous laryngitis is rarely difficult. If the lung symptoms are indefinite it may be confused with syphilitic laryngitis, but the absent history of the latter disease, the negative result of the therapeutic test (mercury and iodides), and the finding of tubercle bacilli in some of the secretion, which may be removed from the ulcers if necessary, will decide its tuberculous nature.

VI. **Syphilitic Laryngitis.**—This is a frequent secondary or tertiary symptom, and occurs also in the hereditary form.

(*a*) *Symptoms.*—These are hoarseness, aphonia, and dysphagia. If secondary, the lesion is an erythema, perhaps with superficial ulceration and the evidences of a mild catarrhal laryngitis. If tertiary, small submucous gummata are present, which may ulcerate deeply, often causing necrosis and exfoliation of the laryngeal cartilages, or slowly heal. In either case the contraction of the resulting scar or fibrous tissue may produce stenosis of the larynx.

(*b*) *Diagnosis.*—This depends upon the history and especially upon the coexisting evidences of the causative disease (see also V preceding). The laryngeal lesions of inherited syphilis appear in the majority of cases within the first year of life; less commonly at 12 to 15 years of age.

VII. **Tumours of the Larynx.**—A tumour of the larynx should be remembered as a possible cause of hoarseness, aphonia, or other alterations in the voice; or of cough, dyspnœa (sometimes sudden), and dysphagia. Laryngoscopic examination will reveal the growth, seated as a rule upon the vocal cords. As the treatment is surgical, special works should be consulted for further information.

VIII. **Laryngismus Stridulus.**—A spasm of the laryngeal adductors occurring in children usually between 6 months and 5 years of age. It is unattended by cough, hoarseness, or other evidence of laryngitis, and is a neurosis, seen most commonly in association with rickets, less frequently with tetany (*q. v.*). At one time it was presumed to be due to enlargement of the thymus gland, and was called "thymic asthma." It is to be distinguished from the spasm of the glottis which may occur in connection with many laryngeal affections. In popular parlance it is variously called "holding the breath," "fit of passion," or "child-crowing."

The attack comes on often as the child wakes from sleep, either at night or in the daytime. The breathing ceases, the face becomes congested and cyanotic, and the seizure terminates suddenly with a high-pitched crowing inspiration. There may be convulsive movements of the hands and feet (carpo-pedal spasm), or, less frequently, general convulsions. The paroxysm may be repeated a number of times during 24 hours. Death during the attack is a very rare consequence. The absence of coryza, cough, hoarseness, or fever, together with the character and frequency of the attacks, will serve to separate it from spasmodic croup.

IX. **Paralyses of the Larynx.**—See pages 257 to 260.

III. DISEASES OF THE BRONCHI

I. **Acute Bronchitis.**—Acute catarrhal inflammation of the bronchial mucosa is usually bilateral, and affects mainly the first and second divisions of the tubes. It is often epidemic, and probably due to a microbic infection. Affecting the bronchioles ("capillary bronchitis"), it is simply a part of a broncho-pneumonia.

(*a*) *Causes.*—Usually a downward extension of an acute nasopharyngeal catarrh caused by cold, and occurs as a symptom in influenza, measles, typhoid fever, malaria, pertussis, and bronchial asthma. Very young, elderly, and debilitated persons are particularly liable; so also are certain individuals.

(*b*) *Symptoms.*—Usually there is a coryza, with chilliness, slight hoarseness, soreness of the throat, a feeling of weakness and oppression, and some general aching of the back and limbs. There is slight fever (100° to 101°), but in severe cases it may rise to 103°, with a corresponding frequency of the pulse. The bronchitic symptoms proper are substernal soreness, rawness, tightness or oppression; cough, at first dry, rough, and irritating, often occurring in severe paroxysms, and by its violence causing muscular pain and soreness along the costal margins. In from one to three or four days the cough loosens and expectoration appears, at first scanty and mucous, later abundant and muco-purulent, with great relief to the patient.

The breathing, except in children or in severe cases with fever in adults, is not increased in rapidity, and there is rarely dyspnœa unless the smaller tubes are involved. Palpation and percussion are, as a rule, negative. Auscultation in the initial stage reveals sibilant and sonorous râles, which in the stage of abundant secretion are replaced by fine and coarse moist or bubbling râles. The breath sounds may be somewhat harsh.

(*c*) *Course and Duration.*—In otherwise healthy adults the fever disappears within a week, and at the end of two weeks convalescence is established, although a moderate loose cough and expectoration may persist for a week or two longer. But in the very old, the very young, or in persons debilitated from any cause, the inflammation may extend to the bronchioles and air cells constituting a broncho-pneumonia (page 928).

(*d*) *Differential Diagnosis.*—At the onset, in sudden and severe cases, bronchitis may simulate lobar pneumonia, but the absence of the physical signs of consolidation, as well as the milder grade of disturbance, will soon decide against the graver disease. The development of broncho-pneumonia during the course of an acute bronchitis is usually indicated by the finding of numerous fine and subcrepitant râles, with patches of slight dulness and weak or distant bronchial breathing, especially at the bases of the lungs. It is well to bear in mind that an acute bronchitis may be the initial symptom of measles or pertussis. The physical signs of acute miliary tuberculosis of the lungs may at the beginning closely resemble those of an acute bronchitis.

II. **Chronic Bronchitis.**—This is usually associated with emphysema, and not uncommonly there are dilatations of the bronchial tubes (bronchiectases).

(*a*) *Causes.*—Occasionally due to repeated attacks of acute bronchitis, but occurs much more commonly as a result of chronic valvular disease, chronic alcoholism, rheumatism, gout, chronic nephritis, syphilis, chronic pulmonary disease, or aortic aneurism. It affects mainly elderly persons, and the symptoms are aggravated during the winter months.

(*b*) *Varieties.*—Certain clinical varieties are recognised.

(1) The most common form occurs in elderly, often gouty, men, and is associated with emphysema, heart disease, arteriosclerosis, and highly acid urine containing a trace of albumin. The cough may

occur only in the morning or at night, with free expectoration. The general health may be unimpaired for years.

(2) Dry catarrh occurs in elderly persons, is associated with emphysema and characterized by violent paroxysms of cough, with no, or but little and tenacious, expectoration.

(3) Bronchorrhœa, a profuse watery or thin and purulent, rarely thick, expectoration, may attend chronic bronchitis in the old or the young, and in the former may be ultimately associated with bronchiectasis and putrid bronchitis.

(4) A form of chronic bronchitis occurring particularly in women has been described by Osler. It comes on between 20 and 30 years of age, and may persist without impairment of the general health.

(*c*) *Symptoms.*—Cough, frequently paroxysmal, worse in the morning and at night, is the most constant symptom, often disappearing in summer and returning in the winter. The amount of expectoration varies; rarely there is none, more commonly it is more or less abundant and muco-purulent or decidedly purulent, occasionally thin and serous. Fever is rarely present, and substernal pain is uncommon, although there may be soreness along the costal margins if the cough is violent and paroxysmal. There may be some dyspnœa on exertion.

The *physical examination* usually shows an enlarged chest with deficient expansion, due to associated emphysema. The percussion note is normal, hyperresonant, or slightly tympanitic. Auscultation reveals prolonged, usually low-pitched and wheezy, expiration over both lungs, with bilateral fine and coarse, dry and moist, râles. Exceptionally there may be slight dulness or impaired resonance with fine crepitations at the bases, due to a certain amount of œdema and passive congestion. The physical signs in some cases may be negative.

(*d*) *Diagnosis.*—This is usually easy. The heart, arteries, and urine should be examined in order to ascertain whether the bronchitis is secondary to cardio-vascular or renal disease.

There are certain cases which suggest pulmonary tuberculosis, but the discrimination can generally be made by the absence of the fever, circumscribed consolidation, and emaciation, which attend phthisis, as well as a failure to find tubercle bacilli in the sputum.

III. **Putrid Bronchitis.**—(*a*) *Causes.*—In rare cases this is primary, following, or alternating with, a chronic bronchitis, but in the large majority of instances it is indicative of bronchiectasis, gangrene, abscess, empyema with perforation of the lung, pulmonary actinomycosis, or decomposition of the material contained in phthisical cavities.

(*b*) *Symptoms.*—The sputum has a fetid and disgusting odour, is usually copious, thin, and grayish, and on standing separates into two layers, the upper a greenish fluid covered with frothy mucus, the lower consisting of a thick sediment in which may be found pea-sized gray or yellow masses (Dittrich's plugs), composed of bacteria, pus, leptothrix, fat crystals, and detritus. The physical signs vary according to the associated or causative conditions (*q. v.*).

Fetid bronchitis, when an event in the ordinary chronic form, may be announced by irregular chills, high fever, and a greater degree of general weakness, caused by septic absorption from the decomposing secretion. Abscess, gangrene, pneumonia, or metastatic cerebral abscess may result, or the affection settle back into the usual course of a chronic bronchitis, with or without the persistence of the offensive character of the sputum.

IV. **Bronchiectasis.**—The dilatation may be cylindrical or saccular, usually partial and often bilateral; but, especially in congenital cases, may be general and unilateral, involving all the bronchia of one lung.

(*a*) *Causes.*—Occurs most commonly in connection with chronic bronchitis and emphysema, chronic phthisis, broncho-pneumonia in children, adhesive pleurisy, or interstitial pneumonia; and occlusion of a bronchus, by foreign bodies or by the pressure of a tumour or aneurism, whereby the accumulated secretion distends the tubes. Very rarely it is a congenital anomaly.

(*b*) *Symptoms.*—Small bronchiectases, such as occur in chronic bronchitis, emphysema, and phthisis, may not give rise to suggestive symptoms, and the condition may be quite unsuspected.

If there are one or more saccular dilatations of considerable size, the characters of the cough and expectoration are distinctive. The cough is paroxysmal, usually occurs in the morning, and is due to the accumulation of secretion in one or more large sacs. When filled, the cavity overflows either spontaneously or because of a change of position, thus irritating the adjoining healthy mucosa and exciting cough. After the sac is emptied a period of quiescence succeeds until a reaccumulation takes place. The expectoration is copious, and a large quantity is discharged in a short time. On standing, the gray or grayish-brown sputum separates into two layers, the upper thin, mucoid, and covered by a brownish froth, the lower consisting of a thick granular sediment containing pus cells, granular *débris*, fatty-acid crystals, and occasionally red cells and hæmatoidin crystals. Nummular masses are not common, nor are elastic fibres found unless the walls of the dilatation are ulcerated. The sputum usually has a peculiar sour or stale odour; occasionally it is extremely offensive because of the presence of a putrid bronchitis (III, preceding). Hemorrhage may occur; fever and dyspnœa are not common, except as caused by a coexisting thoracic affection.

The *physical examination* may be negative unless there are saccular dilatations sufficiently large and superficial to afford the signs of cavity. If the sac is empty there are tympanitic percussion, pectoriloquy, and cavernous or amphoric respiration, with occasional metallic râles. If the sac contains fluid the percussion note is dull, and loud gurgling râles are heard.

(*c*) *Diagnosis.*—Moderate dilatations are not recognisable, but if there is evidence of a cavity at the base of the affected lung in chronic interstitial pneumonia or chronic pleurisy, it is a bronchiectasis. (1) Compared with a phthisical cavity, a saccular dilatation lies, as a rule, toward the base rather than the apex of the lung, the signs persist but do not increase, the sputa are rarely nummular, the tubercle bacillus is absent, there is usually no fever and but little loss of flesh and strength, and the previous history is not that of pulmonary tuberculosis. (2) Actinomycosis of the lung may give rise to conditions which closely resemble bronchiectasis, but an examination of the sputum will enable the differentiation. (3) A localized empyema which has perforated the lung and established communication with a bronchus is discriminated with difficulty from bronchiectasis, but there is usually a history of an acute pleurisy, with a sudden onset of recurring paroxysms of cough and expectoration. Pulmonary abscess (*q. v.*) and pulmonary gangrene (*q. v.*) are, as a rule, readily distinguished from bronchial sacculations.

(*d*) *Prognosis.*—In rare instances cerebral abscess and pulmonary abscess or gangrene may occur. Pulmonary osteo-arthropathy may follow. Ordinarily the condition, although incurable, permits a long and fairly comfortable life.

V. **Fibrinous Bronchitis.**—A rare disease, acute or chronic, attended by the formation and expulsion of fibrinous casts (Fig. 253, page 653) of a bronchus and its branches. It occurs most frequently in males (2 to 1) between 20 and 40 years of age, and in the spring months. It is sometimes hereditary, and a certain epidemicity has been noted. It is frequently associated with tuberculosis and chronic heart disease, less frequently with chronic pleurisy, herpes, impetigo, and pemphigus, rarely with pneumonia. The attacks may coincide with the menstrual period. Its causation is unknown.

(*a*) *Symptoms.*—Very rarely the disease is acute, beginning with chills and high fever, and followed by dyspnœa, violent paroxysms of cough, and occasionally hæmoptysis.

More commonly the attack comes on like an ordinary severe bronchitis. Fever is not always present. The cough becomes violent and paroxysmal, and there are dyspnœa and a varying degree of cyanosis. Usually in a few hours, sometimes days, expectoration occurs, the sputa, often blood-stained, containing rounded ball-like masses, which, when unravelled, prove to be casts of a secondary or tertiary bronchus with its terminal branches. Hæmoptysis, sometimes profuse, may accompany or follow the expulsion of the casts. With the discharge of the mould the cough and dyspnœa subside, perhaps only to return in 1 or 2 days, and thus continue for a week or longer. The attacks may recur at intervals of weeks, months, or years, or there may be but one attack in a lifetime. In some instances the recurrences have a definite and regular period of intermission. The same bronchus is usually involved at each attack.

The *physical signs* are, as a rule, those of a severe bronchitis. A very dry râle (*bruit de drapeau*) is described. If a large bronchus is obstructed there may be diminished vocal fremitus, weak or absent respiration, and deficient expansion or inspiratory retraction of the lower ribs over the affected side. Dulness may be present if the lung area supplied by the plugged bronchus is collapsed.

(*b*) *Diagnosis and Prognosis.*—The diagnosis depends entirely upon the discovery of the casts. They may require to be distinguished from the casts of diphtheria by bacteriological examination.

The prognosis is usually favourable, although the disease may cover a period varying from 5 to 15 years. Extremely acute attacks may prove fatal.

VI. **Bronchial Obstruction.**—(*a*) *Causes.*—This may be due to foreign bodies in the bronchia and to thickening of the bronchial walls by inflammation or neoplasm, or to external pressure upon a bronchus by abnormal thoracic masses such as mediastinal or pulmonary tumour or abscess, aneurism, enlarged bronchial or mediastinal glands, hydatids, and large pleural effusions or pleural neoplasms.

(*b*) *Symptoms.*—Obstruction of the smaller bronchi does not give rise to appreciable symptoms. If a main or large bronchus is closed there will be marked dyspnœa, perhaps with inspiratory retraction of the lower sternum and lower ribs and intercostal spaces, particularly upon the affected side. The larynx will move with respiration, but to a much slighter extent than when the obstruction is at the glottic opening. The *physical examination* reveals deficient expansion upon the affected side, diminished vocal fremitus, a normal percussion sound, diminished or absent respiratory murmur, and, possibly, large and small dry râles at the narrowed or obstructed point. Later there may be dulness if the lung collapses. It is necessary to exclude laryngeal stenosis by visual examination.

VII. **Bronchial Asthma.**—The pathology of this disease is unsettled. Some regard it as a pure neurosis, a spasm of the bronchial muscles; others as a neurotic hyperæmia and swelling of the bronchial mucosa, with an exudate of mucin. It is probable that the second view applies to the majority of cases. It may alternate with neuralgia and epilepsy.

(*a*) *Causes.*—An underlying and peculiar irritability of the nervous system or bronchial muscles and mucosa, akin to that in hay fever, must be predicated. The exciting causes are extraordinarily diverse. An attack may be precipitated by the inhalation of dusts or odours from certain flowers or plants or emanations from animals. In one locality or climate attacks will occur, in others there is perfect freedom from the seizures. Strong emotions, fatigue, indiscretions in diet, and, more rarely, the presence of intestinal parasites, or, in women, pelvic disease, may be responsible. Diseases of the upper air-passages, such as nasal polypi, hypertrophic rhinitis, and chronically enlarged tonsils, are often strongly predisposing causes. A frequent initial event is an acute bronchitis.

The disease often begins in childhood. It is more common in men than in women (2 to 1); half of the cases are hereditary; and it occurs rather more frequently in winter and spring than in the summer, unless associated with hay fever.

(*b*) *Symptoms.*—The attack may begin abruptly, but in about 50 per cent of cases there are various premonitory symptoms, such as anxiety, depression, nervousness, irritability, vertigo, drowsiness, headache, neuralgia, chilly feelings, substernal tightness, polyuria, or flatulence and digestive disturbances.

Ordinarily the paroxysm commences at night, often during sleep; less commonly while awake, or during the day. There is a sense of breathlessness, with thoracic oppression and constriction, soon developing into the most intense dyspnœa. The patient may rush to the window for air, or sit up, placing his hands on the bed or the arms of a chair in order to gain additional support for the extraordinary muscles of respiration. Cyanosis soon appears, the face is anxious and wet with perspiration, the hands and feet are cold, the temperature subnormal, and the pulse frequent and small. The attack reaches its height in from a few minutes to several hours, at which time the dyspnœa lessens, and there is often a violent fit of coughing and the raising of a tenacious and scanty expectoration, with great relief to the patient. The eosinophiles in the blood are much increased during the attacks, constituting from 25 to 50 per cent of the total differential leucocyte count.

Physical examination during the attack reveals a distended chest, as the lungs are overfull of air which can not be expired. Moreover, as the thorax is thus in the inspiratory position, its movements, in spite of the violent respiratory efforts, are extremely limited, and the diaphragm is lowered and almost immobile. The respirations are normal or decreased in frequency; inspiration is short and quick; the expiration is prolonged and wheezy because of the difficulty in expelling the previously inspired air through the narrowed tubes—an expiratory dyspnœa. Percussion is normal or hyperresonant. On auscultation a multitude of sonorous and sibilant râles are heard, during both inspiration and expiration. Toward the close of the attack, and during its course if bronchitis coexists, moist râles of various sizes are perceived.

The sputum in the early stage of the attack is expectorated with great difficulty and contains rounded gelatinous pellets, an examination of which will, in every case of true bronchial asthma, reveal the presence of Curschmann's spirals (Fig. 254, page 653). Late in the attack the sputum is muco-purulent and the spirals disappear. Leyden's crystals and eosinophiles may also be found.

(*c*) *Course, Duration, and Prognosis.*—The attacks may be repeated for from 2 to 5 or 6 nights, with or without wheezy cough and respiration during the intervals. The set of paroxysms may recur at intervals of a month or a year, more or less. If the recurrences are frequent, severe, and long continued, emphysema, right-heart dilatation, and chronic bronchitis usually ensue, with permanent dyspnœa and wheezy asthmatic respiration, the purely spasmodic seizures ceasing or diminishing in frequency and intensity.

However alarming the appearance of the patient, death never takes place during the attack. On the other hand the disease is essentially chronic, although there may be prolonged periods of freedom. Death may occur in old cases from the resultant emphysema and cardiac lesions.

(*d*) *Diagnosis.*—This is usually easy. The dyspnœa (the so-called asthma) of renal and cardiac disease is not attended by the dry, sonorous, and piping râles and other physical signs of true asthma, although fine, moist crepitations may be heard; nor is the subjective dyspnœa of hysteria. In glottic spasm or abductor paralysis the dyspnœa is distinctly of the inspiratory type, voice changes may be present, and the multitudinous dry râles of asthma are conspicuously absent.

IV. DISEASES OF THE LUNGS

I. **Pulmonary Congestion.**—Two forms are recognised: *active* and *passive.*

(I) **Active Congestion.**—In the great majority of cases active hyperæmia is a symptom or associated condition in connection with certain pulmonary diseases, such as bronchitis, pneumonia, tuberculosis, or pleurisy. In certain instances it is possible that it may occur as a primary and independent affection, which may quickly disappear or be followed by œdema, or, in rare cases, by reason of its intensity, terminate in death. This condition results from drunkenness and exposure to severe cold or great heat, or from violent exertion.

The *symptoms* are rapid breathing, cough, blood-tinged frothy sputa, somewhat harsh respiration, with fine moist râles, and an absence of fever, unless some febrile or inflammatory condition coexists. The *diagnosis* of this condition, aside from pulmonary œdema and abortive pneumonia, must be made with caution.

(II) **Passive Congestion.**—Of passive hyperæmia two varieties are recognised: *mechanical* and *hypostatic.*

(*a*) *Mechanical Congestion.*—(1) Causes.—In the vast majority of cases passive hyperæmia is a result of affections of the left heart, especially mitral stenosis or incompetency, or of emphysema. In rare instances it is due to pressure by tumours. The essential element in either case is the presence of an obstacle to the return of the blood to the left ventricle, with resulting chronic congestion (brown induration) of the lungs.

(2) Symptoms.—Dyspnœa, cough, and the expectoration of frothy, often blood-stained, sputa containing "heart-disease cells" (page 652). Hæmoptysis may occur. So long as the compensation of valvular defects is unbroken these symptoms do not appear.

(*b*) *Hypostatic Congestion.*—(1) Causes.—In general it occurs in fevers and conditions of great debility attended by feebleness of the heart, and is favoured by a prolonged dorsal position. It is common in long-continued typhoid fever; paralyses, especially cerebral; prolonged coma; abdominal tumours, tympanites, or ascites; and wasting diseases like tuberculosis and carcinoma.

(2) Symptoms.—Subjective and rational symptoms are usually lacking, but physical examination will disclose, over the bases posteriorly, slight dulness, weak, or harsh, perhaps broncho-vesicular, respiration, and moist râles. The vocal fremitus may or may not be increased. If patches of broncho-pneumonia exist the breathing may be truly bronchial.

II. **Pulmonary Œdema.**—(*a*) *Causes.*—This condition—an effusion of serum from the distended capillaries into the alveoli and their walls—is almost invariably secondary to the various forms of pulmonary congestion, inflammation, abscess, infarction, or tuberculosis. The œdema may be *local*, in the immediate neighbourhood of a circumscribed, usually inflammatory, lesion; or *general*, arising from causes similar to those which produce congestion, or constituting a terminal event in states of great debility. The factors which appear to be instrumental in causing the transudation are increased tension in the pulmonary vessels, increased fluidity of the blood, an abnormal permeability of the vessel walls due to nutritive changes, and left ventricular weakness.

The diseases with which œdema is most commonly associated are pneumonia; the cachexias, especially that due to carcinoma; grave or fatal anæmias; acute and chronic nephritis; acute specific fevers with heart weakness; cardiac valvular disease; and cerebral apoplexy or injuries.

(*b*) *Symptoms.*—The onset of this condition may be extremely sudden, especially in nephritis. More commonly there is simply an increase in the manifestations of the pre-existing causative disease.

The symptoms of pulmonary œdema are increasing dyspnœa, cyanosis, cough, and an abundant, frothy, watery, rarely tenacious, expectoration, which may be blood-stained if congestion is also present. Fever is absent unless the œdema is due to some inflammatory or other febrile condition. The *physical signs* are impaired resonance or slight dulness over the bases, with absent or weak, perhaps broncho-vesicular, breath sounds. There are abundant large and small râles, of an unusually liquid character, over the involved areas.

(*c*) *Diagnosis.*—This depends mainly upon the presence of numerous unusually moist râles, both large and small, and slight dulness at the bases, particularly if the temperature is normal.

III. **Broncho-pulmonary Hemorrhage** (*Hæmoptysis*).—See page 278.

IV. **Pulmonary Hemorrhage or Infarction.**—Under this head may be included *pulmonary apoplexy* (diffuse infiltration), and *embolism* or *infarction*.

(I) **Diffuse Hemorrhagic Infiltration.**—In septicæmia or pyæmia, excessively severe or malignant fevers, and in certain diseases of the brain there may be an extensive infiltration of the lungs with blood. If the patient lives, pneumonia, abscess, or gangrene results. This condition is not common and the symptoms are indefinite. There may be dyspnœa, cyanosis, hæmoptysis, and collapse symptoms with signs of rapid pulmonary consolidation.

(II) Pulmonary Embolism and Thrombosis.—Circumscribed infarctions are due to stasis, thrombosis, or embolism of some of the branches of the pulmonary artery, whereby the vessels are blocked and characteristic wedge-shaped hemorrhagic areas or infarcts result. Emboli may be non-septic or septic.

CAUSES.—*Non-septic* embolism arises most commonly from chronic cardiac disease, especially mitral stenosis, or, less frequently, regurgitation. Thrombi may form in the right auricle or in the systemic veins and, either entire or disintegrated into smaller portions, become emboli; or thrombosis or stasis take place in the branches of the pulmonary artery itself.

Septic emboli originate from a gangrenous or a suppurative focus, such as exists in pyæmia, and as they are practically masses of pathogenic bacteria, pulmonary gangrene or metastatic abscesses will result from their lodgment in the lung.

SYMPTOMS.—(1) *Of Non-septic Embolism.*—If the embolus is sufficiently large to occlude a main branch of the pulmonary artery sudden death may occur. If a medium-sized, but still large, branch is the seat of the obstruction there will be cough, hæmoptysis, mental anxiety, intense dyspnœa, syncope, and perhaps coma and convulsions. If the smallest branches are involved there may be no recognisable symptoms; or there may be moderate dyspnœa, cough, and slight hæmoptysis; or the expectoration, especially at the onset, may consist of a small quantity of nearly pure blood or a gelatinous bloody mucus, the blood subsequently disappearing. It is not, at first certainly, the rusty sputum of a pneumonia. Cough and hæmoptysis occurring during the course of chronic cardiac disease is very suggestive of embolism, although the spitting of blood may result from passive congestion.

The *physical signs* in slight infarctions are negative. If the infarction is large and, as is usually the case, occupies the lower lobe, there will be near the base circumscribed dulness, increased fremitus and voice sounds, broncho-vesicular or bronchial breathing, and moist râles—i. e., the evidences of a strictly limited consolidation. There may be pleural friction accompanied by transient pain.

(2) *Of Septic Embolism.*—If the first metastasis of a gangrenous or suppurative focus is to the lungs, the earlier symptoms caused by the septic embolus will resemble those just described as being caused by a non-septic plug, followed in due time by the evidences of pulmonary abscess (*q. v.*) or gangrene (*q. v.*). Occurring as a part of an already existing pyæmia, the symptoms are those of the causative disease *plus* the physical signs of a primary consolidation, with subsequent abscess or gangrene.

Diagnosis.—The sudden onset of dyspnœa and pleuritic pain, the expectoration of a moderate quantity of blood or bloody mucus, the appearance of the physical signs of a circumscribed consolidation at the base, without fever, together with the presence of chronic heart disease or other acknowledged causative condition, will enable a diagnosis.

Prognosis.—In small non-septic infarcts not unfavourable; in septic infarcts, grave.

V. **Lobar Pneumonia.**—See page 792.

VI. **Broncho-pneumonia.**—An infectious inflammation of the terminal bronchi and their communicating air cells.

Causes.—The disease may be *primary*, the previous health having been good; or *secondary* to some antecedent disease. Broncho-pneumonia attacks especially the very young, three fourths of the pneumonias in children under 5 years of age being of this variety; the very old, especially if they are the subjects of some chronic and weakening ailment; or the debilitated of any age. It is most common among the poorer classes because of insanitary surroundings. Rickets and chronic diarrhœa also predispose.

(1) The *primary* cases are usually due to cold and exposure.

(2) The *secondary* cases follow acute bronchitis and the infectious fevers, especially measles, whooping cough, diphtheria, scarlet fever, erysipelas, and smallpox. "Aspiration" pneumonia is of this type, and is secondary to the inhalation of particles of food or drink, an accident which may occur when the larynx is insensitive, as in the coma of cerebral apoplexy, uræmia, or other condition attended by prolonged unconsciousness. It may follow operations on the mouth, nose, and trachea, or the inhalation of ether; or hæmoptysis; or occur in connection with cancer of the larynx or esophagus; or result from the aspiration of the secretion from a bronchiectatic cavity or a purulent pleurisy which has perforated the lung. In all these cases infective or irritant particles enter the bronchial tubes.

(3) The *bacteriology* of broncho-pneumonia is of interest. No one organism is responsible for the disease. Among those which are most commonly found are the *Pneumococcus lanceolatus*, *Streptococcus pyogenes*, *Staphylococcus aureus et albus*, the influenza bacillus (Pfeiffer's), and the bacillus of diphtheria. As a rule the infection is a mixed one, at least two varieties coexisting. The most constant organism in the primary form of the disease is the pneumococcus, which may exist alone; in the secondary form the streptococcus, usually in combination with one of the other organisms.

Symptoms and Clinical Varieties.—The mode of onset varies. If there has been no antecedent disease and the attack is primary, it begins abruptly with a chill and a rapid rise in temperature, resembling, in this respect, a lobar pneumonia. On the other hand, if there is a pre-existing bronchitis, either simple or specific, of the larger tubes, the onset is less abrupt and there is rarely a distinct chill.

In either case the typical symptoms are cough, which may be violent and painful; dyspnœa and rapid respiration (40 to 60 to 80), with an expiratory moan; fever, varying from 102° to 104°; rapid pulse and, after a time, cyanosis. The physical signs at first are simply those of a bronchitis of the smallest tubes, abundant sibilant and subcrepitant râles without dulness; later, and depending upon the presence of patches of consolidation, there may be slight dulness with harsh or broncho-vesicular respiration, especially at the bases and on either side of the spine. If the consolidated areas are sufficiently numerous and confluent the dulness may be decided, the breathing may be bronchial, the vocal fremitus distinctly increased, and marked bronchophony be present. In cases of extensive consolidation there may be inspiratory retraction of the lower sternum and lower ribs, indicative of deficient lung expansion.

There are certain, sometimes considerable, variations in the symptoms and clinical type of the disease which demand consideration.

(1) In severe cases, the sensitiveness of the nerve centres having been decreased as an effect of poisoning by carbon dioxide, the dyspnœa and cyanosis steadily increase, the cough lessens, the respirations are shallow and ineffectual, although rapid, and the râles become larger and moister. The patient is drowsy but not quiet, and death ensues from cardiac weakness, especially of the overdistended and labouring right ventricle. This type of the disease is the suffocative catarrh of the old writers.

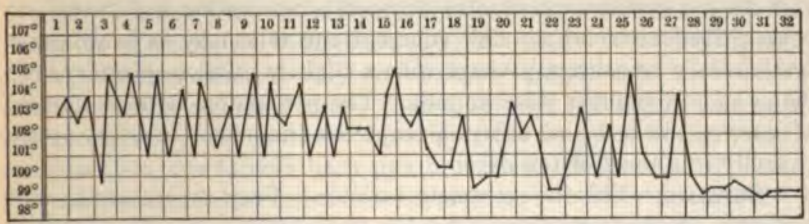


CHART XXII.—Temperature curve of prolonged broncho-pneumonia; recovery (Holt).

(2) The extremely remittent type of the fever in many cases of broncho-pneumonia, especially in children, is noteworthy, as the frequency of its occurrence is not always appreciated, and from a certain resemblance to the temperature curve of malaria a diagnosis of the latter disease may be made (Chart XXII).

(3) The primary form in infants and young children sets in abruptly, with high fever. The physical signs are those of a somewhat circumscribed moderate consolidation rather than of a diffuse bronchitis, and the disease bears a close resemblance to a lobar pneumonia. Indeed, it frequently terminates by crisis.

(4) The primary form in adults may begin like a severe and acute bronchitis, but the fever, prostration, cough, and dyspnœa are more marked than in a bronchitis, and the expectoration is tenacious and rusty or blood-stained.

(5) As with lobar pneumonia in children, so with broncho-pneumonia, especially if the onset is sudden, the initial symptoms may be predominantly cerebral (delirium, drowsiness, convulsions, coma), or gastro-intestinal (nausea, vomiting, more rarely diarrhœa), the pulmonary symptoms and signs being masked, or not appearing until several days have elapsed.

(6) The secondary form begins as a bronchitis, oftentimes gradually. If during convalescence from an acute infection, especially measles or pertussis, there is an increase in the fever with cough, dyspnœa, and rapid breathing, the presence of broncho-pneumonia is assured, even though physical examination reveals nothing but fine moist râles at the bases or diffused through both lungs, without evidences of consolidation.

(7) Certain mild cases are seen in adults which are not commonly recognised as broncho-pneumonia. Following a severe ordinary acute bronchitis of the larger tubes in otherwise healthy persons, the cold may in common parlance "hang on" for more than the ordinary 2 or 3 weeks. There is an irritative cough with scanty expectoration, slight fever (99.5° to 101°), and although the patient may continue at his ordinary vocation, he feels poorly, his appetite is impaired, and his sleep is restless. A careful physical examination will reveal circumscribed areas where fine subcrepitant râles may be heard upon rather deep breathing, perhaps with a slightly harsh respiratory murmur and intensified voice sounds. The percussion note is normal. Under rest in bed and other appropriate treatment recovery occurs usually in a week or ten days.

Duration.—This is extremely variable. Very severe cases, especially in children, may prove fatal in from 3 to 6 days. The duration of the common type of broncho-pneumonia ending in recovery varies from 1 to 3 weeks; in some instances the disease is protracted to 6 or 8 weeks, rarely even to 10 or 12 weeks. Death or recovery may occur at any time. As a rule the decline of the fever is by lysis.

Terminations.—These are resolution; suppuration or gangrene, practically only in the aspiration or deglutition pneumonia of which they are common sequelæ; and chronic interstitial pneumonia, especially if the original broncho-pneumonia is tuberculous.

Differential Diagnosis.—The cardinal symptoms are fever, usually remittent, cough, dyspnœa, rapid respiration, and *bilateral* fine or subcrepitant râles, *with* or *without* evidences of moderate or patchy consolidation. A patient with fever, rapid breathing, and a chest so full of large and small moist râles on both sides that the respiratory murmur can not be heard, has broncho-pneumonia. Atypical cases are most common in infants. Under 3 years of age broncho-pneumonia is of much more frequent occurrence than lobar pneumonia.

The differential diagnosis is mainly between primary broncho-pneumonia with extensive confluent consolidations and lobar pneumonia (page 792), or the protracted cases of broncho-pneumonia and the broncho-pneumonic form of tuberculosis (page 806). As capillary bronchitis *is* broncho-pneumonia, nothing need be said regarding a discrimination between them.

Prognosis.—Broncho-pneumonia is always a grave disease. In children of the better-housed classes the mortality varies from 10 to 30 per cent; in hospitals and among the very poor, from 30 to 50 per cent. The primary cases have a good prognosis; the fatality is greatest in the secondary forms. Aspiration or deglutition broncho-pneumonia, because of its frequent termination in abscess or gangrene of the lung, usually causes death.

VII. **Chronic Interstitial Pneumonia.**—More or less localized, but often extensive, fibroid changes occur in the lungs in every case of pulmonary tuberculosis, abscess, tumour, hydatids, emphysema, and pleurisy. Excluding these, there is a diffuse cirrhosis, usually affecting an entire lung, less frequently both lungs, which is here considered.

(*a*) *Causes.*—Chronic (diffuse) interstitial pneumonia may in rare cases be a sequel of acute lobar pneumonia, more commonly of a broncho-pneumonia; or may result from compression of the lung and extension of fibrous tissue from a chronic pleurisy; or be a consequence of syphilis; or arise from the pressure of a new growth, or an aneurism, or a foreign body in a bronchus. A special form due to the inhalation of dust is described elsewhere (see Pneumonokoniosis).

(*b*) *Symptoms.*—There is a chronic cough, with slight dyspnœa often present only on exertion. The cough may be slight or severe, with a moderate or profuse muco-purulent expectoration. Hæmoptysis is not uncommon. As the bronchial tubes are apt to become dilated, the special symptoms and signs of bronchiectasis (page 920) may be superadded. The disease is extremely chronic, and may be protracted for from 10 to 20 years or longer, with but moderate loss of flesh and strength, the patient remaining able to perform light manual labour.

In advanced cases the *physical signs* are: Marked retraction, approximation of the ribs, and deficient or absent expansion of the affected side, with compensatory enlargement of the opposite healthy side. The spine is curved, with its concavity toward the diseased lung, and the shoulder on the same side droops. The heart is drawn over by the shrinking lung, and, when the left lung is diseased and retracted, the heart is uncovered and there is a wide, visible impulse in the 2d, 3d, and 4th interspaces. The vocal fremitus is increased, unless there is considerable thickening of the pleura, when it is diminished or absent. The percussion note is dull, flat, or wooden, except over bronchiectatic cavities, which, if present, will cause a tympanitic or amphoric note. Over the apex and in the axilla the breath sounds are broncho-vesicular or bronchial; over bronchiectases, cavernous, or amphoric; over the base, weak, distant, or absent. Large and small, dry or moist, râles, and quite frequently rubbing and creaking friction sounds, are present. Over the healthy lung there may be hyperresonance and exaggerated respiration. The pulmonary second sound is accentuated and murmurs may develop, especially at the tricuspid valve, when the right ventricle begins to fail because of the extra work imposed upon it.

Death may result from intercurrent disease; from profuse hæmoptysis; from cardiac failure; or from gradual asthenia.

(*c*) *Differential Diagnosis.*—Practically the only disease from which it requires differentiation is pulmonary tuberculosis, with fibroid changes so extensive (fibroid phthisis) that the symptoms and physical signs of the two conditions are identical. If both lungs are affected it speaks for tuberculosis, but the only absolute evidence is the finding of tubercle bacilli in the sputum.

VIII. **Pneumonokoniosis.**—(*a*) *Nature and Causes.*—This is a chronic interstitial pneumonia due to the inhalation of dusts incident to special employments. According to the cause, certain varieties are recognised—*anthracosis*, or coal-miner's disease; *chalicosis*, "stonecutter's phthisis" or "grinder's rot," caused by the inhalation of mineral dust, in stonecutters, millstone makers, and grinders of cutlery; and *siderosis*, caused by the inhalation of metallic particles by workers in iron (especially), also in brass and bronze. Fibrosis may be due further to the breathing of vegetable dusts by grain shovellers, and workers in cotton, flax, and tobacco.

(*b*) *Symptoms.*—These come on gradually after years of exposure to the causative environment. The earliest symptoms and signs are those of a chronic bronchitis, then of associated emphysema, and finally of chronic interstitial pneumonia, with or without bronchiectasis. In the later stages the lungs may become tuberculous and the signs of ulcerative cavities become manifest.

(*c*) *Diagnosis.*—This depends upon the history of long-continued inhalation of dust; the presence of chronic bronchitis, emphysema, and fibrosis; and particularly upon an examination of the sputum. In anthracosis it is black; in siderosis it is of a reddish colour; in chalicosis the microscope reveals the shining angular particles of silica. Whether the process has become tuberculous must be ascertained by an examination for tubercle bacilli.

(*d*) *Prognosis.*—In the early stages favourable if the patient can leave his obnoxious employment. Otherwise, and in advanced cases, the prognosis is grave, although the disease is very chronic.

IX. **Phthisis Pulmonalis.**—See pages 806 to 811.

X. **Pulmonary Atelectasis.**—(*a*) *Causes.*—Collapse of the lungs—a partial or entire disappearance of air from the alveoli by compression of the lung or absorption of the air—may be congenital or acquired. The *congenital* variety occurs in the newborn or prematurely-born infant, as a result of deficient breathing power or obstruction of the air passages. An *acquired* atelectasis may be due to obstruction caused by the formation of mucus in bronchitis of the smaller tubes; to compression of the lung by large pleural or pericardial effusions or pneumothorax, thoracic aneurism or tumour, or great enlargement of the heart; to the weakened respiratory action caused by various paralyses; to upward pressure upon the diaphragm by abdominal effusion, tumour, or meteorism; or to thoracic deformities.

(*b*) *Symptoms.*—These are rarely distinctive, as the condition occurs usually in combination with broncho-pneumonia, especially in the milder forms of the latter which constitute the "grippy chest."

If the areas of collapse are small there is no dulness, the respiratory sounds are weak or absent, and at the end of inspiration there are localized fine or subcrepitant râles, often with little or no fever. Larger areas may present a weak broncho-vesicular or bronchial breathing with appreciable dulness; and if the collapse is extensive, as in the atelectasis of the newborn, the respiration is rapid, there is cyanosis, the lower half of the chest is retracted during inspiration, the extremities are cold, and there may be muscular twitching or even convulsions.

XI. **Emphysema.**—This term embraces *interstitial* emphysema, the form in which there is a rupture of the air cells, the contained air passing into the interlobular tissues; and *vesicular* emphysema, in which there is overdistention of the air cells and atrophy of their walls. The following varieties are recognised:

(I) **Interstitial Emphysema.**—This is due to wounds of the lung; or to rupture of air cells during a violent fit of coughing, or straining in childbirth or at stool, or during convulsions. The condition cannot be recognised unless the air travels from a rupture at the root of the lung along the trachea to the tissues of the neck, causing subcutaneous emphysema; or an air sac ruptures into the pleura with the consequent formation of a pneumothorax in otherwise healthy persons. A friction sound of a crumpling character resembling that heard in pleurisy is claimed to be somewhat characteristic of interstitial emphysema.

(II) **Hypertrophic Emphysema.**—The lungs are enlarged because of the atrophy and great distention of the air cells.

(*a*) *Causes.*—A congenital and often hereditary weakness of the lung tissue, probably a defective development of the elastic fibres, must be predicated, after which a persistently high intra-alveolar air tension accomplishes the dilatation. The disease may follow chronic bronchitis, whooping cough, and bronchial asthma, all of which cause increased intrapulmonary pressure; so also with heavy lifting, blowing on wind instruments, and glass-blowing.

(*b*) *Symptoms.*—The disease, in nearly all instances, begins insidiously, often in early childhood. In a well-developed case there is dyspnœa, which may be constant or perhaps felt only on exertion, often with wheezy breathing and laboured expiration. Cyanosis is very common, and may be extreme although the patient is about. Cough and frequent attacks of bronchitis, often of an asthmatic character, sometimes with severe cyanosis, are common. Ultimately the cough, like the bronchitis, becomes persistent and chronic. The cough may for some years disappear during the summer and return in the winter. Intercurrent paroxysms of spasmodic asthma are not uncommon. The temperature is usually subnormal and the surface of the body noticeably cool. The pulse is weak but not rapid. As a result of the obstruction to the flow of blood through the lungs the right ventricle may become hypertrophied, followed in course of time by dilatation, tricuspid insufficiency, and consequent widespread passive congestion in the systemic veins. There may be a slow loss of strength and flesh.

The *physical signs* in a well-marked case are quite distinctive. Inspection shows the emphysematous chest (Fig. 267). The expansion is greatly lessened, the principal movement of the chest is vertical, the upper abdomen retracts during inspiration, and the expiratory movement is prolonged. The sterno-mastoid muscles are prominent and hypertrophied. The neck veins are distended, perhaps pulsating. The apex beat is usually not palpable, but there is commonly a marked epigastric pulsation and systolic shock over the ensiform appendix. The vocal fremitus is lessened. The percussion note throughout is abnormally clear and hyperresonant, at times slightly tympanitic. The normal limits of pulmonary resonance are extended in every direction, the cardiac dulness is lessened or disappears, the upper limits of hepatic and splenic dulness are lowered by one or two interspaces, and there is resonance extending to an unusually high level above the clavicles, often with supraclavicular bulging. The characteristic auscultatory finding is that of a greatly prolonged, low-pitched, often wheezy, expiratory sound, while the inspiratory element is short, weak, or even absent. Large and small, dry or moist, râles, due to a coexisting bronchitis, are often heard, and may be so abundant as to obscure the respiratory murmur. Over those portions of the lung which are not greatly distended there may be exaggerated or harsh breathing. Vocal resonance may be increased, diminished, or absent. The pulmonary second sound is usually accentuated, and the signs of right ventricular hypertrophy and dilatation or tricuspid insufficiency (*q. v.*) may be found.

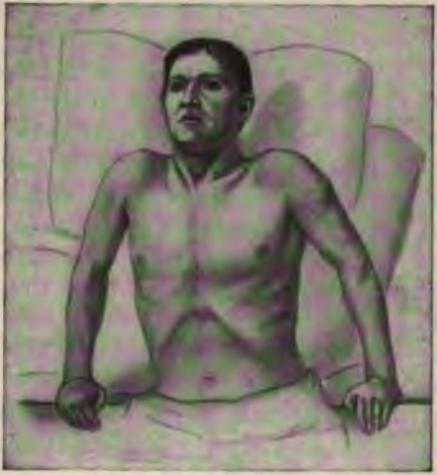


Fig. 267.—Emphysematous chest.

An acute vesicular emphysema may develop quite suddenly, during attacks of angina pectoris and the pseudo-asthmatic seizures of cardiac disease, because of the combination of violent inspirations with cyanosis and pulmonary congestion. Percussion shows a great increase in the volume of the lungs, and upon auscultation there are prolonged expiration and universally distributed sibilant râles.

(*c*) *Course and Prognosis.*—With the exception of the rare acute form, the course of the disease is essentially chronic and progressive. It is incurable, although not inconsistent with a long life. Frequent attacks of bronchitis aggravate the disease, and death may occur from an intercurrent pneumonia or the development of pulmonary phthisis. The right heart sequelæ—dropsy, hæmoptysis, or acute dilatation and cyanosis—may terminate life.

(*d*) *Diagnosis.*—The symptoms and physical signs in a well-marked case are usually sufficiently distinctive to avoid mistakes. It may require discrimination from the following conditions:

(1) Chronic Bronchitis.—Hypertrophic emphysema is separated from simple chronic bronchitis by the dyspnœa, the altered shape of the chest, the hyperresonance, and the low-pitched and prolonged expiration characteristic of the former.

(2) Pneumothorax.—This is usually unilateral and, as a rule, of sudden development and urgent character. The percussion note is of a distinctly tympanitic quality rather than simply hyperresonant, coin percussion elicits the bell sound, and there is a line of flatness at the base of the lung. Auscultation reveals amphoric respiration without vesicular quality, and there may be metallic tinkling and succussion sounds. Nevertheless the so-called spontaneous pneumothorax may result from the rupture of an emphysematous bleb.

(3) Pleurisy with Effusion.—The hyperresonant or slightly tympanitic (Skodaic) percussion sound over a lung compressed by a pleuritic effusion may suggest emphysema, but the unilateral flatness, the fever, and other evidences of pleural inflammation should prevent such a mistake.

(III) **Atrophic Emphysema.**—Senile or small-lunged emphysema is a primary atrophy of the lungs, the air cells coalescing to form a series of large vesicles. The chest is small and the intercostal spaces narrowed by the increased obliquity of the ribs consequent upon the diminution in the volume of the lungs, a condition directly opposite to that which obtains in hypertrophic emphysema. The subjects have had, for years, chronic bronchitis, winter cough, and dyspnœa, and present an old and withered appearance.

(IV) **Compensatory Emphysema.**—When there is deficient expansion of one lung or a portion of a lung, the other lung or a part of the same lung will expand to compensate for the areas which have ceased to functionate. Thus one lung becomes the seat of a compensatory emphysema when its companion is crippled by cirrhosis, lobar pneumonia, a large pleural effusion, or a pneumothorax. Localized vicarious emphysema occurs in the neighbourhood of broncho-pneumonic areas and tuberculous deposits or scars, and especially at the anterior margins of the lung if extensive pleural adhesions prevent its expansion. The condition is occasionally to be recognised by the local finding of some of the physical signs which are indicative of general emphysema.

XII. **Abscess of the Lung.**—(*a*) *Causes.*—An acute suppurative pneumonia caused by pyogenic organisms, usually the *Streptococcus pyogenes*, or the *Staphylococcus*, less commonly by the *Pneumococcus*, *Bacillus typhosus*, or other specific germ. The suppuration may be diffuse; more commonly one or more abscess cavities are formed. Abscesses, usually small and multiple, occasionally large, may follow a lobar or broncho-pneumonia, but, aside from suppurative infiltration, this is a rare sequence, except with inhalation or deglutition pneumonia (including foreign bodies), in which it is common. The causative organisms may reach the lung in infective emboli during the course of pyæmia, puerperal septicæmia, purulent osteomyelitis, or ulcerative endocarditis, with or without the production of infarcts. The abscess may result, by extension or perforation, from a purulent pleurisy, or the perforation of an hepatic abscess or suppurating hydatid cyst.

(*b*) *Symptoms and Diagnosis.*—If the abscess follows inspiration-pneumonia and is of sufficient size, the condition is easy of recognition. There will be a chill, high fever, and sweating; physical examination reveals the signs of a cavity, and an examination of the expectoration will afford characteristic findings. The sputum is yellow or greenish, with an odour which is offensive but not putrid like that of gangrene, and contains particles of lung tissue and elastic fibres, often in large numbers. If the abscess is too small to cause cavity signs, or if, as in septic embolism, there are multiple small abscesses, the physical signs are indefinite and the symptoms are overshadowed by those of the general pyæmia. In the non-pyæmic cases ulcerative endocarditis of the left heart and septic nephritis may occur as secondary infections, in which case the symptoms of these lesions will supervene.

(*c*) *Prognosis.*—As a rule death follows. In the abscesses resulting from pneumonia there are occasional recoveries, and if the abscess is single and accessible, surgical aid may avail.

XIII. **Gangrene of the Lung.**—This results from infection of a necrotic area in the lung by the organisms of putrefaction, *plus* an abnormal vulnerability of the tissues.

(*a*) *Causes.*—Diabetes in particular, and general debility from long-continued fevers, predispose. It may be a consequence of lobar pneumonia, tuberculous or bronchiectatic cavities, and hemorrhagic infarctions; or of the lodgment of foreign bodies in the bronchi, especially particles of food, or infective particles from disease of the upper air passages, as in deglutition or inhalation pneumonia; or the perforation of a carcinomatous ulcer of the esophagus into the lung or bronchus; or be due to pressure upon the bronchi by aneurism or tumour; or obstruction of the pulmonary artery by embolism or pressure. An embolus from a gangrenous area elsewhere may initiate it. It may also result from the direct extension of disease of the ribs or of the necrotic changes attending malignant growths of the esophagus or stomach. It is obvious that in the majority of these cases there is death of the part, either with simultaneous or secondary putrefactive infection, and that gangrene is a symptom rather than a disease.

(*b*) *Symptoms.*—There is irregular, usually moderate, fever, with prostration, rapid pulse, and loss of flesh. Infrequently the general symptoms may be slight.

The ***physical signs*** are those of cavity, provided the latter is sufficiently large and so situated as to be accessible to the usual methods of determining its presence. If the cavities are small and deep-seated they will elude recognition, and only the signs of the bronchitis, which always coexists, can be detected.

The characteristic symptoms are the intensely fetid odour of the breath and the character of the sputum. The expectorated material is usually abundant, and when allowed to stand separates into three layers, the uppermost consisting of a thick, grayish froth; the middle, of a watery, occasionally greenish or brownish, fluid; and the undermost of a heavy greenish-brown sediment containing fragments of lung tissue, abundant elastic fibres, pus, blood pigment, fatty crystals, granular matter, bacteria, and leptothrix. Red cells are often present, not infrequently in macroscopic quantities. Dittrich's plugs (page 920) are not found. Profuse hæmoptysis may occur from erosion of a large vessel; the pleura may be perforated, causing emphysema or pyopneumothorax; and cerebral abscess may be a consequence, as in bronchiectasis.

(*c*) *Diagnosis.*—The distinguishing symptom is the horrible odour of the breath, which may permeate the air of a large ward. It is more intense than that of putrid bronchitis or abscess, and this fact, together with an examination of the sputum, will aid in separating gangrene from either of the affections just mentioned. Nevertheless considerable difficulty may be experienced in the differential diagnosis.

(*d*) *Prognosis.*—In the majority of cases death results. If the gangrene is strictly circumscribed, recovery may occur through encapsulation, with discharge of the broken-down tissues by way of the bronchi. In accessible localized cavities, whose situation can be accurately determined, surgical interference has been successful.

XIV. **New Growths in the Lungs.**—The most common forms are carcinoma and sarcoma; rarely primary and unilateral, usually secondary and bilateral. The primary forms occur with equal frequency in each sex; the secondary involvements are more common in women. The secondary neoplasms may originate by contiguity from disease of the mammary gland (most common), pleura, mediastinum, or esophagus; or by metastasis from carcinoma of the liver, uterus, or rectum, or an osteo-sarcoma. The associated lesions are pleurisy, either carcinomatous or sero-fibrinous, with, perhaps, a hemorrhagic effusion; and enlargement of the tracheal, bronchial, cervical, and, rarely, the inguinal, lymph glands.

(*a*) *Symptoms.*—The disease may be quite latent so far as pulmonary symptoms are concerned. Indeed, in any case, the symptoms are variable, depending upon the size, localization, multiplicity, and other characters of the growths. There may be at first simply the evidences of a bronchitis. Ultimately there is *cough*, with the so-called prune-juice or currant-jelly, rarely grass-green, sputum, due to an admixture of blood or altered blood pigment; *pain*, depending largely on the degree to which the pleura is involved; and *dyspnœa,* which may be paroxysmal and caused by pressure on the trachea. The breath and sputum may be offensive because of putrefactive infection of necrotic areas (gangrene).

Certain pressure symptoms will be present according to the size and site of the growth. Thus there may be dysphagia, from pressure on the esophagus; dyspnœa, from pressure on the trachea or large bronchi; hoarseness and aphonia, from pressure on the recurrent laryngeal nerve and consequent vocal-cord paralyses; distention of the veins, cyanosis, and œdema of the face, neck, and one or both of the upper extremities, from pressure on the intrathoracic venous trunks; and displacement of the apex beat, the heart having been dislocated. Fever is occasionally present, cachexia develops, and profuse hæmoptysis has been noted. The *physical signs* are variable, depending partly on the growth, but largely on the presence or absence of pleural effusion. The affected side may be enlarged and immobile if the tumour is large, although the bulging is more commonly due to fluid. The superficial veins of the chest may be distended, and the face, neck, and upper extremities œdematous. There is dulness or flatness, the vocal fremitus is diminished or absent, pleural friction sounds are frequent, and the breath sounds are usually weakened. Less commonly there is bronchial breathing. The cervical or axillary glands may be found enlarged and hard.

(*b*) *Diagnosis.*—Primary growths are difficult to diagnose, and a positive decision must often be withheld until somewhat distinctive symptoms are manifest, viz., the progressive cachexia, the peculiar sputum, the pressure symptoms, the glandular enlargements, and the finding of unilateral, irregular, and indefinite physical signs.

In secondary growths, the occurrence of such pulmonary symptoms as have been described, in conjunction with the presence of malignant disease elsewhere, or a history of the previous removal of a carcinomatous or sarcomatous growth, should enable a diagnosis.

If the pleura is involved, an examination of the aspirated fluid (page 715) may reveal characteristic cells.

(*c*) *Prognosis.*—The disease ends fatally after an average duration of from 6 to 8 months, with extremes of 6 weeks and 2 years.

V. DISEASES OF THE PLEURA

I. **Pleurisy in General.**—Inflammation of the pleura is doubtless, for the most part, a symptom of a condition rather than a separate disease. In the majority of cases it is excited by micro-organisms or their products; less frequently by an intoxication such as that of nephritis or gout; cold, exposure, and injury simply predispose. Pleurisy may be acute or chronic; and, according to pathological character, fibrinous, sero-fibrinous, purulent, or hemorrhagic. One of these may shift into another, or terminate in a chronic form, but in a considerable number of cases the original character of the inflammation persists without change—e. g., a sero-fibrinous pleurisy remains sero-fibrinous.

No one micro-organism is found in acute pleurisy. Thus the *Bacillus tuberculosis* is responsible for many cases, either primary or secondary, although the germs occur in such scanty numbers that they are rarely detected except by the injection of large quantities of the exudate into guinea-pigs. In the pleurisy associated with lobar pneumonia the pneumococcus is found, although the germ may be present without pneumonia; and in that occurring with bronchopneumonia and some cases of lobar pneumonia the streptococcus is the active agent. In purulent pleurisy the pneumococcus or streptococcus may be discovered. Occasionally noted are the *Staphylococcus*, *Bacillus coli communis*, Friedländer's bacillus, typhoid bacillus, diphtheria bacillus, gonococcus, proteus, and the bacillus of anthrax. The three principal bacteriological forms of pleurisy are the *tuberculous*, *pneumococcic*, and *streptococcic*.

II. **Acute Fibrinous Pleurisy.**—*Causes.*—Dry or plastic pleurisy (without fluid exudate) may perhaps occur as a result of cold, but much more commonly it is secondary, especially to lobar pneumonia, less frequently to pulmonary abscess, gangrene, carcinoma, or infarctions; or to pyæmia, rheumatic fever, chronic nephritis, or chronic alcoholism; or originate by extension from pericarditis, peritonitis, or hepatitis. It has a special relation to tuberculosis, occurring sometimes as a primary infection, more commonly as a secondary event to a pulmonary tuberculous focus.

Symptoms.—There are stitch pains in the side, usually in the neighbourhood of the nipple, increased by movement and especially by inspiration, and accompanied by a dry and painful cough. Both cough and respiration are restrained, and the patient bends toward the affected side in order to minimize the pain. For the same reason the breathing is hurried, shallow, jerking, and mainly abdominal in type. Fever is usually present, but seldom exceeds 101°, and in mild cases may hardly rise above the normal.

Physical examination reveals deficient expansion on the affected side, with rubbing, grazing, or crepitating friction sounds. Unless the amount of fibrin is considerable there will be no change in the vocal fremitus or the percussion note.

Diagnosis and Prognosis.—The severe pain of pleurodynia and intercostal neuralgia may closely simulate a dry pleurisy, but the absence of fever and friction sounds in the former two renders the discrimination easy. Recovery is usual after a duration of from a few days to 2 or 3 weeks, but repeated attacks lead to extensive adhesions and thickening of the pleura. The possibility of its occurrence, either at the base or the apex of the lung, as a symptom of tuberculosis, should be borne in mind.

III. **Sero-fibrinous Pleurisy.**—May be the second stage of a dry pleurisy, but there is often a serous exudate from the first.

Causes.—These are essentially the same as for II preceding. The proportion of cases due to *tuberculous infection* is variously estimated at from one third to three fourths, usually secondary to pulmonary tuberculosis, occasionally to tuberculous peritonitis. Other cases arise in connection with rheumatic fever, lobar or bronchopneumonia, typhoid fever, pericarditis, carcinoma or cirrhosis of the liver, and chronic nephritis. Exposure to cold and damp, or injuries of the chest, quickly predispose.

Symptoms.—As a rule the onset is gradual. There is pain in the side, usually referred to the nipple or axilla, less commonly (when the diaphragmatic pleura is involved) to the abdomen or the lower part of the back. It is sharp and catching, and aggravated by coughing, deep breathing, or other movements. When effusion occurs and the inflamed surfaces are separated the pain lessens or disappears. There is dyspnœa, due at first to the pain, later, unless the effusion has occurred very slowly, to pressure of fluid upon the lung. Large, slowly formed effusions, as in latent pleurisy, may be unattended by dyspnœa except on exertion. In severe cases orthopnœa and cyanosis may be manifest. The patient is apt to lie upon the affected side. Cough, usually short and dry, or with a slight, occasionally blood-streaked expectoration, is an early symptom. There is fever, which at the onset may be 102° to 103°, often falling in a day or two to 101° or even less, and usually disappearing, always by lysis, in from 1 to 3 or more weeks. The affected side may be 1 or 2 degrees warmer than the other. The pulse is frequent, and in large effusions may present abnormalities in rhythm and size. Except during the absorption of the fluid, when it may increase to 80 or more ounces, the total daily output of urine is diminished. The bowels are usually constipated. Nausea and vomiting are infrequent initial symptoms.

The **physical signs** are important, and vary with the three stages of the disease: the *dry* stage, the stage of *effusion*, and the stage of *resorption*. The signs of the first stage are those of acute fibrinous pleurisy (see II preceding); of the second as follows:

Inspection and Palpation.—The affected side expands imperfectly, and if the effusion is large there is an increase in its size with obliteration or bulging of the intercostal spaces. Œdema of the chest walls and fluctuation are very seldom manifest. Depending upon the quantity of the effusion the apex beat is displaced to a varying extent: in right-side exudates, to or beyond the left mammillary line in the 4th or 5th interspace, or even into the axilla; in left-side exudates it may lie behind the sternum and be imperceptible, or be carried to or to the outside of the right mammillary line in the 3d and 4th interspaces. It should be remembered that the liver or the spleen may be pushed downward by the effusion, and the displacement may simulate enlargement of these organs. The vocal fremitus is diminished or absent according to the amount of the effusion—a most valuable sign—although it may persist with large exudates if there are conducting bands of adhesion between lung and chest wall, is present in infants especially while crying, and is less reliable in women than in men. *Mensuration.*—In large effusions, after making due allowance for the normally larger size of the right chest, the affected side is found upon measurement to exceed the other by ½ to 1½ inches, especially at the end of expiration.

Percussion.—There is impaired resonance, passing into dulness or absolute flatness as the effusion increases. This is usually first perceived posteriorly. If, by gentle percussion, the upper limit of the dulness is determined, it is found to be at a higher level posteriorly than in front. With a moderate effusion, the patient sitting upright, the line of dulness has the "S" curve. This line, beginning rather low down in the back, passes upward from the spine and curves obliquely across the back to the axillary region, whence it descends anteriorly to the sternum. Movable dulness—a change in the position of the line of dulness obtained by marking it in the mammillary line while erect, and then while lying down—is an unmistakable sign of fluid, but can not be demonstrated in very large or encysted effusions. The dulness or flatness of fluid has a peculiar resistant quality readily recognised by practice. Skoda's resonance, a tympanitic percussion note, which may be elicited under the clavicles and above the level of the fluid posteriorly, is very suggestive of effusion. With large effusions cracked-pot resonance and the tracheal tone may be obtained. On the right side the dulness is continuous with that of the liver; on the left side, in the mammillary line, it may extent downward and abolish the tympanitic resonance of Traube's space. Grocco's triangle is a valuable sign.

With reference to the *amount of fluid*, it may be clinically useful to consider as a *slight* effusion one which causes distinct dulness only at the base posteriorly; a *moderate* one, dulness rising to the 4th rib anteriorly; a *large* one, dulness up to the 2d rib; and a *copious* effusion, dulness to the clavicle, perhaps passing beyond the sternum to the opposite side.

Auscultation.—Commonly the breath sounds over the fluid are weak or absent, but not infrequently in large effusions there is distinct but distant bronchial breathing. At and above the level of the fluid the breathing is broncho-vesicular or even bronchial. The vocal resonance is usually annulled over the body of the effusion, but there may be bronchophony. At the upper line of the fluid there is often a peculiar quavering, hesitating quality of the voice sounds, or the bleating sounds of egophony may be present. In children the voice and breath sounds may have a metallic or amphoric quality, which, with the loud râles sometimes heard, will strongly but incorrectly suggest the presence of a cavity. The ready transmission of the whispered voice (Baccelli's sign) is indicative of a serous rather than a purulent exudate. If the portion of the pleura which overlies the heart is inflamed there may be a pleuro-pericardial friction sound.

Varieties.—(1) *Latent Pleurisy.*—In a certain proportion of cases the onset of the disease is insidious, with little or no pain, a subfebrile temperature, and dyspnœa, slight, and manifested only on exertion. The patient is conscious simply of an indefinite malaise; but on examination a copious effusion may be found, which has occurred so gradually that the intrathoracic organs have been able to adjust themselves to the changed conditions without notable distress.

(2) *Diaphragmatic Pleurisy.*—Pleuritis, usually dry, sometimes serous, and limited to the diaphragmatic pleura, may occur alone, or be a part of a general pleurisy. The pain is sharp and severe, and is felt in the epigastrium, especially in a line from the end of the 10th

rib to the ensiform cartilage. It is aggravated by pressure upon the insertion of the diaphragm at the 10th rib, and by breathing or swallowing. The abdominal muscles are fixed, and the respiration is of the thoracic type, sometimes with severe dyspnœa and, occasionally, anginal attacks, vomiting, and severe cough. There may be fever, perhaps of high degree. An entire absence of physical signs with the presence of severe subjective symptoms is characteristic of this variety of pleurisy. Rarely the inflammation is purulent.

(3) *Interlobar Pleurisy.*—In all cases of acute pleurisy the serous surfaces lying between the lobes are inflamed, and not very uncommonly they become adherent, thus inclosing fluid, either serous or purulent, between the apposed portions of the lung. This takes place most frequently close to the root of the right lung, between the upper and middle lobes. Such a collection, usually small in amount, may perforate a bronchus with resulting purulent sputum, an event which, as the other symptoms are quite indefinite and the history of a previous pleurisy is sometimes absent, may be the first suggestive evidence of the condition. The diagnosis is extremely difficult, and may require a number of exploratory punctures.

(4) *Encysted Pleurisy.*—The effusion, sometimes serous, more commonly purulent, is encapsulated by the formation of limiting adhesions between the pleural surfaces. The loculi or sacs may be present in any part of the chest, often in that part of the pleura between the midaxillary line and the spine, or between the base of the lung and the diaphragm. With a pleuritic history suggestively circumscribed dull areas may be, but are seldom, encountered. Usually the diagnosis is beset with difficulties, and is made by an assiduous use of the aspirating needle in suspected cases presenting indefinite pleuritic symptoms and signs.

(5) *Tuberculous Pleurisy.*—See page 811.

Diagnosis.—In the majority of cases there is little difficulty in making a correct diagnosis from the symptoms, particularly the physical signs. The following conditions may cause uncertainty:

(1) *Pneumonia.*—The bronchial or amphoric respiration and bronchophony which may be present in some cases of pleurisy, especially in children, may be very perplexing, and a pneumococcus pleurisy at the time of onset may closely simulate a lobar pneumonia. In the latter disease there is an initial rigour, the fever is higher, the prostration more decided, the dyspnœa of greater intensity, and there is rusty sputum. Labial herpes is suggestive, and the unilateral flush on the cheek is not seen, as a rule, in pleurisy. Moreover, in pneumonia the dulness is less resistant, the vocal fremitus and vocal resonance are increased, not diminished or absent; there is no dis-

placement of heart, spleen, or liver, and the intercostal spaces do not bulge. Finally, the crucial and safe test of exploratory puncture is decisive, and should always be employed in a doubtful case. It should, of course, be remembered that pneumonia and a pleuritic effusion may coexist (pleuro-pneumonia).

(2) *Hydrothorax.*—The majority of the physical signs are identical with those of pleurisy, but the absence of fever, pain, and friction sounds, together with the history of disease of the heart or kidney in hydrothorax, usually enables a ready differentiation.

(3) *Pericardial Effusion.*—This, if extremely copious, may closely simulate a left-side pleural effusion. But in pericardial effusion there is pulmonary resonance at the base, tympanitic or dull tympanitic resonance in the axilla and around the border of the distended sac, and the heart is not displaced to the right of the sternum. Moreover, the dyspnœa is extreme in comparison with the apparent amount of effusion, the pulse is small and paradoxical (page 397), and there is often a history of antecedent rheumatic fever.

(4) *Intrathoracic Growths.*—Carcinoma of the lung or pleura, or hydatid cysts of the pleura, may cause dulness, absent or weak breath sounds, and displacement of the apex beat, thus closely simulating a pleural effusion. But the dulness is more circumscribed and lies most commonly in the middle or upper part of the chest, while the vocal fremitus and resonance are often preserved, thus resembling consolidation rather than fluid. The history, too, is apt to differ from that of a pleurisy. It is to be remembered, however, that pleural effusion is a common concomitant of such growths (page 920), and, if present, a differential diagnosis is extremely difficult or impossible until pressure symptoms become manifest.

(5) *Hepatic Abscess or Cyst.*—This, when of sufficient size and suitably located, may push the diaphragm high up and cause dulness and weak or absent respiration at the right base, thus simulating a pleural effusion. The upper line of dulness in such cases is immovable and often curved, convexity upward, and a friction sound is audible over the dull area, which would not be the case if the pleural surfaces were separated by fluid.

Course and Prognosis.—In non-tuberculous cases the fever lasts from 1 to 3 weeks, according to the severity of the attack, but the non-febrile stage is extremely variable. Small effusions may disappear very rapidly, but the resorption of large effusions may require many weeks or several months if not aspirated. In tuberculous pleurisy the resorptive process is apt to be slow. If a sero-fibrinous pleurisy becomes purulent the fever persists. As the effusion is resorbed the physical signs gradually shift back to the normal, and rubbing, creaking, or crepitating friction sounds become audible when the previously separated pleural surfaces are again in contact. Dulness and weak breath sounds may persist at the base for a few or many months. The *prognosis* is usually good, and even in tuberculous cases complete recovery may take place. In rare instances sudden death may occur, due possibly to œdema of the opposite lung, cardiac degeneration, or embolism or thrombosis of the pulmonary artery or of the heart itself.

IV. **Purulent Pleurisy.**—(*a*) *Causes.*—Empyema is a rare sequence of acute sero-fibrinous pleurisy, except in children, in whom effusion, if not purulent at the onset, may rapidly become so. It is common as a secondary lesion in infections, especially scarlet fever, pneumonia, pulmonary tuberculosis, or pyæmia, rarely in measles, pertussis, and typhoid fever. Finally, it may follow perforation of a tuberculous or gangrenous cavity or a subphrenic abscess into the pleura, carcinoma of the lung or esophagus, fracture of a rib, or a penetrating wound of the chest.

(*b*) *Symptoms.*—The onset may be sudden and severe, with cough, dyspnœa, and pain in the side, subsequently becoming less marked. More commonly it comes on insidiously, as a secondary event, and the thoracic symptoms are mild or even lacking. Fever is always present, usually irregular and intermittent, and often accompanied by chills and sweats. There is always a leucocytosis, often of high grade; and the urine may contain indican and albumoses, findings indicative of a purulent rather than a serous effusion.

The *physical signs* are identical with those of sero-fibrinous pleurisy (page 924), except for certain suggestive modifications. In empyema there may be distention of the veins and œdema of the chest wall, signs which are extremely rare in serous effusions. The enlargement of the affected side is much greater, and bulging of the interspaces much more common, especially in children, and in them the breath sounds over the exudate may be of a distinctly bronchial character. The whispered voice is not as a rule transmitted through a purulent effusion (Baccelli's sign). In neglected cases a fluctuating, reddened pouting or protrusion may be noted, indicating an imminent external discharge (*empyema necessitatis*), or a fistulous opening may already be present. This takes place most commonly in front, at the 5th interspace, but may occur in the 3d, 4th, or 5th space, or posteriorly, at the angle of the scapula. There may be multiple openings. The displacement of the liver and heart is greater in empyema than with the same amount of serous effusion. In certain cases of empyema, very rarely in serous pleurisy, there is a distinct pulsation, synchronous with the heart's action, and almost invariably in left-side exudates. It may be observed in 2 or 3 interspaces, or over the front and side of the chest, seldom posteriorly; or the protruding tumour of an empyema necessitatis may pulsate. The finding of a tender point, by deep pressure, especially along the line of the interlobar fissures, is an important sign in localizing a small collection of pus.

(*c*) *Course and Prognosis.*—Very seldom the purulent fluid is absorbed, with resultant great thickening of the pleura and retraction of the chest wall. Perforation of the chest wall is common, and in rare cases a communication may be established with the stomach, esophagus, peritoneum, or pericardium. The pus may enter a bronchial tube either by way of a perforation, or by percolation through the spongy lung tissue when its protecting pleural covering has become necrotic. The pus has been known to pass down along the spine to the iliac fossæ and point at the usual situations of a lumbar or psoas abscess.

The *prognosis* depends somewhat on the nature of the primary disease. A pneumococcus empyema has a favourable outlook; tuberculous and streptococcic empyemas are less promising. Drainage by way of a perforated bronchus is, in a considerable number of instances, followed by recovery; so also with perforation of the chest wall. Much depends on an early diagnosis and prompt surgical treatment. That no case, however desperate, should be deprived of operation is an axiom the truth of which has been repeatedly verified. The prognosis is rather better in children than in adults, but judging from personal experience the percentage of recoveries, taking all cases together, should be large.

(*d*) *Diagnosis.*—The discrimination between sero-fibrinous pleurisy and empyema depends upon exploratory puncture. A failure to discover micro-organisms in a creamy pus is suggestive of the tuberculous nature of the inflammation. A pulsating pleurisy may be differentiated from an aneurism by the pleuritic history, the absence of bruit and diastolic shock, and the location of the swelling farther to the left than is usual in aneurism.

V. **Hemorrhagic Pleurisy.**—A bloody exudate may be due to tuberculosis or carcinoma involving the pleura; hepatic cirrhosis or chronic nephritis; the severer types of the acute infections when complicated by pleurisy; accidental wounds of the lung during aspiration; and occasionally, in otherwise healthy persons, to unknown causes. Pure blood in the pleural cavity (*hæmothorax*) may be due to penetrating wounds of the chest; the rupture of an aneurism; or the pressure of a tumour on the thoracic veins.

VI. **Chronic Pleurisy.**—Two varieties are recognised:

Chronic Pleurisy with Effusion.—This is usually a sequence of acute sero-fibrinous pleurisy; less commonly the condition may be found without a history of a definite time of onset. The *physical signs* are identical with those of acute pleurisy with effusion. Attention may be drawn to the condition only by slight dyspnœa upon exertion and a subfebrile evening rise of temperature. The effusion may remain for months or years without diminution in amount or change in character. If, as may occur, especially in children, it becomes purulent, the symptoms of empyema supervene.

Chronic Dry Pleurisy.—Adhesive pleurisy may be a sequel of acute or chronic pleurisy with a serous or purulent effusion, the fluid exudate having been resorbed or discharged; or it may be primary and dry from the beginning, and perhaps of tuberculous origin. Bronchiectases may develop. A primitive dry pleurisy may originate a chronic interstitial pneumonia, and is sometimes associated with adhesive (proliferative) peritonitis and pericarditis.

The *subjective symptoms* are slight. There may be occasional stitch pains and dragging sensations in the affected side, but the general health is often unimpaired for years.

The *physical signs* in the milder degrees of chronic adhesive pleurisy comprise slight retraction of the affected side, with impaired expansion, moderate dulness, and weakened breath sounds. In the severer cases, especially those which follow empyema necessitatis, there is marked shrinking of the affected side, the spine is curved, the shoulder sags, and the expansion is poor or absent. The heart may be drawn to the right or left by the retraction of the fibrous tissue. The vocal fremitus is diminished or absent, and there is moderate or well-marked dulness, with a weak respiratory murmur and occasional friction sounds, over the lower half of the side involved. In rare instances there may be unilateral flushing or sweating of the face or dilatation of the pupil, probably due to an involvement, in the indurative process, of the first thoracic ganglion at the apex of the pleural cavity.

VII. **Hydrothorax.**—A non-inflammatory pleural effusion is usually a part of a general dropsy due to cardiac disease, in which it is as a rule unilateral and on the right side; to renal disease, in which the hydrothorax is almost always bilateral; and, also usually bilateral, to poverty or disease of the blood, as in pernicious anæmia, leucæmia, scurvy, malaria, syphilis, carcinoma, and severe chronic diarrhœa or dysentery. The *symptoms*, aside from those due to the causative affection, are dyspnœa, cyanosis, orthopnœa, and pseudo-asthmatic paroxysms, their severity depending on the amount of the fluid. The *physical signs* are mainly those of pleuritic effusion (page 942).

VIII. **Pneumothorax.**—Air alone is rarely present in the pleural cavity, the latter almost always containing serum (sero- or hydropneumothorax), or, much more commonly, pus (pyopneumothorax) in addition to its gaseous content.

Causes.—The most common cause is a perforation of the lung due to the rupture of a tuberculous cavity or cheesy focus (90 per cent of all cases), or of cavities due to inhalation pneumonia, abscess, gangrene, bronchiectasis, suppurating bronchial glands, hemorrhagic infarctions, or hydatid cysts. It may be caused by perforation of the lung by an empyema, or perforation of the pleural cavity by an air-containing subphrenic abscess or abscess or carcinoma of the esophagus. The growth of the gas bacillus in a pleural exudate is a rare cause; so also is the rupture of normal or emphysematous air vesicles, especially though not solely as the result of violent coughing or muscular exertion in lifting or straining. Penetrating wounds of the chest, including as a rarity exploratory puncture, may be responsible. It is most frequent in adult males, and most common on the left side (2 to 1).

Symptoms.—Rarely the onset may be gradual and without urgent symptoms. Usually there are sudden and severe pain in the side, intense dyspnœa, often with cyanosis, and the pulse is frequent and weak. In the gravest cases, when the perforation is large and runs obliquely so that it has a valvular action, the pleural cavity becomes tensely distended with air, the lung is totally compressed, and symptoms of collapse—syncope, sweating, subnormal temperature, and thready pulse—ensue, perhaps soon followed by death.

The *physical signs* are characteristic, depending upon the simultaneous presence of air and fluid in the pleural cavity, for in the large majority of instances pleurisy with effusion, seldom serous, usually purulent, succeeds upon the perforation. The affected side is markedly distended and immobile. The vocal fremitus is diminished or absent. The percussion sounds are variable, depending partly upon the size and shape of the air-containing cavity, largely upon the degree of tension of the contained air and the presence or absence of an effusion. Over the fluid at the base there is dulness or flatness. Over the air there is usually a tympanitic note, often with a somewhat amphoric quality; or there may be hyperresonance; or dull tympany (Skodaic resonance); or, when the tension is extreme, actual dulness—a fact to be remembered. Wintrich's change of sound (page 438) or cracked-pot resonance may be elicited when there is a free communication between a bronchus and the pleural cavity. The transition from the upper limit of fluid dulness to the tympanitic area is abrupt, and movable dulness is more easily detected than in simple pleural effusions. The voice sounds are ringing and amphoric. The heart, liver, and spleen are displaced to a greater extent than in serous pleurisy, and in left pneumothorax the cardiac dulness may disappear. Usually the respiratory murmur is diminished or absent, but there may be distinct, but distant and generally weak, amphoric respiration. Ringing râles may be heard, also metallic tinkling upon coughing or deep inspiration. The succussion sound and the coin sound are extremely characteristic phenomena.

Differential Diagnosis.—The history and the physical signs are so distinctive that a mistake in diagnosis is not common.

An extremely large *pulmonary cavity* may afford a tympanitic percussion note and ringing or amphoric râles, but the succussion sound and, except in rare instances, the coin sound are absent. Moreover, the heart, liver, and spleen are not dislocated, vocal fremitus is usually increased, and there is depression rather than bulging of the interspaces. A differentiation of pneumothorax from the rare *subphrenic pyopneumothorax* (page 907) may be required.

A *distended stomach*, by giving rise to tympanitic percussion over the left thorax, with succussion sounds and metallic tinkling, may simulate a left pneumothorax, but the thorax itself is not distended, and a careful physical examination, together with the history, will usually prevent confusion.

Because of the occasional occurrence of a dull percussion note over a pneumothorax a diagnosis of *pleurisy with effusion* may be made; or, on the other hand, an unusually sonorous percussion sound in *emphysema* may suggest pneumothorax; but in both instances the succussion sounds, metallic tinkling, and coin sounds are absent.

Diaphragmatic hernia, a very rare condition, which is usually due to violent exertion or a crushing injury, but is occasionally congenital, may exactly simulate pneumothorax, the air-containing stomach and intestines lying within the pleural sac. A history of severe traumatism, the presence of cooing or mumbling sounds (normal to the abdomen) over the chest, and perhaps the abrupt disappearance of the thoracic symptoms consequent upon a sudden return of the protruding viscera to below the diaphragm, may suggest the correct diagnosis.

Prognosis.—In the cases due to tuberculosis, gangrene, or abscess of the lung, death usually takes place within a few weeks. Subsequent to empyema the prognosis is more favourable, and the cases occurring in previously healthy individuals frequently recover. The mortality in all cases is about 70 per cent (West).

IX. **New Growths of the Pleura.**—These almost always arise by extension from carcinoma of the lung, less commonly by metastasis from the lung or mammary gland. Primary malignant disease of the pleura (endothelioma) is of rare occurrence.

The symptoms and signs of *secondary* disease of the pleura are those of carcinoma of the lung (page 938) *plus* the evidences of a chronic pleurisy, with or without effusion. The previous occurrence of malignant disease of the breast, and the finding of many mitotic cells in the aspirated fluid suggest the nature of the pleuritic disease. *Primary* carcinoma of the pleura affords simply the evidences of chronic pleurisy, with or without effusion. Irregular slight fever with occasional subnormal falls may be present; there is pain, rather greater than that of pleurisy; and cachexia may develop. The diagnosis is rarely made during life, beyond a suspicion.

X. **Diseases of the Mediastinum.**—Under this heading are comprised *tumours*, *abscesses*, and *enlarged glands*.

(I) MEDIASTINAL TUMOURS.—The most common of these are sarcoma, carcinoma, and lymphoma. The principal points of origin are the lungs and pleura, the lymph glands, and the remains of the thymus gland. Males between 30 and 40 years of age are most commonly affected.

Symptoms.—The symptoms are due to intrathoracic pressure, and the disease is latent until the growth is of sufficient size to encroach upon the surrounding structures.

Dyspnœa is constant, apt to occur early in the disease, and is usually due to pressure on the recurrent laryngeal nerves or the trachea, less commonly to displacement of the heart or great vessels, or the presence of a large pleural effusion. Occasionally the dyspnœa may be paroxysmal. A brazen cough, often violent and paroxysmal, and due to pressure upon the recurrent laryngeal nerves ("aneurismal cough"), usually accompanies the dyspnœa; a husky or hoarse voice, or aphonia, also may be manifest. The pulse may be either abnormally slow or rapid, owing to pressure or irritation of the pneumogastric or sympathetic nerves; and, rarely, sympathetic involvement may cause unequal pupils or localised flushings. There are often evidences of pressure upon the vessels (or thrombosis), most commonly the superior vena cava and its tributaries. The visible veins of the head, neck, upper chest, and upper extremities become distended, the parts may be cold, cyanotic, and œdematous, and the fingers may be clubbed. There may be bulging of the sternum or a protruding, perhaps pulsating, swelling over or at one side of the sternum, due to involvement and erosion of the bone, particularly if the growth be a lymphoma. The cervical glands may be enlarged. There may be unilateral retraction of the chest, and the apex beat may be thrust away from its normal position. There are dulness and diminished or absent vocal fremitus over the areas at which the growth is in contact with the chest wall; and over the same areas the breath sounds are absent or weak, rarely bronchial, the vocal resonance is usually lacking, and the sounds of the heart are not transmitted. There may be a systolic bruit over a protrusion. In many cases the signs of pleural effusion are superadded.

The *site of the growth* may be suggested by a certain grouping of symptoms and signs, as follows:

Anterior Mediastinum.—The physical signs of pressure are prominent: mainly venous distention, œdema, voice changes, dyspnœa, or pupillary inequalities, with enlarged cervical glands and bulging and erosion of the sternum. The growth may be palpable in the episternal notch. It commonly originates in the thymus gland.

Middle and Posterior Mediastinum.—The physical signs are slight as compared to the symptoms, of which the most important are early and persistent dyspnœa, brazen paroxysmal cough, dysphagia, occasional fever, and frequent cachexia. There may be œdema of the upper abdominal wall from pressure on the azygos veins. The growth here usually originates in the lymph glands.

Pleura and Lung.—If the growth starts in the lung or pleura the pressure symptoms are, at first certainly, slight, and the clinical picture is that of a pleurisy, with pain, cough, and effusion. The pain increases, the cervical glands may enlarge, the cough persists, and there may be a bloody mucoid expectoration. The patient rapidly becomes anæmic, thin, and cachectic.

Differential Diagnosis.—Mediastinal tumour is to be separated from *pleurisy with effusion* by the fact that in the latter the physical signs are limited to the lower rather than the upper half of the chest, there is fever, and the onset is as a rule more acute; from *pericarditis with effusion*, by the pyriform rather than irregular dulness, the finding of the apex beat in, rather than outside of, the area of dulness, and the usual presence of fever, in pericarditis.

Most commonly, the differential diagnosis wavers between a mediastinal growth and thoracic aneurism—a problem often difficult, and not seldom impossible, of solution. Both conditions give rise to pressure symptoms which may be identical. The diastolic shock, ringing aortic second sound, tracheal tugging, and, if an external swelling is present, expansile pulsation, of aneurism are absent in mediastinal tumours. In the latter pain is less severe, and cachexia and enlargement of the cervical and axillary glands are much more common. Furthermore, aneurisms usually, mediastinal tumours rarely, have a duration of more than 18 months.

(II) MEDIASTINAL ABSCESS.—A rare condition, which may be *acute*, usually due to injury, less commonly to infectious diseases, especially erysipelas, smallpox, measles, and rheumatism; or *chronic*, generally of tuberculous origin; and is most frequently seated in the anterior mediastinum. The majority occur in males.

Symptoms.—In *acute* abscess there is sharp, often throbbing, substernal pain, with fever, perhaps also with chills, profuse sweats, and prostration. If the abscess is of sufficient size there may be cough and dyspnœa from pressure. If, as may happen, the abscess points at an intercostal space, or erodes and perforates the sternum, a fluctuating and pulsating swelling may appear, or it may become palpable in the episternal notch. Previous to the appearance of a swelling, dulness upon percussion over the seat of the abscess may be the only physical sign. The pus may discharge into the trachea or esophagus after perforation, and in rare instances has made its way into the abdomen. A *chronic* abscess may afford signs similar to those of mediastinal growths.

Differential Diagnosis.—A fluctuating and pulsating abscess may be differentiated from aneurism by the absence of diastolic shock, expansile pulsation, and murmur; and in the acute abscess by a history of injury and the presence of chill, fever, and sweat. Exploratory puncture with a fine needle is allowable. An acute abscess is separated from a mediastinal growth by the febrile symptoms; a chronic abscess, by puncture. In many abscesses, especially the chronic form, spontaneous cure occurs by inspissation of the pus.

(III) MEDIASTINAL LYMPHADENITIS.—Inflammation of the mediastinal lymph glands may be simple or suppurative.

Simple.—The glands are much swollen in influenza, pertussis, measles, tuberculosis, and broncho-pneumonia; and, to a lesser degree, in all inflammatory affections of the lungs and bronchi. Whether enlargement of these glands can be determined by the presence of dulness over the upper sternum, or over the upper part of the interscapular region and the dorsal vertebræ as far down as the fourth (GUENEAU DE MUSSY), is open to question.

Suppurative.—Suppuration of the mediastinal glands is most commonly tuberculous, but may follow the preceding. There are no physical signs, but the pus may discharge, after perforation, by way of a bronchus or the esophagus, or, as in a reported case, perforate the aorta. This condition is to be remembered as a possible explanation of a single profuse expectoration of pus during the course of pulmonary tuberculosis, as in a case under my care.

SECTION IV

DISEASES OF THE CIRCULATORY SYSTEM

(See also Section XXXI, Part I, pages 326 to 411)

I. DISEASES OF THE PERICARDIUM

I. **Pericarditis in General.**—Inflammation of the pericardium occurs as a result of irritation from materials contained in the blood, or by extension of an inflammatory process from neighbouring structures, or in consequence of injury.

From 30 to 70 per cent of the cases of pericarditis are caused by the toxine of rheumatic fever or acute tonsilitis. Less commonly it is due to pyæmia and scarlet fever; still more rarely to smallpox, influenza, diphtheria, and measles; and may be of tuberculous origin. It is also associated with gout; nephritis, especially the chronic interstitial form; scurvy, purpura hæmorrhagica, and diabetes. By extension it may complicate pleuro-pneumonia, especially in children and topers; pleurisy, particularly if tuberculous; mediastinal tumour or abscess; ulcerative endocarditis, and cardiac valvular disease, especially at the aortic orifice; disease of the vertebræ, ribs, and sternum; disease of the bronchial glands and the esophagus; ruptured aortic aneurism; and even result, by perforation into the pericardium, from disease of the abdominal organs. The following varieties of pericarditis are recognised: *plastic* or fibrinous, *sero-fibrinous*, *suppurative*, *hemorrhagic*, and *adhesive*. One of these forms may shift into another. Different micro-organisms have been found in the exudate, although attempts to discover them are not always successful. The *Staphylococcus*, *Streptococcus*, *Pneumococcus*, *Bacillus coli*, and tubercle bacilli have been encountered.

II. **Acute Plastic Pericarditis.**—*Symptoms.*—This, the most common variety, is usually secondary to some existing disease. In the majority of cases special subjective symptoms are lacking and only a routine examination of the heart enables its recognition. In other instances there may be vague sternal discomfort or constriction, or actual pain, usually slight, exceptionally severe and anginose in type. It is felt over the pericardium, occasionally extending to the left arm; or at the ensiform cartilage and over the upper abdomen. It is seldom increased by pressure. There may be dyspnœa, palpitation, and perhaps a weak and irregular pulse. Fever, rarely exceeding 102° to 102.5°, is present. The *physical signs* are friction fremitus, not always present and usually felt over the lower præcordial area, and pericardial friction sounds. These sounds, superficial and intensified by pressure, usually double, rarely single or triple, are synchronous with, but last longer than, the first and second sounds of the heart. The usual points of maximum audibility, which often change with position, are in the fourth and fifth interspaces and the neighbouring parts of the sternum, or over the aortic area; less frequently at the apex or along the whole length of the sternum. As a rule, their audibility is limited to small areas; and they vary from time to time in position and character. The quality of the sound is described as grating, rubbing, whizzing, or creaking. There may be a pleuro-pericardial friction sound.

Diagnosis.—The pericardial friction sounds are so distinctive that mistakes are not common. The to-and-fro friction sound may suggest aortic incompetency, but the superficial character of the pericardial rub, its variability, increased intensity upon pressure, and the lack of exact correspondence with the events of the cardiac cycle, together with the absence of cardiac hypertrophy and "shot" pulse, will separate it from the valvular lesion. Very seldom there is a fine systolic crepitation at the base or the apex, due perhaps to abnormal dryness or calcareous changes of the pericardium without inflammation.

Course and Prognosis.—Plastic pericarditis is never fatal, but often constitutes the first stage of the sero-fibrinous form; or may cause (especially tuberculous) extensive thickening and adhesion.

III. **Sero-fibrinous Pericarditis.** —This occurs most frequently as a sequel of plastic pericarditis in connection with acute rheumatic fever, sometimes preceding the articular symptoms, more rarely appearing without antecedent joint inflammations; also with nephritis, pulmonary tuberculosis, and septicæmic conditions.

Symptoms.—In many cases, usually those secondary to some already existing disease, the onset is insidious, with an entire absence of subjective symptoms. In other, especially primary, cases there is præcordial pain, discomfort, or distress, aggravated by pressure over the lower sternum. As effusion occurs, dyspnœa, left-side decubitus, even orthopnœa, become manifest. The face is cyanotic and anxious, the breathing is laboured, often irregular, the pulse small and perhaps paradoxical. Aphonia, irritative cough, dysphagia, and distention of the veins of the neck may result from the pressure of a large effusion. There is fever, usually irregular, and varying from 101° to 103°. In the graver cases there may be constant restlessness and persistent insomnia, followed by delirium, stupor, and coma. The condition may be such as to resemble delirium tremens, or the patient may exhibit great mental depression.

The *physical signs*, after effusion has taken place, are numerous and important. There is præcordial bulging in children, even with a moderate exudate; and in adults, if the effusion is copious, the anterior and lateral portions of the chest are enlarged, the intercostal spaces are prominent, the nipple is elevated, and there may be œdema of the walls. The left lobe of the liver may be depressed by the weight of the fluid, and in consequence the epigastric region becomes distinctly prominent. The apex beat and the thrust of the heart become less and less palpable and may finally disappear as the effusion increases—an important sign. Friction fremitus and friction sounds may persist at the base, rarely at the apex, and are most readily elicited when sitting upright, but are ordinarily not heard over the body of the heart in any but slight effusions. Percussion shows a much increased triangular area of dulness, within which a feeble apex beat is felt. Dulness in the fifth interspace to the right of the sternum is present even with slight effusions; and in large effusions there may be a limited area of slight or marked dulness in the left infrascapular region. The lung having been forced back and compressed, there may be dull tympany, with weak or bronchial breathing in the axilla to the left of the triangular area of dulness. The heart sounds at the apex are muffled, faint and distant; at the base the second sounds, especially the pulmonary, may be clear and accentuated. There may be a systolic endocardial murmur.

Course and Prognosis.—The acuteness and rapidity of the disease are variable. The effusion may reach its height within 2 or 3 days and the process of resorption then begin; or it may require several weeks of gradual increase to attain the maximum, as in the so-called subacute and chronic cases. Rheumatic effusions may disappear in 2 or 3 weeks after absorption has begun. In large and fatal effusions death occurs by asthenia at the end of 2 or 3 weeks; or, in severe cases attended by delirium and extreme restlessness, in a week or 10 days. In moderate sero-fibrinous effusions recovery is the rule, with resulting adhesion between the pericardial surfaces.

Differential Diagnosis.—The insidious onset of the disease leads to many diagnostic errors of omission, and unless the case has been watched from the beginning, it may be difficult and at times impossible, especially if the chest walls are unusually thick, to distinguish between a copious pericardial effusion and a dilated heart, possibly also a pleural exudate. As a rule, with proper opportunities for observation from an early period, the diagnosis is readily made. The differential points are as follows:

(1) *Pleurisy with Effusion.*—See page 945.

(2) *Dilatation of the Heart.*—In this, as compared with pericar

dial effusion, the apex beat is visible, diffused, and wavy, and the shock or impulse of the heart is more clearly palpable; the area of dulness is not so distinctly triangular, it does not vary with change of position, and, although it may be quite as extensive as that of an effusion, the impulse of the heart will be visible or palpable over its entire extent. Only in rare instances is the dilatation so great as to compress the lung and cause a dull tympanicity in the axillary region. Instead of its distant muffled character in pericardial effusion, the first sound, although short and weak, is distinctly and clearly heard. Friction sounds and fremitus are absent. Moreover, there is usually no pain or fever, and commonly there is a history of chronic cardiac disease.

IV. **Purulent Pericarditis.**—*Causes.*—May follow a serous pericarditis, or may be purulent from the outset, particularly when due to tuberculosis, sepsis, or the acute infections.

Symptoms, Diagnosis, and Prognosis.—The physical signs are identical with those of a serous pericarditis; so also are the symptoms, except that there may be recurring chills, an irregular or suppurative type of fever, and a greater degree of prostration. The only positive evidence of empyema of the pericardium is obtained by exploratory puncture, but the special symptoms mentioned, together with the presence of an antecedent affection which is capable of causing it, may lead to a correct conjecture as to the purulent nature of the effusion. The prognosis is unfavourable. The pus may discharge into the pleura, esophagus, stomach, or bronchi.

V. **Hemorrhagic Pericarditis.**—The effusion of tuberculous or cancerous pericarditis, whether serous or purulent, is especially apt to be hemorrhagic, so also with the effusion occurring in those who are old, debilitated, or the subjects of scurvy, purpura, etc.

VI. **Chronic Adhesive Pericarditis.**—*Varieties.*—Usually a sequel of the acute forms. If there is simply adhesion between the visceral and parietal layers of the pericardium, there are, as a rule, no recognisable symptoms indicative of the condition. But if, in addition, the chronic inflammatory process extends to the mediastinal and pleural tissues external to the parietal layer of the pericardium, and the latter in consequence becomes adherent to the pleura and the chest walls (indurative mediastino-pericarditis), extreme cardiac hypertrophy and dilatation may result, especially in persons under 30 years of age. In some cases, especially in children, there may be an associated proliferative peritonitis, involving particularly the peritoneal covering of the liver and spleen.

Symptoms.—The condition is often latent and discovered only at autopsy. In other cases the symptoms are those of cardiac hypertrophy and dilatation and sequent myocardial failure.

The *physical signs*, when present, are as follows: Inspection may show a marked bulging of the pericardium, and the wavy cardiac impulse may be visible over a large area. There is systolic retraction of the apex, and if the heart is extensively adherent to the diaphragm a systolic tug over the left 7th and 8th ribs anteriorly, and the left 11th and 12th ribs posteriorly, may be seen. For a similar reason the diaphragm may not be able to descend during inspiration, and the usual epigastric respiratory movement is lacking. There may be diastolic collapse of the jugular veins (Friedreich's sign), but this is of little value. The apex beat may not change position when the patient is turned upon his left side, the adhesions preventing. There may be a diastolic shock, a sudden rebound of the heart walls during diastole, after having been drawn together during systole against the resistance of the adhesions. The pulsus paradoxus may be present. The area of cardiac dulness is much increased, and its outlines above and to the left may not be changed by deep inspiration, the pleuro-pericardial adhesions preventing the normal intrusion of the lung between the heart and the chest wall. Murmurs are not necessarily present, but they may be heard as evidence of pre-existing valvular disease, or are due to relative insufficiencies at the mitral, tricuspid, and pulmonary openings. Rarely there is a distinct presystolic murmur. Dry or crackling râles (mediastinal friction sounds) may be heard occasionally along the sternum. The liver may be greatly enlarged, with or without ascites.

VII. **Hydropericardium.**—A non-inflammatory effusion (dropsy of the pericardium) is usually a part of the general dropsy of renal or cardiac disease, and may be associated with hydrothorax; rarely it occurs alone, especially in scarlatinous nephritis. It may also be caused by the pressure of an aneurism or mediastinal growth, or by thrombosis of the cardiac veins. The *physical signs* are those of pericarditis with effusion, excepting the friction sounds, pain, and other evidences of inflammation. Dyspnœa is practically the only symptom, and the condition is often unrecognised.

VIII. **Hæmopericardium.**—Pure blood in the pericardium occurs as a consequence of the rupture of an aortic aneurism, aneurism of the coronary arteries, and rupture of the heart; or from penetrating wounds of the heart. If due to the bursting of an aneurism, death occurs rapidly from mechanical interference with the action of the heart. In wound or rupture of the heart the blood may escape slowly and life be prolonged for hours or days with dyspnœa, signs of effusion, and steady failure of the heart muscle.

IX. **Pneumopericardium.**—Air in the pericardium is usually caused by a penetrating wound. Less commonly it is due to a perforation from the lungs, especially of a tuberculous cavity, or an empyema, or ulcerative or malignant disease of the esophagus or stomach; and in some cases to the growth of the gas bacillus in an existing pericardial effusion. The pericardium always becomes inflamed as a result of a perforation, and effusion, usually purulent, results. The *symptoms* are substantially those of pericarditis with effusion. The *physical signs*, when gas and fluid coexist in large amount, are: An area of dulness over the fluid and of marked tympanicity over the gas, the dull area changing its site and shape to a notable degree with a change of posture. The apex beat may be feeble or absent, and the heart sounds weak and distant. Friction sounds may be heard, together with the curious and characteristic splashing or "water-wheel" sounds (page 383).

II. DISEASES OF THE HEART

I. **Acute Endocarditis.**—Acute inflammation of the lining membrane of the heart is usually confined to the valves, is almost invariably a secondary process, and in many, if not all, cases is caused by micro-organisms, cold and exposure predisposing. The most common organisms are the streptococcus, staphylococcus, pneumococcus, and gonococcus; less frequently the diphtheria bacillus, *Bacillus typhosus*, *Bacillus coli*, influenza bacillus, and others.

Clinically two forms are recognised, *simple endocarditis* and *malignant* or *ulcerative endocarditis*, although the pathological process is practically the same in both and the differences are mainly in severity and malignancy.

(I) Simple (Benign) Acute Endocarditis.—*Causes.*—Most frequently associated with acute rheumatic fever, tonsilitis, chorea, pneumonia, scarlet fever, and phthisis; less commonly with smallpox, chicken-pox, typhoid fever, measles, erysipelas, and diphtheria. Occurs as an intercurrent or terminal event in the subjects of carcinoma, nephritis, gout, and diabetes; or during a chronic endocarditis.

Symptoms.—Except in a small minority of cases the disease is latent, and there are no symptoms pointing to the heart. There may be fever, or an increase in that which may be already present as a part of the causative disease, with palpitation and increased frequency, possibly also irregularity, of the pulse. Præcordial pain and dyspnœa are of rare occurrence. The *physical signs*, unfortunately, are extremely unreliable. Unless there is hypertrophy or dilatation due to pre-existing valvular disease, or dilatation caused by an associated acute myocarditis, inspection, palpation, and percussion will reveal nothing abnormal. Auscultation usually discovers a soft, blowing, systolic murmur, heard most commonly over the mitral area, less frequently over the aortic cartilage, and not transmitted to any great distance. If the heart has been watched from the beginning there may have been a roughening or murmurish prolongation of the first sound, slowly evolving into a murmur.

Diagnosis.—The presence of one of the causative diseases is of great importance in making a probable diagnosis. A soft blowing murmur may be present in many acute febrile diseases without endocardial inflammation, but it is more likely to indicate endocarditis if it is heard only or most distinctly over the mitral area; less likely if over the aortic area; and least likely if over the pulmonary area. The most reliable physical sign, in conjunction with the history, is the observed development of a roughened, murmurish first sound into a well-marked systolic mitral murmur. It is hardly possible to recognise attacks of simple endocarditis recurring in a case of chronic valvular disease, as the physical signs of the latter are practically unaltered by the supervention of an acute benign valvulitis.

Prognosis.—Favourable as to life, but unfavourable as to the subsequent occurrence of chronic valvular defects.

(II) **Malignant (Ulcerative) Endocarditis.**—*Causes.*—Rarely primary; in the great majority secondary to a pre-existing disease, especially to pneumonia; less frequently to septicæmia, erysipelas, puerperal fever, and gonorrhœa; least commonly to acute rheumatic fever, typhoid fever, scarlet fever, smallpox, diphtheria, and tuberculosis. Chronic valvular disease strongly predisposes. It is to be borne in mind that a benign endocarditis may become malignant, and that there are many grades of severity between the extremes.

Symptoms and Clinical Varieties.—The manner of invasion and the symptoms of ulcerative endocarditis are so variable that it is best to consider first the *general symptoms*, which are present in the majority of cases, and, second, the *clinical forms* of the disease.

If, as usual, it is a condition secondary to one of the causative diseases previously enumerated, there may be simply an increase in the height of the fever or a change in its type. Ordinarily, however, there are chills, high and irregular fever followed by sweats, sometimes very profuse, with delirium, and progressive weakness and emaciation. These symptoms are also present in the rare primary form of the disease occurring without a recognisable antecedent cause. The fever is not invariably irregular and remittent, but may be of the continued type. In some cases delirium may be followed by stupor or coma. There may be præcordial oppression and dyspnœa. Jaundice and a diffuse roseolous or papular erythema may be present.

Certain phenomena, of much diagnostic value, are due to the lodgment of septic particles or vegetations which are detached from the inflamed endocardium and swept to various parts or organs of the body. Thus there may be embolism of the spleen with localized peritonitis, pain, and enlargement of the organ; of the kidney, with pain and hæmaturia; of the liver, with pain and perihepatitis; of the cerebrum, with hemiplegia; of the retina, with dimness of vision; of the stomach and intestines, with vomiting and diarrhœa (not necessarily of embolic origin); of the skin and subcutaneous tissues, with ecchymoses or petechial spots; parotid abscess; or abscess or gangrene in any part of the body. In right-side endocarditis there may be pulmonary infarcts with resulting pneumonia, pleurisy, abscess, or gangrene. Pericarditis and acute suppurative meningitis have been noted. Leucocytosis is usually, but not always, present.

The *cardiac physical signs* may be absolutely lacking. Commonly there are one or more systolic murmurs of variable intensity either at apex or base. The signs of an associated pericarditis, pleurisy, or pneumonia may be discovered. If, as frequently happens, the endocarditis has supervened upon chronic valvular disease, the physical signs of the existing valvular defects, with their resultant hypertrophy or dilatation, will be present.

Four clinical varieties or groups are recognised, the *cardiac*, *septic* or *pyæmic*, *typhoid*, and *cerebral*.

(1) Cardiac Type.—This group embraces those cases in which an acute malignant endocarditis occurs in the course of chronic valvular disease. The onset is often abrupt, initiated by a chill, and followed by high fever, which may be regularly or irregularly remittent or intermittent, with or without recurring chills. Many of the symptoms of the pyæmic or typhoid forms—see (2) and (3) following—may be present. The already existing murmurs may remain unchanged in character, or may become more intense and of a more blowing quality. These cases may be acute and fatal, or chronic, lasting from several months to a year or even longer; and in exceptional instances recovery may take place after a varying period. The latter can not properly be classed as malignant, but are examples of a severe although benign endocarditis.

(2) Septic or Pyæmic Type.—The symptoms are those of a pyæmia (page 776), and the attack usually occurs in connection with suppurating wounds, acute necrosis, or puerperal infection. It may be an initial or a secondary event in the pyæmic process, and indeed is an arterial pyæmia. The cardiac symptoms may be overshadowed

by the general symptoms unless the occurrence of suggestive embolic phenomena attracts attention to the heart. This type may ensue in the course of chronic heart disease without a recognisable focus of infection. It is attended with rigours, high fever, and sweats, and may closely resemble quotidian or tertian malarial fever. The duration varies from a few weeks to 3 or 4 months.

(3) Typhoid Type.—This closely simulates typhoid fever. There may be early prostration, irregular fever, delirium, drowsiness, or coma, with diarrhœa, abdominal tenderness and distention, dry brown tongue, parotitis, and a cutaneous rash, sometimes petechial. Cardiac signs and symptoms may be absent, and when present their significance may be quite unappreciated.

(4) Cerebral Type.—A small proportion of cases resemble an acute cerebro-spinal (page 751) or cerebral meningitis with either acute delirium or coma as the principal symptom.

Duration and Prognosis.—Usual duration from 5 to 6 weeks, except the cases supervening upon chronic cardiac disease, which may be protracted for several months. Occasionally death occurs within a few days. If the clinical term "malignant" is taken at its full value the prognosis is invariably fatal, the few cases which recover belonging in strictness to the benign form.

Differential Diagnosis.—If the disease supervenes upon chronic valvular disease, and there are chills, irregular fever, sweats and embolic phenomena, the diagnosis is usually not difficult. The immediately previous occurrence of one of the causative diseases or conditions, e. g., pneumonia or sepsis, is of much value. In the absence of chronic cardiac disease or other suggestive antecedent ailment, or of present cardiac or embolic symptoms, the larger number of cases are to be separated from intermittent malarial fever (see page 789), or, more commonly, typhoid fever (see page 743). In a certain proportion of cases a differential diagnosis is impossible.

II. **Chronic Endocarditis.**—An overgrowth of fibrous tissue, affecting mainly the valves of the heart, which by subsequent contraction causes deformation of the valve segments.

Causes.—It may be, and usually is, a *sequel* of acute endocarditis, of rheumatic origin in the majority of cases; or may be *primary* and begin insidiously. Its course in either case is essentially chronic and progressive. The causes of the first are the same as those of acute endocarditis in general (page 959); of the second, alcoholism, gout, plumbism, syphilis, diabetes, and long-continued heavy muscular exertion. Arteriosclerosis, atheroma, and chronic interstitial nephritis are often associated with the second group, sometimes as cause, sometimes as effect, and have the same etiology.

Symptoms.—Clinically chronic endocarditis manifests itself as chronic valvular disease, and its symptoms are those of various disturbances and ultimate failure of the circulation. About one half of the cases follow rheumatic endocarditis. If the valve segments are thickened, curled, and retracted they fail to close, and *incompetency* with regurgitation results; if the edges of the segments become adherent, *stenosis*, with obstruction to the blood current, ensues. A diseased valve segment may rupture.

III. **Chronic Valvular Disease.**—See also pages 332 to 338.

(I) **Aortic Incompetency.**—*Causes.*—Occurs mainly in men of middle age, and is due most commonly to prolonged and severe muscular exertion, alcohol, gout, syphilis, or lead, all of which may initiate slowly progressive deforming changes in the valve; less frequently to rheumatic endocarditis, rarely to rupture. Occasionally it is relative, the aortic ring enlarging, either in consequence of aortic sclerosis and dilatation or aortic aneurism, to an extent which prevents apposition of the segments, even though normal. Sclerosis of the aorta, often with atheroma and calcification which may involve the coronary arteries, is a frequent associated lesion.

Symptoms.—In this lesion the left ventricle tends to be overdistended, and dilatation precedes hypertrophy. So long as the compensation is efficient there may be no cardiac symptoms; but in a certain proportion of cases, even though compensation is maintained, there may be dull præcordial aching and oppression, or more frequently a sharper pain which radiates into the neck and the arms, the left shoulder and arm in particular. Paroxysms of true angina pectoris may occur. Dyspnœa, præcordial distress, and palpitation often result from slight exertion, and vertigo, faintness, flashes of light before the eyes, tinnitus aurium, and a throbbing headache may be manifest, especially if the patient gets up quickly from a recumbent posture. Occasionally there is redness and a feeling of heat in the skin, followed by copious sweating.

When the heart begins to fail dyspnœa, sometimes orthopnœa, rarely with cyanosis, appears, especially at night. General anasarca is not of frequent occurrence, except when mitral incompetency coexists, but œdema of the lower extremities may be an early symptom, and is favoured by the marked anæmia which is commonly associated with aortic incompetency. Cough, pulmonary congestion or œdema, and perhaps hæmoptysis, may be present. Restless sleep and frequent distressing dreams are very common. Mental symptoms, such as irritability, depression, or peevishness, are often present, and melancholia or other forms of insanity, perhaps with suicidal propensities, will appear in a small number of cases. In the later stages of the disease prostration becomes evident, and there may be recurrent endocarditis with moderate irregular fever and embolic phenomena.

Physical Signs.—The præcordium may bulge, especially in children, and there is a large area of visible impulse. The carotids and other accessible arteries (brachial, femoral, etc.) are seen to pulsate violently, and there is epigastric throbbing. The capillary pulse is readily perceived, and in some instances the veins of the hands and feet may pulsate. The impulse of the heart, unless dilatation predominates over hypertrophy, is strong and heaving, and a diastolic thrill is occasionally felt. The apex beat is palpable in the 6th or 7th interspace outside of the mammillary line. The pulse has the water-hammer character, "Corrigan's pulse." The area of cardiac dulness is greatly increased, extending mainly downward and to the left unless marked dilatation is present.

Upon auscultation a diastolic murmur is heard over the sternum (see Fig. 122, page 376), soft, blowing, rarely harsh, and often almost inaudible. With it is frequently associated an aortic systolic murmur, generally due to roughening of the segments, and not indicative of aortic stenosis. If the incompetency is marked the aortic second sound will be *replaced* by the diastolic murmur. At the apex a systolic mitral murmur is often heard, due to actual or relative mitral insufficiency, less commonly a presystolic mitral murmur (Flint's). Double murmurs may at times be heard in the carotid, subclavian, and femoral arteries, and a short systolic flapping sound (not murmur) is common in the same and even smaller arteries.

Diagnosis.—This is usually easy. A diastolic murmur, even though faint, over the sternum, the throbbing arteries, the peculiar pulse, and the hypertrophy of the left ventricle, constitute a reliable combination of signs. The tremendous pulsation of the innominate and right carotid arteries has led to a mistaken diagnosis of thoracic aneurism (*q. v.*), and indeed there may be a certain degree of dilatation of the first portion of the aorta, with some increase of dulness, in connection with aortic incompetency.

Prognosis.—Ultimately unfavourable, although good compensation may be maintained for years. Sudden death occurs in a larger proportion of cases than with any other valvular defect.

(II) **Aortic Stenosis.**—Narrowing of the aortic orifice without a certain degree of valvular incompetency is of rare occurrence, and indeed is much less frequently met with than the latter.

Causes.—Occurs mainly in old men, and is usually due to slow sclerotic, atheromatous, and calcareous changes, often constituting a part of a general arteriosclerosis and involving the coronary arteries. Infrequently it is a result of rheumatic and other forms of endocarditis; may be congenital; and occasionally the orifice is of normal size, but opens into a dilated aorta—relative stenosis.

Symptoms.—So long as the hypertrophy compensates for the obstruction the disease is latent. The earliest symptoms of beginning muscular failure are vertigo and faintness, due to cerebral anæmia, with præcordial oppression or anginal pain and palpitation after exertion. In the later stages the mitral and subsequently the tricuspid valves may become relatively insufficient because of cardiac dilatation, with the usual indirect evidences of general venous stasis (page 316). Embolic phenomena and Cheyne-Stokes respiration may become manifest.

Physical Signs.—The apex beat is carried to the left and downward, and, unless hypertrophic emphysema coexists, is strong and forcible. A systolic thrill, often of marked intensity, is apt to be felt over the aortic area. The area of heart dulness is increased, provided the oftentimes associated emphysema does not mask it. The aortic second sound is usually weak, and a systolic murmur (described at page 375) is heard over the aortic area, frequently associated with the diastolic murmur of aortic incompetency; and if relative mitral insufficiency has been established there will be a blowing systolic murmur at the apex. The rather characteristic *pulsus tardus* (pages 406, 407) is present.

Diagnosis.—An aortic systolic murmur is in the majority of cases not due to stenosis, but if it is harsh, rough, or musical, and associated with thrill, cardiac hypertrophy, and *pulsus tardus* in an elderly person, a diagnosis of this lesion is permissible.

(III) **Mitral Incompetency.**—May be due to deformation of the valve segments or shortening of the chordæ tendineæ; or to dilatation of the ventricle with relative incompetency. It constitutes at least one half of all cases of valvular disease. As consequences of this defect, first the left auricle, then the left ventricle, finally the right ventricle and auricle become dilated and hypertrophied.

Causes.—The *organic* defects are usually due to rheumatic or other form of endocarditis, or constitute a part of a general arteriosclerosis; while *relative* incompetency results from left ventricular dilatation due to aortic stenosis or incompetency, or succeeds the hypertrophy of chronic nephritis or arteriosclerosis, or the muscular weakness of severe anæmia or protracted febrile diseases.

Symptoms.—While compensation is perfect there are no subjective evidences of cardiac disease. If good, though not perfect, there is moderate dyspnœa on exertion, the face may have a slightly cyanotic tint, and the venous radicles of the cheeks are plainly visible. Clubbing of the fingers is common in cases of long duration, especially in children. There may be cardiac palpitation. There is a marked tendency to attacks of bronchitis and the slighter grades of gastro-intestinal disturbances. Undue muscular exertion may produce severe, but at this stage usually temporary, pulmonary congestion, œdema, or hæmoptysis. When compensation is broken and dilatation exceeds hypertrophy, the pulse becomes extremely irregular, there is palpitation, dyspnœa, or orthopnœa; cough, sometimes with frothy, bloody sputum; cyanosis and slight jaundice. There are evidences of gastro-intestinal catarrh (nausea, diarrhœa), hemorrhoids, and enlargement of the liver and spleen. There is œdema, beginning in the feet and extending upward, often with ascites and hydrothorax. The urine is scanty, high coloured, and contains albumin and often casts. All of these symptoms are evidences of general venous congestion (Fig. 100, page 337). Starting during sleep is a common and most grievous occurrence.

Physical Signs.—In children the præcordium may be prominent. The apex beat lies to the left and below, according to the degree of hypertrophy, and the area of visible and palpable pulsation is greatly increased. If compensation is still good the impulse is the heaving and forcible stroke of hypertrophy; if broken, the weak, wavy, diffuse pulsation of a dilated heart. Occasionally there is a systolic thrill at the apex. There may be epigastric pulsation due to right ventricular hypertrophy and dilatation; as well as pulsating jugulars and pulsating liver, significant of tricuspid insufficiency. The pulse is small and compressible, and when the heart fails is absolutely arrhythmic. There may be contractions of the heart which are not represented at the wrist (ineffectual systole). The area of cardiac dulness is increased, both to left and right, because both left and right ventricles are dilated and hypertrophied. A blowing or musical systolic murmur, either accompanying or replacing the first sound, is heard at the apex, transmitted to the left, and heard posteriorly at the angle of the scapula (Fig. 120, page 374). Although its maximum intensity is usually at the apex, it may be loudest or heard only at the base of the heart or along the left sternal edge. Occasionally it is perceptible when the patient is lying down, and disappears if he stands up. The second sound is generally very distinctly audible at the apex; and the second pulmonary sound is accentuated over the pulmonary area. If there is associated tricuspid incompetence there is a systolic murmur in its area.

Diagnosis.—The characteristic physical signs are a systolic murmur transmitted to the left and heard posteriorly; accentuation of the pulmonary second sound, and evidence of both right- and left-heart hypertrophy.

The foregoing combination of physical signs is essential for a diagnosis, as a systolic mitral murmur occurs in various conditions unconnected with organic mitral valvular defects or relative incompetency (page 373). Whether the insufficiency is due to deformation of the valve, or is relative, can not always be determined. If it occurs in the subjects of chronic nephritis, general arteriosclerosis, or aortic lesions, all of which cause hypertrophy and dilatation of the left ventricle, the mitral incompetence is probably relative.

Prognosis.—Life may be prolonged for many years if compensation is maintained. There are apt to be repeated breaks of compensation, each becoming more severe, ending with general anasarca, cardiac dilatation, and death, rarely sudden, usually from progressive weakness of the heart muscle. Not infrequently permanent arrhythmia follows the first rupture of compensation.

(IV) **Mitral Stenosis.**—*Causes.*—Narrowing of the mitral orifice is usually due to endocarditis, generally rheumatic; occasionally, perhaps, to the strain of whooping cough; is more frequent in females and in young rather than elderly persons. The most frequent form of constriction is the buttonhole opening, less commonly it is funnel-shaped. As a result of the narrowing the left ventricle remains of normal size, or becomes smaller, unless mitral incompetency coexists; while the left auricle and subsequently the right ventricle and auricle become hypertrophied and ultimately dilated, with sequent tricuspid insufficiency and systemic venous congestion.

Symptoms.—Except for a varying degree of dyspnœa and palpitation following unusual muscular exertion, there may be an entire absence of symptoms so long as compensation is good. There is a liability to attacks of recurrent endocarditis, and the resulting vegetations may become detached and swept away, thus causing embolic phenomena, especially asphasia or aphasic hemiplegia. Paralysis of the left vocal cord, due to the pressure of the hypertrophied left auricle and simulating one of the effects of aortic aneurism, has been noted. Anæmia and left intercostal neuralgia are very common. When compensation is failing the resulting symptoms are practically those of mitral incompetency. There are more or less constant dyspnœa, frequent and arrhythmic pulse, cough, bronchitis, and congestion or œdema of the lungs, with cyanosis, blood-stained expectoration, or occasionally hæmoptysis. These symptoms are especially apt to occur after severe muscular exertion, and there may be frequent recurrences. Œdema of the lower extremities and general anasarca are not common unless tricuspid insufficiency coexists. Late in the disease ascites may occur, especially in children, and is associated with great swelling of the liver. An enlarged, perhaps pulsating, liver, due to passive congestion, is very frequent in mitral stenosis when the right heart fails. Elevations of temperature, symptomatic of recurrent endocarditis, are not uncommon.

Physical Signs.—Owing to hypertrophy of the right ventricle the lower sternum and 5th and 6th left cartilages may be prominent, particularly in children, and the main impulse of the heart is usually visible over the same area. *Pulsation* may often be seen in the 2d, 3d, and 4th interspaces to the left, rarely also to the right, of the sternum, if the chest walls are not too thick; and pulsation of the epigastrium, due to the hypertrophied right ventricle, is common. The *apex beat*, unless mitral incompetency or other cause of left ventricular hypertrophy coexists, may remain in its normal position or be displaced somewhat to the left, rarely to the outside of the mammillary line, depending upon the degree of right ventricular enlargement. The strongest impulse is usually felt over the lower sternum and 5th and 6th left interspaces, perhaps also in the 4th, 3d, and 2d spaces. The impulse in the left 2d space is ascribed either to the hypertrophied left auricle or to the increased tension in the conus arteriosus and pulmonary artery, both views having supporters. In the bulk of the cases there is the characteristic rough, purring *thrill*, best felt within the nipple line in the 3d or 4th, sometimes the 5th, interspaces. It is diastolic, beginning just after the second sound, ends abruptly with the apex impulse, and is most marked during expiration. It is pathognomonic of mitral stenosis, and when found permits a diagnosis by palpation alone. The area of cardiac *dulness* is increased to the right, as well as upward along the left sternal margin to the 2d rib; but not to the left and downward unless left ventricular hypertrophy coexists as a result of mitral regurgitation. Internal to and a little above the apex beat is a presystolic *murmur*, variously described as rough, rolling, blubbering, churning, vibratory, purring, or hesitating, whose audibility is usually limited to a two-inch circle, but at times may be very widely heard (Fig. 118, page 372). It is variable, appearing and disappearing in accordance with the strength of the auricular systole; may occupy the whole or the middle or the latter part of the diastolic period (Fig. 119, page 373); and may be heard only after exertion. The first sound, in which this murmur abruptly terminates, is short, sharp, and snapping; the pulmonary second sound is loudly accentuated and heard over a wide area, and the aortic second sound is relatively and actually weak. There may be reduplication of the second sound. The murmurs of mitral and triscuspid incompetency, the latter often a secondary result, may be heard. The *pulse*, with marked stenosis, is notably small, and when the heart fails becomes irregular like that of incompetency.

When compensation is broken the thrill and the murmur may disappear, the snap of the first sound remaining. Not infrequently the sounds are reduplicated, the impulse is diffuse and weak, and there may be systolic jugular pulsation and pulsating liver, from tricuspid insufficiency. If the strength of the left auricle and right ventricle is restored, the murmur reappears.

Diagnosis.—The signs which justify a diagnosis of mitral stenosis are: a presystolic thrill and murmur with the characters described; evidence of right-heart hypertrophy, the left heart remaining of normal size; and an accentuated pulmonary second sound. If aortic regurgitation, or aortic stenosis and adherent pericardium are found to exist, the presence of a presystolic mitral murmur is not positive evidence of mitral stenosis (see (2), page 373).

Prognosis.—Ultimately unfavourable, although with proper care many years may elapse before death occurs by gradual failure of compensation, perhaps after repeated temporary breaks.

(V) **Tricuspid Incompetency.**—*Causes.*—Rarely this is primary, the valve segments becoming deformed as a result of right-heart endocarditis during fœtal life or in early childhood; as a rule it is relative and secondary to left heart, especially mitral, valvular disease, or to interstitial pneumonia and emphysema.

Symptoms.—The symptoms are largely dependent upon the causative valvular or pulmonary disease, and are for the most part the evidences of stasis in the lungs and systemic veins.

Physical Signs.—The characteristic and indubitable signs of tricuspid regurgitation are: systolic pulsation in the jugulars, especially the right; swollen and pulsating liver; and the presence of a soft, low systolic murmur over the lower sternum ((*b*), page 377). The pulmonary second sound is accentuated, the cardiac dulness is increased to the right, and there is epigastric pulsation.

(VI) **Tricuspid Stenosis.**—This may be either congenital, and combined with other defects of development, or acquired, occurring as a secondary result to left-heart lesions. It is most commonly associated with mitral stenosis (both largely due to rheumatic endocarditis), less frequently with aortic incompetency, and is very rare as a primary and isolated condition. It occurs in females rather than males (5 to 1). The *symptoms* are those of the associated valvular defects. Facial cyanosis and extreme anasarca are terminal evidences.

Physical Signs.—There may be a presystolic thrill over the tricuspid area; the area of cardiac dulness is enlarged to the right; and a presystolic murmur may be heard ((*a*), page 377). A positive diagnosis is rarely practicable, unless it is an isolated lesion, because of the difficulty of separating the usually co-existent mitral presystolic from a tricuspid presystolic murmur.

(VII) **Pulmonary Incompetence.**—(See (*b*), page 379). A differential diagnosis from aortic incompetency is rarely possible, although the absence of Corrigan's pulse and the non-discovery of left ventricular dilatation and hypertrophy will tend to exclude the latter.

(VIII) **Pulmonary Stenosis.**—This is usually congenital. There is apt to be cyanosis and systemic venous engorgement.

The *physical signs*, when present, are the evidences of right ventricular hypertrophy and the presence of a systolic thrill and murmur in the pulmonary area. The murmur is of a rough or harsh quality, usually strictly localized and apparently superficial, and, of course, is not transmitted into the arteries of the neck, a differential point between it and the similar murmur of aortic stenosis. The pulmonary second sound is weak and may be accompanied or replaced by the diastolic murmur of associated incompetency.

A *diagnosis* of pulmonary stenosis must be made with great reserve because of the very frequent occurrence of pulmonary systolic murmurs, usually of anæmic origin (see (*a*), page 378).

(IX) **Combined Valvular Defects.**—The statistics regarding the relative frequency of certain combinations of valvular defects are variable. In general aortic and mitral lesions most commonly coexist, then mitral and tricuspid, finally aortic, mitral, and tricuspid. The particular combinations in the usual order of frequency are:

1. Aortic incompetency and stenosis and mitral incompetency.
2. Mitral stenosis and incompetency.
3. Aortic stenosis and mitral stenosis.
4. Mitral stenosis and aortic incompetency.

With reference to combined murmurs, see page 379.

IV. **Hypertrophy of the Heart.**—Increased thickness of the muscular walls of the heart—hypertrophy—may exist without dilatation of the chambers (*simple hypertrophy*); more commonly the hypertrophy is associated with dilatation (*eccentric hypertrophy*). So-called concentric hypertrophy—thickened walls with lessened size of the cavities—is a post-mortem contraction event.

Causes.—The hypertrophy may affect *one cavity*, *one side*, or the *whole* of the heart.

(1) *Left Ventricular Hypertrophy.*—May be due to aortic stenosis, aortic and mitral incompetency, which increase the intraventricular pressure; pericardial adhesions and fibrous myocarditis, which interfere with the contraction of the heart muscle and increase its work; general arteriosclerosis and the presence of irritating substances in the blood in gout, chronic nephritis, lead-poisoning, and syphilis, all

of which, either by producing organic narrowing or by causing prolonged contraction of the arterioles, heighten the arterial pressure and create an obstruction to the work of the left ventricle; congenital narrowing of the aorta, aneurism of the same vessel, or stenosis caused by external pressure upon it; persistent overaction of the muscle as in the tachycardia of exophthalmic goitre, the tea, coffee, and alcohol habits, or long-continued neurotic palpitation; and habitual excessive eating and drinking, especially the drinking of enormous quantities of beer. Prolonged and severe muscular work is an additional and sometimes important factor.

Many of these causes operate to produce varying degrees of simultaneous right ventricular hypertrophy.

(2) *Right Ventricular Hypertrophy.*—May be due to mitral stenosis and mitral incompetency; and emphysema, chronic interstitial pneumonia, or extensive pleural adhesions, all of which raise the pressure and increase the resistance in the pulmonary circuit. Valvular lesions on the right side, especially pulmonary stenosis, will also cause hypertrophy of the right ventricle.

(3) *Auricular Hypertrophy.*—This is always conjoined with dilatation, and when affecting the *left auricle* occurs in mitral incompetency or mitral stenosis, especially the latter; when affecting the *right auricle*, it is found in all conditions which raise the pulmonary blood pressure (see (2) preceding); and in tricuspid or pulmonary incompetence or stenosis.

Symptoms.—As a rule the condition—almost invariably conservative—is subjectively latent until the hypertrophied muscle can no longer respond to the demands upon it and ruptured compensation becomes manifest. The earlier symptoms of well-marked hypertrophy, especially of the left ventricle, consist of an indefinite sense of præcordial discomfort or fulness, most marked when lying upon the left side. The sensation is seldom that of pain, and palpitation or a consciousness of the overaction of the heart is usually not perceived except when the patient is neurasthenic or addicted to the overuse of tobacco. There may be a sense of fulness or throbbing in the head, headache, flushing of the face, carotid throbbing, vertigo, tinnitus aurium, flashes of light, exophthalmos, and epistaxis. General arteriosclerosis is a frequent concomitant event, either as cause or result of the hypertrophy, and broncho-pulmonary or cerebral hemorrhage, due to rupture of the sclerotic smaller vessels by the increased force of the heart, may occur.

(1) *Physical Signs of Left Ventricular Hypertrophy.*—The præcordium may be prominent, especially in children, and an extensive impulse is visible. On palpation the impulse is characteristically

slow, heaving, and forcible (unless a notable degree of dilatation coexists, when it is rather more sudden), and the apex beat is displaced downward, perhaps to the 7th or 8th interspace, and to the left even as much as 3 inches outside of the mammillary line. But in the more common degrees of hypertrophy the apex lies in the 6th space, in or a little outside of the mammillary line. The percussion dulness is increased downward, to the left, and vertically (Fig. 109, p. 356). In simple hypertrophy without valvular lesions there are no murmurs, but the first sound is loud, prolonged, and booming, often with a clicking or murmurish quality; if dilatation coexists it is shorter and sharper. Aortic closure is accentuated and clear or ringing, and the second sound often reduplicated, especially in the hypertrophy of chronic nephritis; if the ventricle is also dilated, or the heart action weak, the second sound is less clear and intense. If the hypertrophy is due to valvular defects there will be the physical signs of the special lesions present. If the hypertrophy is unaccompanied by dilatation, the pulse is large, strong, regular, of increased tension, and often not more frequent than normal; if with dilatation, it is softer and as a rule of greater frequency.

(2) *Symptoms and Physical Signs of Right Ventricular Hypertrophy.*—There are no *symptoms* while compensation is maintained, except moderate dyspnœa following unusual muscular exertion; or præcordial discomfort, cough, and dyspnœa, when the hypertrophy is a sequence of emphysema or chronic interstitial pneumonia. In course of time, when dilatation and relative tricuspid incompetency occur, there will be persistent dyspnœa, bronchitis, pulmonary congestion or œdema, cyanosis, hæmoptysis, and evidence of systemic venous stasis. The *physical signs* are (perhaps) an unusual prominence of the lower sternum and the 6th and 7th left cartilages, with a visible somewhat diffuse impulse over the same area, often also in the epigastrium. Unless the chest walls are thick, pulsation is frequently present in the 3d and 4th interspaces to the right of the sternum, particularly if there is much dilatation. The apex beat is carried to the left, usually with but slight downward displacement, and is diffuse, lacking the well-defined thrust of left ventricular hypertrophy. The cardiac dulness is increased mainly to the right, perhaps an inch or more beyond the sternal margin. The first sound over the tricuspid area is louder than usual, and on account of the increased tension in the pulmonary artery the pulmonary second sound is accentuated, and reduplication of the second sound may occur. The radial pulse is of small volume, and if dilatation is present may be frequent and arrhythmic.

(3) *Signs of Auricular Hypertrophy.*—The physical signs of hy-

pertrophy, always combined with dilatation, of the *left auricle*, are few and indefinite. There may be dulness to the left of the sternum in the 3d or 2d interspaces, with a presystolic impulse or wave in the 2d space. The presence of left auricular enlargement may always be inferred if the presystolic murmur of mitral stenosis is heard, or if mitral incompetency exists.

Hypertrophy, never without dilatation, of the *right auricle*, is secondary to incompetency or stenosis of the tricuspid valve with associated right ventricular hypertrophy and dilatation. There is dulness in the 3d and 4th interspaces to the right of the sternum, often with a presystolic wavy pulsation in the same area, systolic jugular pulsation, and evidences of general venous engorgement.

Differential Diagnosis.—Chronic interstitial pneumonia, phthisis with fibrosis and retraction, and chronic dry pleurisy (all on the left side) may, by uncovering the heart, give rise to an extensive area of pulsation, which is at times mistaken for hypertrophy. The less forcible and less heaving impact, together with the evidences of pulmonary disease, will enable a differentiation; so also with the unusually marked impulse often found in deformities of the chest.

The increased area of dulness caused by aneurism, pericardial effusion, and mediastinal growths, may simulate that of hypertrophy, but a careful consideration of the physical signs will usually suffice to exclude these conditions. Displacement of the apex beat by extracardial lesions can generally be discriminated by the absence of a forcible, heaving impulse or of an increased, although shifted, area of dulness. Hypertrophy may be quite overlooked or impossible of recognition in hypertrophic emphysema.

V. **Dilatation of the Heart.**—The walls of a dilated heart are either thicker or thinner than normal, while the size of the cavities is increased out of proportion to the thickness of their walls. If the heart walls are thick and their muscular power ample to sustain the circulation, a considerable increase in the size of the chambers may exist, which is properly termed eccentric hypertrophy (page 970) rather than dilatation.

Causes.—Dilatation depends upon two general causes: (1) increased endocardial pressure, and (2) weakness of the heart walls.

The causes of increased pressure are practically the same as those of hypertrophy (page 970). Whether dilatation or hypertrophy will result from the greater tension depends in part upon the suddenness and severity of the strain. Dilatation is more apt to result from an abrupt and intense increase of pressure; hypertrophy from slighter but persistent strain. If the heart walls are weakened from myocardial inflammation or degeneration, or other cause, they will yield to a normal, much more readily to an abnormally high, degree of endocardial pressure. Thus in valvular lesions with a constant but moderate increase of pressure, progressive hypertrophy takes place until the musculature depreciates, when dilatation will occur. On the other hand, severe and unaccustomed muscular exertion will cause acute dilatation, especially of the right ventricle, followed for weeks, months, or years by dyspnœa and other symptoms of heart strain upon exercise. So also there may be acute dilatation in the myocarditis and parenchymatous or other degenerations or nutritive disturbances of the heart muscle associated with scarlet fever, erysipelas, typhus and typhoid fevers, rheumatic fever, acute endocarditis or pericarditis, the anæmias, and leucæmia. Slow dilatation occurs in the more chronic degenerative and sclerotic processes, often induced by diet, mode of life, and especially the excessive use of alcohol combined with persistent overexertion (irritable heart). Some cases, either acute or chronic, occur without recognisable cause.

Symptoms.—When dilatation takes place slowly the symptoms are those previously described as characteristic of gradual failure of compensation in valvular lesions (page 335). Acute dilatation occurring in fevers or in chronic hypertrophy is indicated by dyspnœa, palpitation, sometimes præcordial oppression or pain, a weak and frequent pulse, and the evidences of systemic venous stasis.

Physical Signs.—A distinct apex beat is often absent, or if present is weak. Usually the impulse is widely diffused, wavy, and undulating; and though plainly visible can not, in many cases, be felt by the palpating hand, a sign of much value. Unless emphysema is present the area of cardiac dulness is increased vertically and transversely. The first sound is short and flapping, often resembling the second sound, which is also weakened. Embryocardia and the gallop rhythm are common. The presence of murmurs due to valvular disease may mask the characters of the sounds; but a murmur, especially that of mitral stenosis, may disappear as the heart weakens. A well-marked apical systolic murmur, due to relative mitral incompetency, may, however, make its appearance, subsequently vanishing if the dilatation is overcome. The pulse is of small volume, weak, frequent, and often extremely arrhythmic.

The signs just described relate mainly to *left ventricular dilatation*. Those which may enable a diagnosis of predominating *right ventricular dilatation* are the location of the chief impulse below or to the right of the ensiform appendix, with little or no impulse in the usual place of the apex beat, an undulating pulsation close to the left sternal margin in the 4th, 5th, and 6th interspaces, and an excessive increase of the cardiac dulness to the right of the sternum.

A pulsation in the 3d interspace to the right of the sternum, systolic if there is tricuspid regurgitation, is indicative of *right auricular dilatation.* Pulsation, either systolic or pre-systolic, in the 2d space to the left of the sternum, may be manifest, and if pre-systolic, has been affirmed and denied as an evidence of *left auricular dilatation.*

Differential Diagnosis.—Dilatation requires to be distinguished from hypertrophy and from large pericardial effusions.

(1) *Cardiac Hypertrophy.*—A slow, strong, heaving impulse, a distinct although large and rounded apex beat, lying downward and to the left, and the presence of a dull, prolonged, and loud first sound, with an accentuated second sound, are usually sufficient to announce hypertrophy rather than dilatation.

(2) *Pericardial Effusion.*—See page 956.

VI. **Fatty Heart.**—Two varieties are recognised: *fatty degeneration* of the muscular fibres, and *fatty infiltration* or overgrowth, an increase of the normal subpericardial fat.

(I) **Fatty Degeneration.**—Occurs in connection with carcinoma, phthisis, prolonged infectious fevers, severe acute or chronic anæmias, phosphorus poisoning, disease of the coronary arteries, pericarditis, old age, and cardiac hypertrophies in general. In all these instances there is defective nutrition of the heart muscle.

The *symptoms and signs*, so long as dilatation does not occur, are negative; and when present are practically those of dilatation, either acute after severe muscular exertion or chronic and slow in their onset. There may be syncopal or anginal attacks, or seizures of cardiac asthma, especially in the early morning hours; and periods of bradycardia, pseudo-apoplectic attacks, Cheyne-Stokes breathing, and delusional or maniacal mental states. Dyspnœa, palpitation, and a small and irregular pulse are common, the heart sounds may be weak with a galloping rhythm, and the apical systolic murmur of dilatation may develop. None of these signs and symptoms are distinctive, as they occur also in chronic myocarditis, and a *diagnosis* of fatty degeneration is rarely more than probable.

(II) **Fatty Infiltration.**—This is almost always a part of general obesity, affecting men rather than women, and occurring usually between 40 and 70 years of age. No symptoms are present until dilatation occurs, after which there may be bronchitis, vertigo, and pseudo-apoplectic and syncopal attacks, with feeble pulse and heart sounds.

The *diagnosis* depends upon the presence of obesity, *plus* the evidences of cardiac weakness.

VII. **Myocarditis.**—Two varieties: *acute* and *chronic.*

(I) **Acute Myocarditis.**—(1) Acute *circumscribed* myocarditis or abscess of the heart occurs in pyæmia, diphtheria, typhoid fever, malignant endocarditis, and other septic conditions. The condition can not be diagnosed, beyond a suspicion, during life.

(2) Acute *diffuse* myocarditis, of which parenchymatous degeneration or cloudy swelling forms a goodly part, is met with mainly as a result of the infectious fevers such as smallpox, typhus and typhoid fevers, diphtheria, scarlet fever, and acute rheumatic fever; or in connection with acute endocarditis or pericarditis. The *symptoms* are simply those of marked cardiac weakness and do not point to the cause of the feebleness. There is usually a small, feeble, frequent, and often irregular pulse, perhaps with palpitation of the heart and a tendency to syncope. The *physical signs* are practically those of cardiac dilatation, which in varying degrees is such a common and immediate sequence of the myocardial inflammation.

(II) **Chronic (or Fibrous) Myocarditis.**—*Causes.*—The fibroid heart may succeed acute myocarditis, but in the majority of instances it is secondary to lesions of the coronary arteries, especially obliterating arteritis, less commonly thrombosis or embolism (white or anæmic infarcts); or to interference with the coronary circulation as in valvular disease of the heart; or is associated with hypertrophy; and also occurs, affecting the superficial layers of the muscle, as a result of chronic endocarditis or pericarditis. The remoter causes are gout, alcohol, lead-poisoning, syphilis, rheumatism, chronic nephritis, and diabetes, i. e., the usual agencies which cause arteriosclerosis.

Symptoms.—The condition may be quite latent. Angina pectoris and a weak, irregular, often slow pulse (50 to 30), are somewhat characteristic; and when the sclerosed heart is slowly failing and dilating, dyspnœa, cardiac asthma, palpitation, præcordial constriction, and evidences of general venous stasis appear. There may be recurring, sometimes fatal, pseudo-apoplectic seizures, which may be preceded by occasional vertigo and syncopal attacks; or true apoplexy may terminate life or cause a hemiplegia. Chronic mania or other form of psychosis may develop.

Physical Signs.—The signs are practically those of a dilated heart, often with a systolic murmur at the apex (relative mitral incompetency) and a galloping rhythm.

Diagnosis.—As Osler well says: "For practical purposes we may group the cases of myocardial disease as follows:

"(1) Those in which sudden death occurs with or without previous indications of heart trouble. Sclerosis of the coronary arteries exists, in some instances with recent thrombus and white infarcts, in others extensive fibroid disease, in others again, fatty degeneration. Many patients never complain of cardiac distress, but enjoy unusual vigour of mind and body.

"(2) Cases in which there are cardiac arrhythmia, shortness of breath on exertion, attacks of cardiac asthma, sometimes anginal attacks, collapse symptoms with sweats and extremely slow pulse, and occasionally marked mental symptoms. These are the cases in which the condition may be strongly suspected, and, in some instances, diagnosed. It is rarely possible to make a distinction between the fatty and fibroid heart.

"(3) Cases in which there are cardiac insufficiency and symptoms of dilatation of the heart. Dropsy is often present, and with a loud murmur at the apex it may be difficult, unless the case has been seen from the outset, to determine whether or not a valvular lesion is present."

VIII. **Aneurism of the Heart.**—The aneurism may involve either the valves of the heart or its walls. The former results from acute endocarditis, which softens or erodes one or more cusps, causing thinning and bulging or actual perforation of the segment, in the latter case with consequent incomptency. Aneurism of the wall usually affects the apical portion of the left ventricle, and is caused most commonly by chronic myocarditis, less frequently by acute endocarditis, pericarditis, or gumma. In well-marked cases the thinned portion of the wall projects sufficiently to constitute a rounded tumour which may equal the heart itself in size. The heart may rupture. The *physical signs* are not in the least distinctive. There may be a visible pulsating swelling in the region of the apex, occasionally with pressure perforation of the chest wall; but as the condition is rare and the physical signs are practically those of cardiac hypertrophy or dilatation, a positive or even a probable diagnosis during life is exceedingly infrequent.

IX. **Rupture of the Heart.**—Rare, and usually takes place in the anterior wall of the left ventricle, most commonly as a result of fatty or fibroid degeneration, less frequently gumma, abscess, or acute coronary embolic softening. The usual immediate cause is overexertion. In most cases this accident is immediately fatal. If life is prolonged, as it may be for several hours or even days, there will be intense præcordial pain and oppression, with the symptoms of internal hemorrhage (page 166). The physical signs of pericardial effusion rapidly develop.

X. **Cardiac Neuroses.** — These are *palpitation*, *tachycardia* (page 392), *bradycardia* (page 394), *arrhythmia* (page 395), and *angina pectoris.*

(I) **Palpitation.**—This is an overstrong and usually too frequent action of the heart, either regular or irregular, *of which the patient is uncomfortably conscious.* The presence of the subjective sensation, not simply an overrapid heart action, constitutes the cardinal characteristic of this neurosis. There is often an underlying abnormal excitability of the nervous system.

Causes.—Occurs more commonly in women than in men, especially at puberty, the menstrual period, or the menopause. Frequent causes are anæmia, hysteria, neurasthenia, dyspepsia, worry, anxiety, or strong emotions; overuse of tea, coffee, tobacco, and alcohol; less frequently the acute fevers; conjoined illness, excitement and unaccustomed physical exertion (irritable heart of soldiers); and occasionally organic valvular or myocardial disease.

Symptoms.—The condition is usually paroxysmal, not often continuous or, if so, is attended with exacerbations. In the milder cases there is simply a consciousness of sinking or fluttering of the heart; in the more marked instances the heart throbs or beats violently, usually with increased rapidity (110 to 160), often irregularly, and there is mental anxiety with sensations of dyspnœa and oppression, or even nausea and præcordial pain. The peripheral arteries may pulsate strongly. The face and skin may be flushed. A copious amount of clear pale urine may be voided after the attack. A paroxysm usually terminates within an hour, but may continue for hours or days. In the majority of cases *examination of the heart* affords negative results. There may be a more widely diffused and forcible impulse than normal, with clear, sharp, or accentuated heart sounds. Murmurs due to anæmia or the rapid action of the heart may be heard, especially in the pulmonary area, seldom at the apex. Ordinarily murmurs are absent.

Diagnosis.—The presence of a subjective consciousness of the heart beat is essential to the diagnosis. Rapid heart action, not perceptible to the patient, is tachycardia, not palpitation. Examination of the heart during the intervals of the attacks will separate the cases of purely nervous palpitation from those which are symptomatic of anæmia or chronic valvular lesions. The *prognosis* is good as to life, but if the attacks are frequent and prolonged for years hypertrophy of the heart may ensue.

(II) **Angina Pectoris.**—A symptom, not a disease.

Causes.—With very rare exceptions stenocardia or breast pang is associated with arteriosclerosis, which may be general or local, but in either case affects the aorta, at its origin, and the coronary arteries. The latter may be narrowed at their roots, or their main divisions be the seat of an obliterating endocarditis. Myocardial changes usually coexist. It occurs most frequently in aortic insufficiency and adherent pericardium, much less commonly with mitral lesions. The *exciting causes* of an attack are muscular exertion, strong mental emotion, gastric distention or disturbance, and exposure to cold. The attacks, in the great majority of cases, affect men, usually over 40 years of age.

Symptoms.—The attack begins suddenly, with pain, usually intense and excruciating, in the region of the heart. The pain radiates into the neck, the left shoulder, and down the arm to the fingers, sometimes to the right arm and down the body. There is also a sense of cardiac constriction, often with coldness and numbness of the præcordium and the fingers. The face is pale or ashy gray and betrays a feeling of intense anxiety. The face and body are often covered with large drops of cold perspiration. A sense of impending death is a usual and characteristic symptom. There may or may not be dyspnœa, sometimes associated with wheezing or asthmatic breathing. The arterial tension is usually increased, and while the action of the heart may be arrhythmic, it is often regular and normal. The patient may be restless, but more commonly holds himself quiet and passive in fearful expectation of what may happen.

The attack lasts from a few seconds to two minutes, and often terminates with eructations, nausea and vomiting, or the voiding of a large quantity of clear pale urine.

Variations and Course.—There is much variation in the severity of the ailment in different persons and in the same person at different times. The milder cases present præcordial oppression and discomfort, with slight cardiac pain radiating to the neck and arm; and there are all grades between this and the severe seizures previously described. Death may occur in the first attack; or there may be frequent paroxysms for a number of successive days; or the seizures may be few and spread over many years.

Differential Diagnosis.—An attack of severe cardiac pain having the characteristics previously noted is probably a true angina, but if in addition there are evidences of arteriosclerosis or aortic valvular lesions, and the seizure occurs in men over 40 years of age, it is without a doubt true angina pectoris. A positive diagnosis can not be made in the slighter painful manifestations unless arterial and valvular lesions are recognisable. Confusion may arise in connection with the following conditions:

(1) Locomotor Ataxia.—The girdle sensation and sharp neuralgic pains of locomotor ataxia if seated in the thorax, and especially if associated with arteriosclerosis, may bear a rather close resemblance to the milder forms of true angina, but the presence of the Argyll-Robertson pupil and disorders of co-ordination, with the absent patellar reflexes, will declare for the former. Moreover, the attacks in this case are often independent of exertion or other exciting cause.

(2) Gastralgia.—See page 858.

(3) Pseudo-angina.—Three varieties of *false angina*, not associated with organic changes, have been described. *Neurotic Form.*—Seen in hysterical or neurasthenic women or in neurasthenic men, often with coexisting dyspepsia. The attacks usually occur at night with paroxysmal substernal pain, palpitation, and a sense of cardiac fulness or distention. This condition may be also a sequel of influenza. *Vasomotor Angina* (Nothnagel).—Consists of a primary coldness, numbness, or stiffness of the extremities, and pallor of the face (vasomotor spasm) followed by syncopal sensations and severe præcordial pain. *Toxic Angina.*—Due to the overuse of tea, coffee, and tobacco. In addition to the ordinary palpitation and irregularity of heart action which may be caused by these forms of poisoning, there may be cardiac pain of such severity that it is properly termed a pseudo-angina. It is sometimes associated with coldness of the extremities, faintness, and weak pulse.

It is not always easy to differentiate between true and false angina, especially in women, and some serious mistakes have been made. The importance of the history, and in particular of a searching and careful physical examination, must be strongly emphasized. The following table (Huchard, quoted by Osler) is of much service:

TRUE ANGINA	PSEUDO-ANGINA
Most common between the ages of 40 and 50 years.	At every age, even 6 years.
Most common in men. Attacks brought on by exertion.	Most common in women. Attacks spontaneous.
Attacks rarely periodical or nocturnal.	Often periodical and nocturnal.
Not associated with other symptoms.	Associated with nervous symptoms.
Vasomotor form rare. Agonizing pain and sensation of compression by a vice.	Vasomotor form common. Pain less severe; sensation of distention.
Pain of short duration. Attitude: silence, immobility.	Pain lasts 1 or 2 hours. Agitation and activity.
Lesions: sclerosis of coronary artery.	Neuralgia of nerves and cardioplexus.
Prognosis grave, often fatal.	Never fatal.
Arterial medication.	Antineuralgic medication.

Prognosis.—In true angina always grave; in pseudo-angina always favourable. The accuracy of the prognosis in a given case depends upon the correctness of the diagnosis. It must not be forgotten that sudden death occurs in many cases of angina at other times than during a paroxysm.

XI. **Congenital Anomalies of the Heart.**—(*a*) *Causes and Varieties.*—These anomalies result from an arrest of development, or œtal endocarditis, or both. With reference to the frequency of the ifferent lesions and their association, Holt's tables are as follows :

he Frequency of the Different Lesions in 242 Autopsies upon Cases of Congenital Cardiac Anomaly

efect in the ventricular septum	149	cases;	only lesion in	5	cases.
efect in the auricular septum or patent foramen ovale	126	"	"	" 9	"
ulmonic stenosis or atresia	108	"	"	" 6	"
atent ductus arteriosus	68	"	"	" 3	"
bnormalities in the origin of the great vessels	45	"	"	" 0	"
ulmonic insufficiency	17	"	"	" 0	"
ricuspid insufficiency	6	"	"	" 0	"
ricuspid stenosis or atresia	3	"	"	" 0	"
itral insufficiency	1	"	"	" 0	"
itral stenosis or atresia	6	"	"	" 0	"
ortic insufficiency	1	"	"	" 0	"
ortic stenosis or atresia	6	"	"	" 0	"
ransposition of the heart	2	"	"	" 0	"
ctocardia	1	"	"	" 1	"

The most Frequent Associated Lesions

ulmonic stenosis, with defect in the ventricular septum	92	cases;	only lesion in	20	cases.
ulmonic stenosis, with defect in the auricular septum	52	"	"	" 8	"
efects in both septa	82	"	"	" 17	"
ulmonic stenosis and defects in both septa	36	"	"	" 21	"

(*b*) *Symptoms.*—The most distinctive symptom is cyanosis, which as been noted in about 90 per-cent of all cases. It may be slight nd manifest only after exertion; or limited to the small extremities; r the entire surface may be continuously leaden and livid, the *morbus eruleus* or "blue disease." In the great majority of cases this ymptom makes its appearance within the first week or, at least, the rst month of life; in rare cases this and other symptoms may not ow themselves for periods varying from 1 to 16 years after birth. cool skin, cough, dyspnœa on exertion, and extreme clubbing of e fingers and toes are frequent symptoms; less commonly œdema, ropsy of cavities, and bleeding from the nose or lungs.

(*c*) *Diagnosis.*—The cardinal physical signs of congenital cardiac isease in children are cyanosis, murmurs, and right ventricular ypertrophy—a combination which permits a diagnosis of a congenital sion. The murmur in two thirds of the cases is systolic, usually ud and rough, and most intense over the pulmonary area. The ght ventricle is hypertrophied because, under fœtal conditions, the lk of the work falls upon it, and practically all malformations volve a continuance of the prenatal task.

To determine the exact lesion or defect which is present is always extremely difficult, and in the majority of cases the diagnosis of its precise nature must be conjectural. Two sets of conclusions are appended: (A) Holt's, and (B) Hochsinger's, abstracted by Osler.

(A) "*A Systolic Murmur at the Base, with Cyanosis.*—This is the most common combination met with and was present in about one third of all the cases. In over 80 per cent of the cases with these symptoms, pulmonic stenosis was found. The remainder were complicated cases of quite a wide variety. Pulmonic stenosis was usually associated with a defect in one of the cardiac septa, or a patent ductus arteriosus.

"*A Systolic Murmur without Cyanosis.*—In the cases followed to autopsy this was not a frequent combination, being noted but 6 times, and usually dependent upon a defect in the ventricular septum without pulmonic stenosis, or upon tricuspid regurgitation. Judging from my own clinical experience, a systolic murmur without cyanosis is more common than is indicated by these figures.

"*A Systolic Murmur at the Apex with Cyanosis.*—Of the 6 cases with this combination, all were examples of complex malformation, the most frequent lesions being a defect in the auricular septum, transposition of the great vessels, and patent ductus arteriosus.

"*Cyanosis without murmurs* was noted 14 times. It indicates either pulmonic atresia or the transposition or irregular origin of the great vessels.

"*Diastolic murmurs* were heard in 2 cases, and depended upon pulmonic insufficiency.

"*A presystolic murmur* was noted in a single case. It was localized at the right base, and the only lesion was a patent foramen ovale.

"*Absence of both cyanosis and murmurs* was recorded in 5 cases. The lesions found were, atresia of the aorta, both arteries arising from the right ventricle, or defective septa.

"It will be seen that about the only cases in which a fairly positive diagnosis can be made is pulmonic stenosis with a deficient ventricular septum. Enlargement of the right heart, being common to nearly all the varieties, is of no diagnostic value.

"*Diagnosis of Congenital from Acquired Disease.*—Congenital disease may be suspected if the patient is under 2 years of age; if there is no history of previous rheumatism; if the murmur is atypical in its location, character, or transmission; if there is a very loud murmur at the base; if there is cyanosis; and if there is evidence of enlargement of the right heart.

"*Diagnosis of Congenital from Anæmic Murmurs.*—This is often a more difficult matter than to decide between congenital and acquired disease. From a murmur alone one should be very cautious in making a diagnosis of cardiac malformation in a very anæmic infant. Anæmic murmurs are systolic, basic, unaccompanied by enlargement of the heart; usually heard in the carotids, often in the subclavian arteries, but are seldom so loud as those due to malformations. In some cases it may be necessary to watch the effect of treatment or the course of the disease before deciding the question."

(B) "(1) In childhood, loud, rough, musical heart murmurs, with normal or only slight increase in the heart dulness, occur only in congenital heart disease. The acquired endocardial defects with loud heart murmurs in young children are almost always associated with great increase in the heart dulness. In the transposition of the large arterial trunks there may be no cyanosis, no heart murmur, and an absence of hypertrophy.

"(2) In young children heart murmurs with great increase in the cardiac dulness and feeble apex beat suggest congenital changes. The increased dulness is chiefly of the right heart, whereas the left is only slightly altered. On the other hand, in the acquired endocarditis in children, the left heart is chiefly affected and the apex beat is visible; the dilatation of the right heart comes late and does not materially change the increased strength of the apex beat.

"(3) The entire absence of murmurs at the apex, with their evident presence in the region of the auricles and over the pulmonary orifice, is always an important element in differential diagnosis, and points rather to septum defect or pulmonary stenosis than to endocarditis.

"(4) An abnormally weak second pulmonic sound associated with a distinct systolic murmur is a symptom which in early childhood is only to be explained by the assumption of a congenital pulmonary stenosis, and possesses therefore an importance from a point of differential diagnosis which is not to be underestimated.

"(5) Absence of a palpable thrill, despite loud murmurs which are heard over the whole præcordial region, is rare except with congenital defects in the septum, and it speaks therefore against an acquired cardiac affection.

"(6) Loud, especially vibratory, systolic murmurs, with the point of maximum intensity over the upper third of the sternum, associated with a lack of marked symptoms of hypertrophy of the left ventricle, are very important for the diagnosis of a persistence of the ductus Botalli, and can not be explained by the assumption of an endocarditis of the aortic valve."

III. DISEASES OF THE ARTERIES

I. **Arteriosclerosis.**—A chronic inflammatory and degenerative disease of the vascular system, usually involving the arteries (*arteriosclerosis*), sometimes the capillaries as well (*arteriocapillary fibrosis*), seldom the veins (*phlebosclerosis*), or all three (*angiosclerosis*). There is thickening of the vessel walls due to fibrous overgrowth, affecting all three coats, but mainly the intima, of the vessel. The process may be diffuse, involving the aorta and its branches more or less uniformly; or nodular and patchy, occurring principally in the aorta and the larger arteries. The nodular areas may soften (*atheromatous abscess*) and discharge (*atheromatous ulcer*), subsequently becoming calcified; or calcareous deposits may take place in the sclerotic plaque without softening. The diffuse form affects the smaller arteries rather than the aorta, but the nodular variety is often associated.

Causes.—Either as a result of the normal loss of elasticity due to old age, or a similar loss resulting from the degeneration caused by chronic intoxications, or overstretching by prolonged high arterial tension, the vessels become dilated. In order to lessen the abnormally large calibre of the vessel and to restore the normal velocity of the blood stream, the intima thickens—practically a compensatory process. There is in many instances a congenital or inherited lack of elasticity in the arteries which predisposes toward their premature senility. It is more common in males.

The causes of arteriosclerosis are old age; gout, syphilis, alcohol, and lead; muscular overwork; chronic nephritis, which may be cause or result; overeating; and rheumatism, typhoid fever, or scarlet fever. Sclerosis of the pulmonary artery and sometimes of the pulmonary veins may occur in conditions which cause prolonged high pressure in the lesser circuit, especially mitral stenosis and emphysema; and the portal veins may be sclerosed in hepatic cirrhosis.

Arteriosclerosis usually becomes evident between 40 and 55 years of age, but may appear in the early 20's or as late as 60.

Symptoms.—The accessible arteries are thickened or atheromatous, the pulse is the *pulsus tardus* (page 406) and of high tension. Owing to the peripheral resistance offered by the more or less obstructed vessels the signs of left-side hypertrophy are manifest. The apex beat is shifted to the left, the impulse is forcible and heaving, and the aortic closure is loud, ringing, and accentuated; and if the aortic valves and the root of the aorta are atheromatous, it will be of a peculiar harsh quality as well. Late in the disease the evidences of cardiac dilatation may ensue.

In addition to the signs just mentioned, which are common to all cases, there are special manifestations depending upon the fact that the vessels in particular organs or regions may have undergone more decided sclerotic changes than in other vascular areas.

(1) The *cardio-vascular symptoms*, oppression, palpitation, and dyspnœa, on exertion, may be pronounced. If the coronary arteries are particularly involved there may be angina pectoris, or evidences of chronic myocarditis. If dilatation ensues there will be dyspnœa, œdema, dropsies of serous cavities, and diminished urine. Thrombosis of the coronary arteries or rupture of the heart, with sudden death, and aneurism of the heart, are possible occurrences; so also with aneurism in general. (2) *Renal symptoms*, due to interstitial nephritis (*q. v.*), may be present. (3) *Cerebral symptoms* are common. There may be vertigo (a frequent symptom), headache, and ringing in the ears. Vertigo may coexist with attacks of faintness, epileptiform seizures, and bradycardia. Temporary attacks of aphasia, hemiplegia, or monoplegia may occur, probably significant of thrombosis in the smaller almost obliterated cerebral arteries, or of the lodgment of small fibrinous emboli from the roughened aorta. Cerebral hemorrhage may occur, the brittle arteries, often the seat of miliary aneurisms, suddenly rupturing. (4) Because of obliteration, thrombosis, or embolism of peripheral arteries, gangrene of the extremities may ensue. (5) Due to sclerosis of the vessels on the splanchnic area there may be attacks of severe pain in the upper abdomen, lasting from a few minutes to an hour, perhaps recurring several times a day. Such attacks may alternate with, or accompany or be part of, angina pectoris, and are precipitated by the same causes as the latter. (6) Involvement of the spinal arteries in the sclerotic process is responsible for *senile paraplegia*.

In the *acute* form, due to hæmorrhage or thrombosis of these arteries, there is a rather sudden paralysis of both legs, with little or no pain. The loss of power may be marked, with little ataxia; or slight, with well-developed ataxia, depending (Dana) upon whether the central or the postero-lateral arteries are the more involved. Improvement is usual, but relapses are frequent.

The *chronic* form occurs especially in heavy-weights and gouty high-livers. Granular kidneys or diabetes, or both, are apt to coexist. There is a slowly progressive weakness of the legs, often with numbness and some pain upon exertion. The patient complains of an overpowering sense of weariness after mental or physical effort. At first the disturbance of the gait amounts to no more than a shuffling or dragging of the feet; but after a time it becomes difficult to go up or down stairs, or, finally, to walk at all. The prognosis is bad. The basis of the disease is a thickening and obliteration of the blood-vessels, with consequent softening of the gray matter of the anterior bones.

Diagnosis.—Thickened arteries, high tension pulse, left ventricular hypertrophy, and an accentuated aortic closure constitute indubitable evidence of arteriosclerosis. Hard arteries alone do not necessarily imply aortic changes unless the aortic second sound is of a harsh quality. A prolonged first sound is an early sign.

When dilatation has occurred and the murmurs of relative aortic or mitral incompetency are found, it may be difficult or impossible to determine, in the absence of previous observation of the case, whether the condition is a sequel of chronic valvular disease or arises from arteriosclerosis without primary valvular defects.

Prognosis.—Ultimately grave, although the general health may remain good for many years. The symptoms may come gradually—e. g., with polyuria, slight traces of albumin, and a few hyaline casts, as in the granular kidney, due to arteriosclerosis; or suddenly, as in cerebral hemorrhage.

II. **Aneurism in General.**—*Forms.*—A *true aneurism* is a more or less localized dilatation of an artery, the aneurismal sac consisting of one or more of the coats of the vessel. The dilatation may be fusiform, saccular, or cylindrical. A *dissecting aneurism* is one in which the intima ruptures and the blood forces itself between the layers of the vessel wall. A *false aneurism* is a circumscribed collection of blood outside of the vessel, and due to rupture of the latter. When an abnormal communication exists between an artery and a vein it constitutes an *arterio-venous aneurism*; and the communication may be direct, *aneurismal varix*, or a sac may intervene, *varicose aneurism*. The aneurisms of greatest clinical interest to the physician are the true sacculated or fusiform varieties.

Causes.—In the majority of cases aneurisms are due to weakening of the arterial walls by arteriosclerosis. If the latter is diffuse, the dilatation is generally fusiform and irregular; if circumscribed, the yielding is saccular. Of the causes of sclerotic changes, syphilis is the most important in producing aneurism. Prolonged high arterial tension, as in laborious muscular work, cardiac hypertrophy, etc., predispose. A great and sudden strain, as in heavy lifting or violent coughing or straining, may initiate the dilatation if the coats are weakened by previous disease. Non-septic embolism of an artery may cause a dilatation on the proximal side of the obstruction; and, in connection with malignant endocarditis, acute inflammatory or degenerative lesions leading to aneurismal dilatations may result from an extension of the process to the aorta, or the lodgment of infective emboli elsewhere. The arteries may be hereditarily of poor quality. Aneurisms occur more frequently in men than in women, and most commonly between 30 and 50 years of age.

According to their site the following *varieties* of aneurism are clinically recognisable. Of all arteries the aorta is most commonly the seat of aneurism. Aneurisms of the thoracic aorta outnumber those of the abdominal aorta by about 20 to 1.

III. **Aneurism of the Thoracic Aorta.**—A majority of thoracic aneurisms are saccular and involve the ascending portion of the arch, starting not infrequently at the very root of the vessel. Next most frequent are those of the transverse and descending portions of the arch and the descending thoracic aorta, in the order of mention.

Symptoms.—Depending upon the size, site, and direction of growth of the aneurism, the condition may be *latent*, even with large dilatations, or may be manifested by distinct *pressure effects*, with or without external *physical signs.* One does not usually realize, without looking at a cross-section of the chest (Fig. 268), that a transverse line touching the anterior surface of the bodies of the dorsal vertebræ lies about at the middle of the antero-posterior diameter of the thorax. Thus if a thoracic aneurism grows posteriorly there is ample space for the development of a large sac without causing external pulsation.

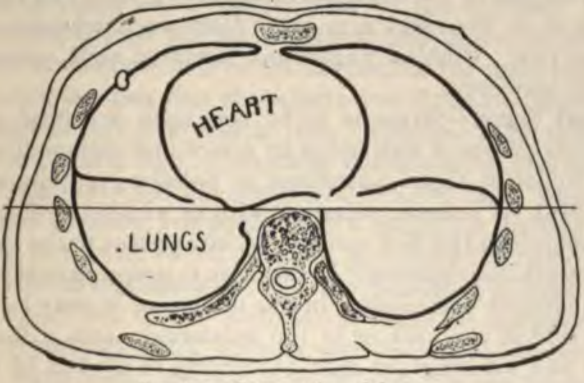


Fig. 268.—Cross-section of thorax.

Aside from the physical signs, later to be described, the most prominent general pressure symptoms are pain, dyspnœa, cough, changes in the voice, and dysphagia. In greater detail these and other symptoms are as follows:

Pain, which, although variable, is usually an early and constant symptom, is apt to occur in paroxysms. It may be steady and boring, often very severe if pressure-erosion of bone is occurring; frequently it is sharp, radiating, and neuralgic, and is reflected to the neck or down the arm (aneurism of arch), or along the intercostal nerves (aneurism of descending aorta), and attacks resembling angina pectoris are not infrequent, especially if the ascending portion of the arch is dilated. Dyspnœa and stridulous respiration, together with a dry, paroxysmal, brazen, ringing, or wheezy cough, and hoarseness or aphonia are significant of pressure on a recurrent laryngeal nerve, generally the left, and causing spasm or paralysis of the corresponding vocal cord, by aneurism of the transverse portion of the arch. Dyspnœa and cough are also due to pressure upon the trachea or the left main bronchus, and the cough may be accompanied by a thin or thick expectoration if bronchitis or bronchiectasis results from the compression of the air tubes. Dysphagia arising from pressure upon or spasm of the esophagus is a somewhat infrequent symptom. Spitting of blood in small quantities, sometimes persistent, may occur, coming from the congested and altered mucous membrane of the trachea at the point of compression; large hemorrhages may arise from rupture of the sac into the trachea, bronchi, or lung. Owing to irritant pressure on the sympathetic there may be dilatation of one pupil, perhaps with pallor of the same side of the face; or miosis, due to paralyzing pressure, perhaps with unilateral congestion and sweating of the face. There may be distention of the veins and œdema of the head and arm due to pressure on the superior vena cava, or of the right arm alone from encroachment upon the subclavian vein. Clubbed fingers and incurved nails, perhaps of one hand only, may occur.

Physical Signs.—Stress is to be laid upon a careful inspection from various points of view aided by direct and oblique illumination in order to detect slight pulsations or pulsating prominences (Fig. 103, page 346). If present, pulsation, with or without swelling, is seen most commonly in the first and second interspaces to the right of the sternum, much less commonly in the same interspaces to the left; and if the innominate participates in the dilatation it may be visible on the right side of the neck or in the episternal notch. Occasionally pulsation and swelling are seen posteriorly to the left of the spine. The swelling, which may be hardly perceptible, or, on the other hand, exceed a large orange in size, may involve the sternum and the adjacent costal cartilages. The overlying skin may be greatly thinned or may have ulcerated through, thus revealing the fibrinous layers of the sac wall, from which blood may ooze. The tumour may be impressible and fluctuating; more commonly, depending on the thickness of the deposit of fibrin, it is firm and resistant. The pulsation of the swelling is expansile—the sac enlarging in every direction—and often forcible and heaving. If there is no visible swelling or pulsation, firm pressure with the fingers of one hand in front, opposed by the other hand placed posteriorly, may develop it to the accustomed touch. There may be a systolic thrill; and a diastolic shock, often well marked, is an important physical sign. The apex beat may be carried downward and to the left, most commonly by reason of the pressure of a large aneurism; less frequently because of simple cardiac hypertrophy due to overwork.

Percussion is negative except in aneurisms of a certain size which approach the chest wall sufficiently to cause dulness. The percussion sound is flat and resistant, and the area of dulness varies in position according to the seat of the aneurism, as will be subsequently described. *Auscultation* may reveal a systolic murmur, largely dependent upon the thickness and irregularities of the fibrinous layers, loudest over the aneurismal sac, and transmitted to the arteries of the neck, but which, taken alone, is of little diagnostic value. When a double murmur is heard the diastolic component is usually due to coexisting relative or organic aortic insufficiency. Unless the murmur of the latter replaces it, the aortic second sound is, in aneurisms of the arch, accentuated, ringing and snappy—the auditory equivalent of the palpable diastolic shock—an important diagnostic sign. Occasionally a diastolic murmur alone may be heard, and perhaps also a systolic whiff in the trachea. The *pulse*, in the arteries beyond the sac, is delayed and altered in character, so that if the innominate is involved the beat of the right radial may be appreciably later than that of the left; but if the dilatation is beyond the innominate the converse is true. Tracheal tugging (page 282), an extremely useful sign in otherwise obscure cases, may be detected.

Diagnosis.—(1) *Of the Aneurism.*—In the cases which present well-marked pressure symptoms *plus* a pulsating swelling, the diagnosis can, as a rule, be safely made, especially if arteriosclerosis is present or its causes operative, and the patient is between 30 and 45 years of age. But if the aneurism is small or deep-seated, or if pressure symptoms alone, without physical signs, are present, particularly when slight, scanty, or indefinite, the diagnosis is always difficult and may be impossible.

(2) *Of its Site.*—In the *ascending portion of the arch* the physical signs often predominate—e. g., external tumour and dulness, manifest usually to the right, rarely to the left, of the sternum, over the 2d and 3d interspaces. The veins of the neck, head, and upper extremities may be swollen from pressure upon the superior cava; occasionally there is œdema of the right arm alone from pressure on the subclavian; and there may be swelling of the lower extremities if the aneurism is sufficiently large to compress the inferior cava.

The innominate artery rarely participates. The heart may be displaced to the left. Aphonia and dyspnœa, due to pressure on the right recurrent laryngeal, are common.

In the *transverse portion of the arch* the pressure symptoms often dominate the physical signs. Thus there may be dyspnœa and cough from pressure on the trachea; dysphagia from pressure on the esophagus; bronchitis, bronchiectasis, perhaps pulmonary abscess, from pressure on a bronchus; brassy cough and aphonia from pressure on the left recurrent laryngeal nerve; pupillary changes from pressure on the upper dorsal or lower cervical ganglia; and rapid loss of flesh due to compression of the thoracic duct. A tumour, if present, appears in the middle line or to the right of the sternum, the manubrium having been eroded; rarely to the left of the sternum. The innominate or left carotid and subclavian arteries may be involved, with corresponding delay and alteration in the radial or carotid pulses. Œdema of the left side of the head and neck indicates pressure on the left innominate vein.

In the *descending portion of the arch* the aneurism exerts pressure against the vertebræ (3d to 6th dorsal) with resulting erosion and destruction. The pain caused by this process is extreme; and finally a large swelling may appear posteriorly in the scapular region. Paraplegia may result from compression of the cord after the vertebral canal has been opened. Dysphagia from pressure on the esophagus is common; and from pressure on the left bronchus, bronchiectasis, abscess, or gangrene may ensue. In aneurism of the *descending thoracic aorta* a tumour may appear posteriorly over or to the left of the lower dorsal vertebræ.

Differential Diagnosis.—The following conditions may require to be discriminated from thoracic aneurism:

(1) *Mediastinal Tumour.*—It may be quite impossible to make this distinction, for if the tumour is deep-seated the pressure symptoms are practically identical with those of an aneurism in the same locality. But if it be a growth there is absence of the tracheal tugging, the accentuated aortic second sound, and the differences in the radial pulse so often associated with aneurism. A tumour may also appear externally and perhaps pulsate, but the expansile character of the pulsation is absent; so also is the strong palpable systolic, and especially the diastolic, impulse or shock of the heart sounds. Moreover, in tumour pain is more common, the cervical or axillary lymph glands may be enlarged and hard, and cachexia may be manifest, whereas the aneurismal patient is often strong and robust.

(2) *Pulsating Empyema Necessitatis.*—The history of a pleurisy, the absence of the diastolic shock and firm expansile pulsation, the

presence of the tumour farther to the left than is customary in aneurism, the usual flatness at the base posteriorly, the absence of pressure symptoms or alterations in the pulse, and, finally, the finding of pus by puncture with a hypodermic needle, will speak against aneurism.

(3) *Aortic Insufficiency.*—In this there may be strong visible and palpable pulsation of the aorta, with or without slight dilatation (not sufficient to be considered a fusiform or cylindrical aneurism) of the vessel, possibly also an increase of dulness over its course. The differentiation may be very difficult, especially as aneurism of the arch may coexist with aortic insufficiency. A positive diagnosis of aneurism should not be made under these circumstances unless a distinct tumour with expansile pulsation is present, or well-marked pressure symptoms are manifest.

(4) *Neurotic (Dynamic) Pulsation of the Aorta.*—A case of this kind came under observation, in which at first there was much uncertainty as to the presence of an aneurism in spite of the fact that the patient was a woman, had no arterial sclerosis, and was not of the alcoholic or labouring class. There was forcible throbbing and actual swelling in the episternal notch and behind the right sternomastoid muscle; the heart sounds, especially the aortic closure, were abnormally distinct and accentuated, and there was a systolic bruit; but, on the other hand, there was increased objective and subjective pulsation of many peripheral arteries, retinal pulsation, throbbing in the head, paroxysms of tachycardia (200 and over), and many neurotic manifestations. At the present time the throbbing has practically disappeared, coincidently with improvement in the ataxic condition of the vasomotor nerves—an improvement to which the administration of suprarenal extract has been distinctly contributory.

(5) *Other Conditions.*—Displacement of the heart and aorta by tuberculous or other retraction of the right lung, or by lateral curvature of the spine, may cause a strong pulsation to the right of the sternum. If an aneurism compresses a bronchus, causing bronchiectasis or abscess, with cough and fever, it may be mistaken for tuberculosis, but the absence of bacilli and the presence of other signs and symptoms of aneurism will usually prevent this error.

Terminations and Prognosis.—The aneurism may perforate externally and terminate fatally by repeated small, or sudden and large, hemorrhages, or rupture into the pleura, pericardium, superior cava, trachea, bronchi, lung, or esophagus, or death take place by asthenia. The prognosis is always bad, although life may be prolonged, under constant threat of sudden death, for a number of years.

IV. **Aneurism of the Abdominal Aorta.**—May be fusiform or sacculated, rarely multiple, and is seated most commonly immediately below the diaphragm in the vicinity of the cœliac axis.

Symptoms.—The principal subjective symptom is pain. If the tumour grows backward, causing erosion of the vertebræ, there may be a dull boring pain in the back, usually with darting neuralgic pain in the abdomen and lateral and posterior lumbar regions. From pressure on the cord there may be numbness and tingling of the lower extremities, followed by paraplegia. If the tumour, as is more commonly the case, grows forward, there may be gastralgic paroxysms, colicky pains, vomiting, or diarrhœa, usually due to pressure, infrequently to embolism of the superior mesenteric artery. Jaundice has been noted.

Physical Signs.—Usually there is marked epigastric pulsation, sometimes with a palpable thrill. There may be a distinct, fixed (exceptionally freely movable) tumour with a firm, expansile pulsation; occasionally, when the tumour is in contact with the diaphragm and receives the direct thrust of the heart, the pulsation is double. If the aneurism is of sufficient size there may be an area of dulness which is continuous with that of the left lobe of the liver. Although the tumour presents most commonly in the epigastric region, it may be found, depending on the direction in which it increases, in the left hypochondrium or lateral and posterior lumbar regions. A large aneurism, if seated just beneath the diaphragm, may not be palpable. A systolic bruit is usually heard over the swelling, but its point of maximum audibility may be over the back. A soft diastolic murmur has been noted. The pulse in the femoral arteries is usually small and delayed.

Diagnosis.—It is to be remembered that visible and palpable pulsation, often violent, is very common, while aneurism of the abdominal aorta is a quite infrequent condition. A diagnosis of aneurism is not to be made unless a distinct tumour is found which can be seized by the hand and presents a strong expansile pulsation. Neurotic or dynamic *throbbing of the aorta* occurs in anæmic and neurasthenic states, particularly in women, and may be so strong as to suggest almost irresistibly the presence of an aneurism. But the latter should be the very last cause assigned as an explanation of abnormal epigastric pulsation. An enlargement or tumour of the *left lobe of the liver*, or a tumour of the *pylorus* or *pancreas*, may simulate aneurism, but the pulsation is not heaving and expansile, and with the assumption of the knee-elbow posture the tumour falls forward and the transmitted pulsation usually disappears.

Terminations and Prognosis.—Except in rare instances of spontaneous cure the prognosis is grave, death resulting in most cases from rupture into the pleura, peritoneum, or retroperitoneal tissues and intestines, especially the duodenum, compression of the spinal cord, closure of the vessel by deposits of fibrin, or intestinal infarction from embolism of the superior mesenteric artery (OSLER).

V. **Aneurism of the Splenic Artery.**—A diagnosis is rarely possible. The symptoms resemble those of gastric ulcer, viz., epigastric pain, vomiting, and hæmatemesis. There may be a tumour in the left hypochondrium, or apparent increase of splenic dulness. The tumour may pulsate and present a systolic bruit.

VI. **Arterio-venous Aneurism.**—An aneurism of the ascending portion of the aortic arch may open into the superior vena cava. The *physical signs*, in addition to those of the aneurism proper, are the abrupt development of venous distention, œdema, and cyanosis of the head, neck, arms, and upper thorax. There may be a thrill, and a humming or buzzing murmur, continuous, but intensified during the systole of the heart.

SECTION V

DISEASES OF THE BLOOD AND DUCTLESS GLANDS

PREPARED BY HENRY P. DE FOREST, M. D.

I. ANÆMIA

A DISEASE of the blood characterized by a reduction of the amount of the blood as a whole, or of the cells, or of the hæmoglobin or albumin. Two forms are recognised: *secondary* and *primary*.

I. **Secondary Anæmia.**—*Causes.*—Any condition or disease which abstracts the blood from the body or deprives it of any one of its essential ingredients may cause a secondary anæmia, such as acute and severe or gradual and long-continued hemorrhages (gastric or intestinal ulcers, bleeders); or the drain upon the albuminous ingredients of the blood due to nephritis, prolonged lactation, tuberculosis, suppuration, or malignant neoplasms; or inanition from any cause; or the action of poisons, either inorganic, such as mercury lead, antimony, arsenic, or the organic poisons of malaria or syphilis. High temperature (fever, sunstroke) interferes with the action of the hæmatopoietic organs. *Blood Examination.*—In hemorrhages the red cells are diminished in number and size and there is a moderate poikilocytosis. Normoblasts appear, and there is a moderate leucocytosis. The hæmoglobin is diminished in greater proportion than the red cells. In disorders causing a continued drain, like nephritis or tuberculosis, the blood coagulates slowly, there is a marked poikilocytosis, a few normoblasts, and a moderate or extreme leucocytosis.

II. **Primary Anæmia.**—Disease of the blood itself. Two varieties are recognised: *chloro-anæmia* and *pernicious anæmia*.

(I) **Chlorosis.**—This is essentially a disease of young girls between the ages of 14 and 17, when secondary sexual changes are taking place. Recurrences may take place later in life.

Symptoms.—As a rule these develop slowly. Menstrual disturbances, delayed menstruation or amenorrhœa, may first attract attention. Languor, malaise, anorexia, and palpitation are common symptoms. Systolic hæmic murmurs at both apex and base, and venous murmurs, are common; diastolic murmurs are rare. The pulse is usually soft and full, but as a result of the altered condition of the blood and the enfeebled circulation, thrombosis and embolism are not infrequent complications. Fever of a mild type, cold hands and feet, fainting, neuralgia, and shortness of breath, perhaps severe dyspnœa after slight exertion, are common symptoms. The appetite is variable. Acids seem to be especially desired, though hyperacidity of the gastric juice is the rule. Digestion is slow. So frequent, obstinate, and habitual is the constipation that by some it is regarded as the real cause of the disease. The skin is often puffy and edematous, and the œdema of the face and ankles may suggest Bright's disease. A yellowish-green tinge, oftentimes pathognomonic, is added to the general pallor of the skin. The eyes are bright, the sclerotics bluish.

Hæmal Symptoms.—The blood flows slowly and with difficulty, clots quickly, and is distinctly paler than normal blood. The essential change is in the amount of hæmoglobin, which is diminished far more in proportion than are the erythrocytes. Twenty to thirty per cent of the normal amount of hæmoglobin is by no means unusual, while the red cells may remain at 80 or 90 per cent of the normal. Aside from their pallor, the erythrocytes may show no changes, but in severe cases poikilocytosis is marked, normoblasts appear, and a slight leucocytosis is the rule.

Diagnosis.—The peculiar colour of the skin, the history of a radical change of climate, poor food, bad ventilation, nervous and emotional disturbances, and persistent constipation, combined with the hæmanalysis, render the diagnosis, as a rule, easy. Chlorosis is to be discriminated from *chronic nephritis* by urinalysis, although the two conditions may coexist, and *pulmonary tuberculosis*, which produces pallor of the skin and bluish sclerotics very apt to be mistaken for chlorosis. Careful physical examination and the sputum analysis will establish the true diagnosis.

(II) **Progressive Pernicious Anæmia.**—Persons of middle age are usually affected, males more frequently than females.

Symptoms.—It is difficult to improve upon Addison's description: "It makes its approach in so slow and insidious a manner that the patient can hardly fix a date to the earliest feeling of that languor which is shortly to become so extreme. The countenance gets pale, the whites of the eyes become pearly, the general frame flabby rather than wasted, the pulse perhaps large, but remarkably soft and compressible, and occasionally with a slight jerk, especially under the slightest excitement. There is an increasing indisposition to exertion, with an uncomfortable feeling of faintness or of breathlessness on attempting it; the heart is readily made to palpitate; the whole surface of the body presents a blanched, smooth, and waxy appearance; the lips, gums, and tongue seem bloodless, the flabbiness of the solids increases, the appetite fails, extreme languor and faintness supervene, breathlessness and palpitation are produced by the most trifling exertion or emotion; some slight œdema is probably perceived about the ankles; the debility becomes extreme—the patient can no longer rise from the bed; his mind occasionally wanders; he falls into a prostrate and half-torpid state, and at length expires; nevertheless, to the very last, and after a sickness of several months' duration, the bulkiness of the general frame and the amount of obesity often present a most striking contrast to the failure and exhaustion observable in every other respect."

Palpitation (common), hæmic murmurs (constant), visible arterial pulsation, full water-hammer pulse, a capillary and, not infrequently, a venous pulse are the principal circulatory symptoms. Hemorrhages, petechial or ecchymotic, in the skin, mucous membranes, and retina are not infrequent. Anorexia, dyspepsia, nausea, vomiting, diarrhœa, becoming progressively worse, are nearly always present. The specific gravity of the urine is low and the colour pale. Not infrequently the excess of iron and the resulting colouring matter in the liver and other parts of the body produce an excess of urobilin and make the urine a dark sherry red. The body is rarely emaciated. The complexion is pale, smooth, waxy, and of a marked lemon-yellow colour.

Hæmal Symptoms.—The red corpuscles are notably diminished; even to 150,000 per cubic millimetre. The hæmoglobin is *relatively increased*—a condition exactly opposite to that found in chlorosis and in some of the secondary anæmias. A constant symptom is a wide variation in the size and character of the erythrocytes; microcytes, megalocytes, normoblasts, and gigantoblasts are all found in varying numbers; poikilocytosis is the rule. The leucocytes are unchanged or occasionally diminished; in grave cases the mononuclear forms increase, and the polynuclear forms decrease.

Diagnosis.—The rapid course of the disease, its apparent gravity, the characteristic colour of the skin, and the results of hæmanalysis are the cardinal symptoms. In the blood itself the relative increase of hæmoglobin, and the presence of the larger forms of cells (megalocytes and gigantoblasts) are of especial importance.

Differential Diagnosis.—*Chlorosis.*—In the "green sickness" the appearance of the patient is widely different, and the results of the hæmanalysis make confusion difficult, if not impossible.

Secondary Anæmias.—Each of the secondary anæmias has a definite cause and associated symptoms. Pregnancy, parturition, atrophy of the stomach, parasites (bothriocephalus, anchylostoma), and the malarial, typhoidal, and cancerous cachexias produce conditions that closely simulate idiopathic pernicious anæmia, but the microscope will usually reveal the difference.

Prognosis.—The majority of patients die, but with recent methods of treatment (arsenic) recoveries have become more common. The predominance of normoblasts (indicating regenerative changes) is a favourable sign; an excess of gigantoblasts or of mononuclear leucocytes indicates a graver prognosis.

II. LEUCÆMIA

The majority of all cases occur in early adult life (20 to 40 years); 60 per cent are males. Two types occur: *spleno-medullary* or *myelogenous*, and *lymphatic* leucæmia (*lymphæmia*). Usually well marked, but transitional or combination forms are occasionally observed.

I. **Spleno-medullary Form.**—By far the most common.

Symptoms.—The spleen gradually enlarges, ultimately it may become enormous, occupying fully one half of the abdominal cavity. It may be immobile from adhesions, or freely movable within the peritoneal cavity. Usually the free and notched border is plainly felt or even seen below the free border of the costal cartilages. A sense of weight, dragging, and oppression in the left side are often the first symptoms for which relief is sought. Rupture has occurred from the intense hyperæmia. At times a splenic *bruit* can be heard; in other cases the vessels entering the hilus are so enlarged that a marked thrill may be felt over the organ. Owing to the pressure of the enlarged spleen the apex beat may be displaced upward and to the left. Other cardiac symptoms are rare. The *pulse* is usually rapid, soft, compressible, but of good volume. As a result of the feeble circulation, œdema of the feet, general anasarca, or abdominal ascites may develop. Epistaxis is common; hæmoptysis rare; hæmatemesis (not common) may be the first symptom, and the loss of blood so great as to cause death. Bleeding gums, extensive purpura of skin and mucous membranes, leucæmic retinitis, and intracranial hemorrhages have been recorded.

Gastro-intestinal symptoms often develop early in the disease. Besides hæmatemesis, intestinal hemorrhages may occur, or a dysenteric process develop in the colon. Nausea and vomiting are early, frequent, and persistent symptoms. Jaundice occasionally occurs, and diarrhœa (even fatal) is not infrequent. Toward the end of the disease pulmonary œdema or pneumonia may develop and be the immediate cause of death. The movements of respiration may be restricted because of the mechanical pressure of the hypertrophied spleen. Dyspnœa, an early and characteristic symptom, is due to the anæmia; so also are the headache, dizziness, or fainting spells frequently observed. As a rule the organs of special sense suffer more severely than does the general nervous system. With the occurrence of intracranial extravasation of blood various sensory or motor disturbances arise according to the location of the exudate; coma (rare), deafness (common), and optic neuritis (rare) are the chief symptoms recorded. Priapism is not infrequent. There is no constant urinary symptom. Hæmaturia (occasional) and an excess of uric acid, nearly always present, seem to bear a direct relation to the size of the spleen and to the excess of leucocytes.

An extraordinary hyperplasia of the red bone marrow occurs in this form of the disease, and as a result the internal tension in certain of the long and flat bones (sternum, ribs) may become so great that nodular tumours develop along the course of the bone. These swellings may be sensitive to the touch or even become the seat of more or less inflammation. The change in the bone marrow is characteristic of this type of the disease, for from this source arise the abnormal forms of leucocytes (myelocytes) which are diagnostic of leucæmic blood. By many, indeed, the disease is called "myelogenous leucæmia," and it is held by some that the rôle played by the spleen is purely passive.

Hæmal Symptoms.—Although anæmia is not a necessary symptom, it sooner or later appears, and the changes which can then be observed in an accurate hæmanalysis constitute the most characteristic evidence of the disease. The leucocytes are enormously increased, in some instances even exceeding the erythrocytes in number, those which are derived from the red bone marrow (myelocytes) characteristically and greatly predominating. Mast cells and eosinophiles are increased. The other forms of leucocytes are often in normal proportion, or even relatively diminished. In general the number of red corpuscles is markedly diminished, but rarely becomes less than 2,000,000 per cubic millimetre. Normoblasts and true gigantoblasts are present in considerable numbers. The amount of hæmoglobin is relatively reduced still further than are the erythrocytes. The hæmoglobin tends to crystallize out and Charcot-Leyden crystals deposit on standing. The alkalinity of the blood is diminished and the fibrin increased; as a result the fibrin network on a blood slide is unusually thick and dense. Owing to the enormous increase in the leucocytes, the blood clots more quickly than usual and the coagulum has a pale or even puslike colour.

II. **Lymphatic Form.**—Lymphatic leucæmia (lymphæmia) is rare; it is more rapid and fatal than the form just described; it occurs in younger subjects, and occasionally develops acutely and terminates fatally in from 2 to 3 months.

Symptoms.—In this form of the disease there is a general lymphatic enlargement. All of the superficial groups may be involved, but never to the extent that occurs in pseudo-leucæmia, nor do the glands become matted together; they are, as a rule, soft, isolated, movable, and may vary considerably in size during the course of the disease. The tonsils and the lymph follicles of the pharynx, tongue, and mouth may be enlarged. Lymphoid growths may occur in the liver, spleen, omentum, thymus gland, and skin. The bone marrow is often replaced by lymphoid tissue.

Hæmal Symptoms.—In this type of the disease the lymph glands, not the bone marrow, are the seat of hyperplastic proliferation. As a result the blood usually shows a complete absence of myelocytes but an enormous increase of lymphocytes (as high as 98 per cent). Eosinophiles and normoblasts are rare.

III. **Diagnosis and Prognosis of Leucæmia.**—Hæmanalysis, the only means of making a diagnosis of leucæmia, discovers the enormous increase of leucocytes; with preponderance of myelocytes in the spleno-medullary form; of lymphocytes in the lymphatic form. As the clinical features of leucæmia, pseudo-leucæmia, and splenic anæmia may be identical, hæmanalysis alone will differentiate them. Most cases prove fatal in from 1 to 3 years. In acute leucæmia death may occur in 2 months from the onset.

III. PSEUDO-LEUCÆMIA

A disease in which there is a progressive increase in size of the lymph-glands and spleen, with a secondary anæmia, without increase (or but slight) in the number of leucocytes, and with the formation of lymphoid growths in the liver, spleen, and other organs.

About one third of all cases of Hodgkin's disease occur in persons under 30 years of age; one third between 30 and 40; three fourths in males. The true nature of the disease is unknown, but it may prove to be of infectious origin.

Clinically pseudo-leucæmia bears a close resemblance to lymphatic tuberculosis (tuberculous adenitis) and primary lympho-sarcoma. Many good observers believe it to be simply a peculiar form of tuberculosis of the lymph-glands. But the view is apparently gaining ground that Hodgkin's disease is a specific entity, and that, when tuberculosis is present, it is often a terminal infection. The studies of Reed and Simmons tend to prove that the enlarged glands in this disease present distinctive histological characteristics, which allow them to be differentiated by the expert microscopist. Moreover, in a large majority of the cases, tubercle bacilli are not to be found in the glands, the results of inoculation experiments are negative, and, in typical examples of the disease, tuberculin causes no reaction.

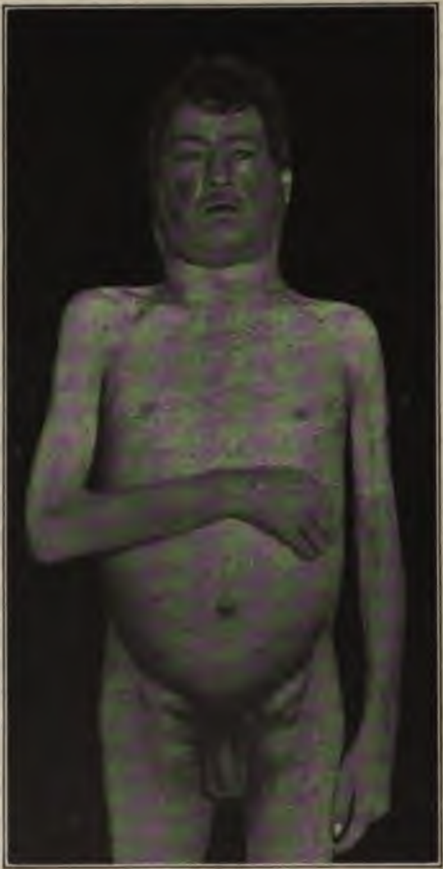


FIG. 269.—Pseudo-leucæmia (Hodgkin's disease). M. E. H. case. Photograph by De Forest.

Symptoms.—Enlargement of the cervical glands is usually the first symptom noticed. Next in order of frequency the axillary, inguinal, and, ultimately, all the lymphatic glands of the body, both deep and superficial, progressively enlarge. In women the chain of glands in direct communication with the breasts may first attract attention. Primarily the glands are isolated and easily movable;

later they fuse together in large masses, and, by thickening and adherence of the adjacent connective tissue, may become firmly fixed. The cervical glands may become enormous, obliterating the neck and giving the head a pyramidal appearance. The resulting pressure upon the trachea often causes extreme dyspnœa. In the axilla or groin the masses are large, even pedunculated, and by pressure upon vessels or nerves may cause pain and œdema of the extremities. The thoracic and mediastinal glands may give rise to marked pressure symptoms, i. e., paroxysmal dyspnœa, paralysis of one or both vocal cords with loss of speech, pulmonary congestion or œdema, and necrosis of the sternum. The large intrathoracic vessels may be so compressed that a collateral circulation is established through the mammary and epigastric veins. Of the abdominal glands the retroperitoneal are most commonly involved, and various visceral displacements often result. The ureters, the iliac vessels, the sacral and lumbar nerves may suffer, and the uterus may be so pressed forward and surrounded by the large lobular masses as to closely simulate a uterine fibro-myoma. Even the mesenteric or hepatic glands may give rise to a variety of pressure symptoms, dependent upon the site of the abdominal tumour. In any of these glands suppuration and necrosis due to pressure, while not common, may develop late in the disease.

The spleen is hypertrophied in three fourths of all cases, and usually contains marked lymphoid growths. The patient may complain of a sense of weight and dragging in the side of his abdomen, or may accidentally feel the tumour mass. Palpation usually discloses the splenic tumour, reaching, in many cases, to or below the navel.

In the early stages the blood remains normal, but as the disease progresses the hæmoglobin diminishes. Next follows a diminution in the number of erythrocytes, and ultimately the severest grades of anæmia may be reached. The qualitative changes found in severe secondary anæmia may be present. It occasionally happens that masses of the enlarged glands suppurate, or pneumonia develops, or some similar cause gives rise to a well-marked leucocytosis. If this occurs the eosinophiles are usually decreased, and, if the cachexia be marked, small numbers of myelocytes may be found. Leucocytosis is, however, to be regarded as an accidental concomitant.

The tonsils usually enlarge late in the disease, and lymphoid growths or polypi, developing in the pharynx or larynx, give rise to symptoms of nasal or laryngeal obstruction. Deafness due to closure of the Eustachian tubes is not infrequent. Nosebleed is common, and ulceration of the mucous membrane of the air passages not uncommon. Dyspnœa, palpitation, or hæmic murmurs are present as the anæmia increases. The glandular masses may displace the heart.

Digestive symptoms are not marked. Dysphagia may be present from pressure upon the gullet or ulceration of the mucous membrane. Pressure or irritation of the sympathetic may cause irregularity of the pupils, or bronzing or pigmentation of the skin; the latter may be caused also by interference with the capillary circulation. Intense itching sometimes occurs.

Fever is usually more or less marked, even from the onset of the disease. It may be of a hectic type, or simulate the paroxysms of malaria. Apyrexia, for periods of a week or so, alternating with fever, has occasionally been noted.

Diagnosis.—Progressive enlargement of the lymphatic glands and of the spleen, together with a nearly normal state of the blood, constitute the distinctive complex of pseudo-leucæmia. It may require discrimination from the following conditions: (1) *Tuberculous adenitis*, especially of the cervical group of glands, is at first extremely difficult to differentiate. The tuberculous process is more common in the young, and involves the submaxillary group more frequently than the glands lying in the anterior and posterior cervical triangles, which are usually first to be involved in Hodgkin's disease. Tuberculous glands, moreover, even when very small, tend to become fused into a firm mass, and suppuration develops early. In tuberculosis of the cervical glands one side only may be affected; in pseudo-leucæmia both sides are usually involved. Removal of the glands and inoculation experiments, or the tuberculin test may be necessary to make a positive diagnosis. (2) *Syphilitic adenitis* may present the same gross appearance, but the history and the ready response to treatment will serve for differentiation. (3) *Leucæmia*, especially of the myelogenous type, is, as a rule, readily distinguished from pseudo-leucæmia by hæmanalysis, though the gross anatomical changes may be indistinguishable. Rare cases have been observed in which pseudo-leucæmia has ultimately merged into leucæmia by transitional changes. (4) *Splenic anæmia* may be at times confused with Hodgkin's disease, but careful examination of the blood will enable a correct diagnosis. (5) *Lymphosarcoma*, for differentiation, requires a skilled microscopical examination of an excised gland.

Prognosis.—The duration of the disease varies from a few months to several years, but practically all cases die. The usual causes of death, in the order of frequency, are asthenia, pressure, and coma.

IV. PURPURA

Symptoms.—The essential feature of this condition is the appearance upon the skin or mucous membranes of minute punctate extravasations of blood beneath the skin (petechiæ), or larger and more diffuse extravasations (ecchymoses). All these are at first bright red, but as absorption takes place the colour gradually becomes darker and purple, finally fading to a brownish discoloration. With these various forms of blood extravasation there may or may not be associated other conditions affecting the joints, nerves, or general functions of the body. The *blood* is changed in its fibrin-forming elements and coagulation is retarded for 10 or 15 minutes.

VARIETIES.—Purpura, whatever form it may take, is of itself a symptom, and as such is indicative of a large number of varied conditions. The pathological classification of purpura is as yet indeterminate, but the following divisions are practically convenient:

I. **Purpuric Diseases of the Newborn.**—(I) *Syphilis Hæmorrhagica Neonatorum.*—This may not be manifest at the time of birth, but shortly thereafter there appear extensive bloody extravasations in the skin and mucous membranes; often a definite hemorrhage from the navel takes place. Jaundice is usually severe. Marked syphilitic changes exist in the liver and elsewhere.

(II) *Hæmoglobinuria Neonatorum* (*Winckel's Disease, Epidemic Hæmoglobinuria*).—This is characterized by marked jaundice with accompanying gastro-intestinal symptoms, constipation, fever, rapid pulse and respiration, and sometimes, about the 4th day, cyanosis. Punctate hemorrhages appear in different parts of the body. In the urine, from similar hemorrhages occurring within the substance of the bladder and kidneys, there is albumin and methæmoglobin. This disease is doubtless infectious, as it is often epidemic in hospitals, but its true nature has not yet been determined. A severe form, with fatty degeneration of the viscera, is sometimes known as Buhl's disease.

(III) *Morbus Maculosus Neonatorum.*—This is a form of hemorrhagic disease in which bleeding occurs from many of the mucous surfaces of the body. The blood may come from the bowels, the stomach, the mouth, or the navel, singly or combined, usually within the first week after birth. Marked jaundice may be associated with this bleeding; fever is usually present.

II. **Symptomatic Purpura.**—This may be due to a variety of causes. *Infectious diseases*—i. e., pyæmia, septicæmia, typhus fever, measles, scarlet fever, and especially certain forms of malignant endocarditis and smallpox—are often accompanied by more or less well-marked purpuric exudations. *Toxic purpura* may follow the entrance into the blood of a great number of poisons; snake bites, mercury, quinine, copaiba, ergot, and even the various iodides, sometimes give rise to this condition.

Cachexia induced by a number of severe and wasting diseases is, in the terminal stages, marked by purpura, as in Hodgkin's disease, chronic nephritis, tuberculosis, scurvy, or even in general debility. *Neuroses* such as hysteria (stigmata), Raynaud's disease, locomotor ataxia, myelitis, and, at times, severe neuralgias, may show this symptom. *Mechanical causes* followed by venous stasis (whooping cough, eclampsia, epilepsy) may give rise to blood extravasations.

III. **Arthritic Purpura.**—This form, known also as rheumatic purpura, is an occasional accompaniment of certain joint affections, and may be classed under three groups:

(I) **Purpura Simplex.**—This is most common in children, and is characterized by more or less pain, petechiæ or ecchymoses on the legs (common) or trunk (rare), fever (usually slight), diarrhœa (occasional), and rheumatism affecting the joints (common).

(II) **Purpura Rheumatica.**—*Schönlein's disease* is characterized by multiple arthritis and an eruption, either a simple urticaria or a distinct purpura, sometimes a combination of the two (*purpura urticaris*). It is often ushered in with sore throat and fever. Œdema may be excessive and general, and bloody blebs may form (*pemphigoid purpura*). The joint conditions are not, as a rule, severe or painful. The urine may be scanty and albuminous. This condition is by many regarded as a special affection.

(III) **Purpura Erythematosa.**—*Henoch's purpura* is found chiefly in the young, and is characterized by gastric pain, vomiting, and diarrhœa, more or less severe joint involvement, an erythematous form of skin lesion, and marked hemorrhages from the mucous membranes. Frequent recurrences are common. If the kidneys are involved, blood and hæmoglobin may be found in the urine and acute or chronic nephritis result. The spleen may enlarge. Angioneurotic œdema may coexist. One, two, or more of these symptoms may be absent. All may appear in a paroxysmal form.

IV. **Hemorrhagic Purpura.**—This disease (*Morbus maculosus Werlhofii*) is most commonly seen in delicate girls. General weakness, diffuse purpura of both skin and mucous membranes first occur. The blood extravasation becomes more and more disseminated, leading to epistaxis, hæmatemesis, hæmaturia, and hæmoptysis, each more or less severe. The resulting anæmia may be profound, leading, in the so-called *purpura fulminans*, to death within 24 hours.

DIAGNOSIS.—The severer forms of purpura must be discriminated from scurvy by the absence of gum symptoms, and the manner of development; and from hemorrhagic smallpox, scarlet fever, or measles, by the history and higher fever of these infectious diseases. Wright urges that the coagulability of the blood be observed in purpuric cases.

V. HÆMOPHILIA

Symptoms.—The cardinal symptom of this curious and rather rare condition is a profuse and uncontrollable bleeding following the most trivial injury. Often, indeed, the worst hemorrhage comes from the merest scratch, and may even occur spontaneously.

External bleedings may be spontaneous, but more commonly follow cuts or wounds of the skin or mucous membranes. Bleeding from the nose is most common; next in order of frequency from the mouth, intestines, stomach, urethra, and lungs. No part of the body is exempt. Interstitial bleedings occur in the form of large extravasations of blood (hæmatomata) following slight bruises, or appear spontaneously as petechial spots. The joints are not infrequently involved, particularly the larger ones, and the lesion may closely simulate an attack of acute articular rheumatism. Pain, fever, or hemorrhage from other parts of the body may soon follow the arthritis. The blood shows a marked slowness in coagulation, the normal period of from 3 to 5 minutes extending to 10 or 15, or even to 45, minutes in hæmophilia, and the blood plates may be scanty or absent. If severe hemorrhage has occurred the usual signs of a traumatic anæmia will be found.

Diagnosis.—The disease is more common in males than in females (10 to 1) and usually develops before puberty. In making the diagnosis the family history is of great importance, as a marked heredity is characteristic of the disease. The tendency is transmitted through the female to the male, though, strangely enough, neither pregnancy nor menstruation are influenced by the condition.

Certain cases, limited in their occurrence to newborn children, while simulating this condition, are distinguished from it by rapid cessation of the bleeding and by the coincident appearance of jaundice and fever. Purpura rheumatica and hæmorrhagica, and scurvy have much in common with hæmophilia but lack the peculiar family history, and occur usually in persons debilitated by improper living. The joint affections may be confounded with tuberculous disease, but ecchymoses and the history of atavism are lacking in the latter.

VI. SCURVY

Scorbutus is essentially a disease dependent upon improper diet continued for a considerable period of time, and hence affecting the entire organism. The age of the patient is an important factor in determining the symptoms, and two well-marked varieties are described: the scurvy of *infants*, and the scurvy of *adults*.

I. **Infantile Scurvy.**—This disease usually becomes manifest before the child is a year old.

Symptoms.—Bottle-fed children, without reference to their home surroundings, may develop this disease as a result of living upon food in which certain essential ingredients are lacking. Malted milk, condensed milk, and the various baby foods on the market are the chief causes of the malady.

The recognition of this disease as an entity is so recent, and the symptoms described by Barlow in his monograph are so clearly described that they are here quoted: "So long as it is left alone the child is tolerably quiet; the lower limbs are kept drawn up and still; but when its diapers are changed, or it is placed in its bath, or otherwise moved, there is continuous crying, and it soon becomes evident that the pain is connected with the lower limbs. At this period the arms may be handled with impunity, but any attempt to move the legs or thighs gives rise to screams. Next some obscure swelling may be detected, first on one lower limb and then on the other, though it is not absolutely symmetrical. The swelling is ill-defined, but is suggestive of thickening around the shafts of the bones, beginning above the epiphyseal junctions. Gradually the bulk of the limbs affected becomes visibly increased, and the position assumed becomes somewhat different from what it was at the outset. Instead of being flexed they lie everted and immobile, in a state of pseudo-paralysis. About this time, if not before, great weakness of the back becomes apparent. A little swelling of one or both scapulas may appear, and one or both arms show similar changes, though rarely as marked as in the legs. The joints are free. In severe cases another symptom may now be found, namely, crepitus in the region adjacent to the junction of the shafts with the epiphyses. The upper and lower extremities of the femur and the upper end of the tibia are the common sites of such fractures, but the upper end of the humerus may also be affected. A very startling appearance may now be observed over the front of the chest; the sternum, costal cartilages, and a portion of the adjacent ribs seem to have been forcibly jammed back toward the spinal column. Occasionally thickenings of varying extent may be observed on the surface of the skull, or even on some of the bones of the face. A remarkable eye symptom may appear; proptosis of one eyeball, with puffiness and very slight staining of the upper lid. In a day or two the other eye presents the same appearance. Little ecchymoses may appear on the conjunctiva. Coincident with these symptoms, and proportional to the amount of limb involved, a very profound anæmia develops. The complexion becomes sallow or earthy coloured, and small ecchymoses, or more rarely petechiæ, appear on various parts of the body. Emaciation is not a marked feature—in fact, during the early stages of the disease the child may appear to be unusually plump and well nourished. Asthenia is, however, well marked. The temperature is erratic; it is often raised for a day or two when successive limbs are involved, but is rarely above 102°. If teeth have appeared, the gums may become spongy and bleed freely; some of the teeth may even fall out."

Diagnosis.—The cardinal symptom is the extravasation of blood beneath the periosteum, with resulting thickening and tenderness of the shaft of the bone. The pain in the legs, their position, and the spongy and bleeding gums are symptoms of nearly equal importance. The disease may be suspected in any child who has difficulty or pain in moving the legs, or in whom paralysis is suspected. The character of the child's diet is of great importance.

Differential Diagnosis.—This is to be made from:

Acute Articular Rheumatism.—The joints, not the shafts of the bones, are involved; crepitus does not exist; the fever is much higher, and the characteristic symptoms of scurvy are absent.

Rachitis.—In this, although the early stages may be indistinguishable, there soon develops the rachitic rosary and the enlargement of the ends of the long bones; pain is, as a rule, absent, and ecchymoses, petechiæ, and spongy gums are not observed. Both may coexist.

Purpura.—There is an absence of the history of improper feeding in most cases, and the rapid improvement under treatment seen in scurvy is not common in purpura.

Infantile Paralysis.—This has a sudden onset accompanied by fever, certain special groups of muscles are affected, and the electrical reactions differ in the two diseases.

Syphilis.—In syphilitic pseudo-paralysis the onset is sudden, and there is loss of motion in the upper or lower limbs, or both, by reason of the separation of the cartilage at the end of the diaphysis. There is usually much pain and crepitation on motion. The difference in the history is still more marked.

Asthenia and *Anæmia.*—Some early cases of scurvy can not be recognised, and are regarded as cases of anæmia or asthenia.

Prognosis.—When the disease is recognised and appropriate treatment instituted at an early date nearly all cases recover.

II. **Scurvy in Adults.**—All ages are attacked by the disease, but the old are more susceptible to it. More males than females are subject to it, probably because of greater exposure to unfavourable conditions of food, weather, and work.

Symptoms.—These develop slowly, after a period of exposure to cold, fatigue, poor and damp quarters, and a diet deficient in green vegetables. Weight and strength are progressively lost. A marked pallor becomes apparent, and the skin gets rough and dry. The breath is offensive; the tongue is swollen, red, and furred; the gums become spongy, bleed easily, and the teeth loosen and may fall out. There may even be ulceration and loss of tissue from the gums and necrosis of the maxillary bones.

The *blood* shows nothing characteristic. Interstitial hemorrhages, petechial in character, soon begin to appear, at first around the ankles, then extending up the legs to the trunk and arms. The mucous membranes of the mouth, bowels, and urinary tract are also the site of similar lesions. On the surface of the body the hair follicles are especially the seat of hemorrhages. Still larger extravasations are determined by insignificant bruises. As in infants, subperiosteal extravasation of blood may occur, but, owing to the firmer structure of the bony tissue, results in the formation of nodes rather than fusiform swellings. Actual hemorrhages may occur, of which epistaxis is the most common, hæmaturia and bleeding from the bowels are next in frequency, while hæmoptysis and hæmatemesis are rare. A curious condition of the skin is produced—"scurvy sclerosis"—a firm, inelastic induration of the subcutaneous tissues with a purplish discoloration of the surface from multiple petechiæ. Large intermuscular clots may break down and indolent ulcers result.

The *circulatory symptoms* are those due to a poorly nourished heart, namely, palpitation, with a feeble and irregular impulse. Hæmic murmurs are common. The *nervous symptoms* are those of low vitality, slow, unresponsive, and depressed mentality, headache, or, with the occurrence of extravasation into the various tissues of the brain, convulsions or hemiplegia. The chief *osseous symptoms* which have been observed are necrosis, separation of the epiphyses or of the costal cartilages, destruction of a recent callus, and effusion of blood into the larger joints. The appetite is lost or uncertain, and there is nausea or repugnance to the mere sight of food; vomiting, delayed digestion, constipation, or diarrhœa. The latter may be so severe that, with the addition of the blood oozing from the intestinal mucous membrane, there may be a condition simulating a severe dysentery. The *respiration* is normal but shallow. Dyspnœa may occur, and infarctions of the lung have been found post mortem.

Diagnosis.—With increased care of the rations scurvy has almost disappeared, even on the smaller ocean craft. The history of crowded and damp quarters, overwork, cold, hardships, and poor food should at once suggest the diagnosis. Isolated cases are more difficult to decide upon, and are with difficulty separated from some forms of purpura. The rapidity of recovery when the food is properly regulated will establish the diagnosis in many instances.

VII. STATUS LYMPHATICUS

(*See page 32*)

VIII. ADDISON'S DISEASE

Causes.—Addison's disease is usually due to tuberculosis of the suprarenal capsules, and the symptoms depend upon the loss of function of these bodies. Most cases occur between 20 and 40 years of age. It predominates in males (2 to 1).

Symptoms.—Its onset is, as a rule, insidious. A light yellow or light brown *pigmentation of the skin* is usually the first symptom to attract attention. It progressively deepens over the entire body, and is especially marked over the pigment-bearing areas, the areolas of the nipples, and the genitals. Finally, a marked bronzing of the entire skin surface is observed, with circumscribed, sometimes extensive, areas of a dark brown or black colour. The exposed or irritated skin surfaces are also deeper in colour. Patches of leucoderma are occasionally observed. The mucous membranes are also pigmented.

Addison called attention to the irritability of the stomach in these cases. Nausea, vomiting, anorexia, abdominal pain, retraction of the abdominal walls, and more or less profuse diarrhœa occur, but all vary greatly in severity and frequency. General languor and debility, slight at first but progressively increasing, are symptoms which occur before the discoloration of the skin suggests the true nature of the disease. These increase in severity till asthenia becomes the most characteristic feature of the disease. Lassitude and muscular prostration are marked even in well-nourished, robust, and muscular persons. The heart's action becomes remarkably feeble, irregular, or paroxysmal. There are accompanying vascular disturbances, such as vertigo, syncope, headache, coma, or convulsions; the syncope may even be fatal. Although anæmia was at one time regarded as an essential and characteristic symptom, it is by no means so common as was once supposed. The number of erythrocytes or the amount of hæmoglobin may even be increased. As a rule the leucocytes show no marked changes, although in a few instances melanin has been observed in them.

Complications.—Acute general miliary tuberculosis may develop at any time during the course of the disease. This is due to the fact that the new growth within the suprarenal capsule is nearly always tuberculous, and the involvement of some one of the blood vessels may lead to the dissemination of the tubercle bacilli to various parts of the body. Symptoms develop according to the distribution of the new foci of disease.

Diagnosis.—The cardinal symptoms upon which the diagnosis rests are pigmentation of the skin with marked and progressive asthenia; in doubtful cases the response to the tuberculin test.

Differential Diagnosis.—Pigmentation of the skin may develop from a great variety of causes and conditions: abdominal neoplasms, particularly melano-sarcoma; pregnancy, uterine disease, torpidity or disease of the liver, ulcer or dilatation of the stomach, exophthalmic goitre, splenic leucæmia, pseudo-leucæmia, scleroderma, comedones; even dirt and the resulting irritation of the skin, have all at times given rise to sufficient discoloration or pigmentation of the skin to lead to the diagnosis of Addison's disease. In argyria the history and the absence of the asthenia will establish the diagnosis.

Prognosis.—Thus far the cases reported have had a fatal termination. The use of suprarenal-capsule extract may in the future modify the mortality.

IX. DISEASES OF THE SPLEEN

We here consider changes developing *primarily* in the spleen.

I. **Movable Spleen.**—*Symptoms.*—These vary with the degree of mobility and size of the gland. Some cases are accidentally discovered by palpation and give rise to no subjective symptoms whatever. In other instances, especially if the organ is enlarged as well as displaced, there is a sense of weight and dragging in the left side, which, when the cause is discovered, gives nervous patients much anxiety. Rarely torsion of the pedicle occurs, and swelling, pain, fever, and even necrosis may follow. The *diagnosis* is easily made by palpation, unless adhesions exist. Exploratory laparotomy may be necessary in obscure cases.

II. **Rupture of the Spleen.**—In the excessive degree of hyperæmia sometimes present in typhoid, malarial, and other fevers, spontaneous rupture may occur. Traumatism is a more common cause. The symptoms of acute and severe intra-abdominal hemorrhage at once develop. Abdominal section confirms the diagnosis. The mortality is high; an immediate operation saves a small proportion.

III. **Infarct, Abscess, and New Growths of the Spleen.**—An infarct of the spleen may occur as a result of emboli or thrombosis in endocarditis, typhoid, and similar affections. Splenic pain, tenderness, and swelling occurring in pyæmia may cause a suspicion of the condition, but, as a rule, the diagnosis is practically impossible; so also with the rare abscesses, cysts, and neoplasms of the gland, unless they reach such a size as to be evident upon percussion or palpation. The true nature of the enlargement is only to be determined by an exploratory operation.

IV. **Splenic Anæmia.**—Primitive splenomegaly occurs at all ages, but is more common in males than in females.

Symptoms.—The spleen is, as a rule, very large, but aside from mechanical discomfort this gives rise to no symptoms. Hæmatemesis is common, and is often the symptom for which the patient first seeks relief. Hæmaturia and purpura have also been observed. Mechanical obstruction is probably the cause of all of these symptoms. Ascites is not infrequent, due either to the enlarged spleen or to the associated anæmia. Œdema of the extremities or general anasarca may occur (rare). Pigmentation of the skin (melanoderma) is seldom observed; in some instances the colour has been as dark as in pronounced cases of Addison's disease. To a group presenting, in addition to the enlarged spleen, cirrhosis of the liver and ascites the name of "Banti's disease" has been given.

The anæmia may cause only a slight pallor, or it may be as intense as that of progressive pernicious anæmia. A relatively high red cell count is found (average 3,400,000), with a relatively low amount of hæmoglobin (average 45 per cent); and the leucocyte count is low (2,500), with an extreme leucopenia (500) in about half of all cases. The differential count of the leucocytes frequently shows a relative lymphocytosis at the expense of the polymorphonuclears. The red corpuscles are changed somewhat; poikilocytosis is common; normoblasts and megaloblasts are not infrequent.

Diagnosis.—The cardinal symptoms are primitive splenomegaly, with anæmia and without enlargement of the lymph glands.

Pernicious Anæmia.—The differentiation may be extremely difficult, but the small size of the spleen and the relatively high amount of hæmoglobin are characteristic of pernicious anæmia.

Splenic Leucæmia.—Those cases of leucæmia in which the leucocytes gradually diminish and remain at their normal number for protracted periods are difficult to differentiate. As a rule the blood examination will determine the diagnosis.

Pseudo-leucæmia.—The hæmanalysis and the size of the spleen may be identical in the two conditions, but the progressive enlargement of the lymph glands is characteristic of Hodgkin's disease.

Cirrhosis of the Liver with Enlarged Spleen.—Whether the cirrhosis be due to alcoholism or to syphilis, the spleen is often enlarged; even a simple hypertrophy of the liver may be followed by splenic enlargement. The two conditions may coexist. A knowledge of the history and progress of each disease is essential to accurate separation of such cases; so also is their behaviour under appropriate treatment (e. g., mercury).

Malaria with enlargement of the spleen and paludal cachexia, may be excluded by the history, the absence of the plasmodium from the blood, and the differences observed on hæmanalysis.

X. CHRONIC CYANOSIS, POLYCYTHÆMIA, ENLARGED SPLEEN

Under this descriptive title (also as Vaquez's disease) a number of writers, notably Osler, Cabot, and Vaquez, have recently written concerning a series of clinical manifestations which apparently occur with sufficient regularity to constitute a new disease entity. The symptoms are persistent cyanosis, and a more or less marked splenic tumour. A limited number of cases have been reported in which the splenic tumefaction was tuberculous. In the remainder no specific cause was found. It is a disease of adult life.

The first noticeable symptom is the cyanosis, which may be general but is most marked over the prominences of the face and in the extremities. Examination of the blood shows a great increase in the erythrocytes and hæmoglobin (11,150,000 and 170 per cent respectively in a case carefully reported by Weber and Watson) and a moderate increase in the leucocytes (8,000), constituting a true plethora. Abnormal cells are not demonstrable. The viscosity is regularly increased. The proportion of the various forms of leucocytes does not seem to be disturbed.

In the necropsy of Weber and Watson's case the bone marrow, bright-red in colour, was found to be surcharged with erythroblasts. All the blood vessels, particularly the smaller ones, were engorged. No abnormalities were demonstrated in the viscera. Various subjective symptoms, due to the vascular engorgement, have been observed, e. g., headache, dizziness, fulness in the head, buzzing in the ears, Menière's vertigo (in one case), occasional attacks resembling paralysis, mental and physical torpor, cough (frequently), dyspnœa, general pains, nausea and vomiting. In all the cases cold weather increased the cyanosis. Urinary changes seem fairly constant, including low specific gravity, traces of albumin, and often hyaline and granular casts.

In almost all the reported cases the vascular tension was much increased. Osler mentions a pressure ranging from 172 to 200 mm. Cardiac murmurs were rarely detected. Constipation is repeatedly mentioned. In most of the cases the condition had been noticed for periods of from 6 to 10 years, progressing toward a fatal termination.

The condition must be differentiated from congenital cardiac malformations, organic heart disease, emphysema, fibroid phthisis, and coal-tar poisoning, e. g., acetanilide. The diagnosis rests on the marked polycythæmia.

XI. DISEASES OF THE THYROID GLAND

I. **Goitre.** — *Symptoms.* — One lobe, both lobes, or the entire gland, may progressively enlarge and form a bilobed tumour extending across the front of the neck. This tumour may be of varying composition: vascular, cystic, or parenchymatous with colloid degeneration. In each instance the resulting disfigurement may be at first the only cause for complaint. Later, as the mass increases in size, pressure symptoms develop according to the structures involved—pain, dyspnœa, aphonia from paralysis of one or both vocal cords, or even sudden death from compression of the vagus on one or both sides.

This same group and train of symptoms develop if the gland becomes the seat of a new growth. Adenomata, carcinomata, and sarcomata are the forms of neoplasm most frequently found in this locality. Abscess of the thyroid, a condition rarely observed, may give rise to similar symptoms.

Diagnosis.—Inspection reveals the enlargement, but its exact nature can only be determined by a surgical operation. The *prognosis* depends entirely upon the size of the tumour and the mechanical disturbances caused by it.

II. **Exophthalmic Goitre.**—Graves's, Basedow's, or Parry's disease is most frequent in early adult life, between 20 and 30, preponderating in women (3 to 1). Several in the same family may suffer. It is probably due to excessive thyroid action (hyperthyrea).

Symptoms.—Four cardinal symptoms characterize this disease: enlargement of the thyroid gland, exophthalmos, tachycardia, and tremor. These do not always develop in the same order, but ultimately all are well marked.

Enlargement of the Thyroid.—As in simple goitre, one or both sides may be affected, although the size of the growth is not so extreme. The thyroid vessels become dilated, and a noticeable thrill may be felt, or even seen, and various murmurs heard over the tumour mass.

Exophthalmos.—Synchronously, as a rule, with the thyroid enlargement one or both eyes become more prominent. At first this may be due merely to the infrequent winking and to the altered adjustment of the eyelid, whereby a line of the white eyeball appears above and below the cornea. In looking downward the upper lid does not follow the movement of the eyeball. Soon an actual protrusion of the eyeball becomes apparent; this becomes more and more marked, and an actual dislocation of the eye from its socket has been observed. Vision remains normal, as a rule, but the arteries of the retina throb, and can be seen to pulsate. A general inflammation of all ocular structures may destroy the eye (rare).

Tachycardia.—The heart action progressively increases in rapidity, although intermission is rare; 140 to 160 beats per minute, or even higher, are not infrequently observed. As a result of this the cardiac impulse may be felt over the entire chest, and even heard at a considerable distance (5 feet in 1 case). The pulsation can be seen over the præcordial region and over the vessels of the neck. A general arterial distention occurs, and is a marked and distressing symptom; the patient may be unable to sleep because of the jarring sound in the ears conveyed by the carotids. The arterioles are dilated and hot flushes with profuse sweats occur. The arterial pulse can be felt in the finger tips. A well-marked venous pulse has been observed (rare). *Tremor* is a well-marked cardinal symptom, fine, general, involuntary, and appearing early.

Miscellaneous Symptoms.—The skin may show a pigmentation closely simulating that of Addison's disease, and leucoderma, severe pruritus, or urticaria. In the underlying connective tissue patches of solid œdema or of myxœdema may appear. As a result of this excess of moisture in the skin and underlying structures a marked diminution in electrical resistance has been noted.

Vomiting and diarrhœa, in paroxysmal attacks, are not infrequent. As a result of impaired nutrition, anæmia, emaciation, and slight fever progressively increase. Marked changes occur in the nervous system. The mind may remain unaffected, but irritability, change of disposition, mental depression, melancholia (rare), or acute mania (more common) have been noted. This last symptom, when it does occur, is of considerable importance, for death sometimes rapidly supervenes. Glycosuria, albuminuria, or diabetes may be observed. These various symptoms vary markedly in intensity from time to time and in different individuals.

Diagnosis.—Bearing in mind the four cardinal symptoms just described, the diagnosis is usually easy. Acute or chronic forms occur. A certain proportion of recent cases recover, but recovery in well-established cases is rare.

III. **Myxœdema.**—In goitre and exophthalmic goitre there is an excessive action of the thyroid gland (hyperthyrea); but in the conditions about to be described the opposite state prevails (athyrea).

(I) **Cretinism.**—*Symptoms.*—May develop immediately after birth, or at any time up to puberty. In the *sporadic* type of the disease the thyroid gland is either congenitally absent or deficient, or fails to develop. The child usually appears like other children until about 6 months of age. About that time marked developmental defects begin to be manifest. Growth of body and mind is retarded. The fontanels do not close; the hair is thin, the skin dry, the tongue lolls loosely from the mouth and appears to be too large. The pale and yellowish face is swollen, and the toadlike eyes peer through a mere slit between the puffy lids (Fig. 37, page 172). The nose is flat, and the few teeth appear late, at infrequent intervals, and promptly decay. The hands and feet of this pot-bellied child are stubby and nearly useless appendages to the short and stumpy limbs. The mind is as deformed as the body; idiocy and imbecility are common terminal stages. In certain parts of the world (Switzerland, France, and Italy) an *endemic* form of this disease exists, associated with goitre. The resulting symptoms are much the same as in the sporadic cases, but there is doubtless a difference in the primary cause not clearly understood at present.

Diagnosis.—Is, as a rule, easy, provided the existence of this disease be borne in mind. Owing to its rarity in America unrecognised cases doubtless occur. The non-deposition of lime salts in the bones, giving rise to the condition known as *fœtal rickets*, and the permanent preservation, in the adult, of childlike characteristics of body and mind, *infantilism*, can easily be differentiated. Formerly nearly every case died. Of the few who survived there resulted a curious form of dwarfs, with short legs, big joints, and the toadlike and repulsive facial expression described.

(II) **Juvenile Myxœdema.**—It occasionally happens that in a child who up to the age of 6 or 8 years has been healthy and normally developed, the occurrence of some of the infectious diseases, or of a direct septic involvement of the thyroid, causes the gland to atrophy or its function to be suspended. The same symptoms then develop, save for the differences in age, as are about to be described.

(III) **Myxœdema in Adults.**—As in exophthalmic goitre, women are much more subject to the disease than men (6 to 1). It is more common in adults between the ages of 20 and 40 years.

Symptoms.—In women these bear no direct relation to menstruation, pregnancy, or the menopause. The body, as a whole, becomes more bulky and appears to be generally swollen or edematous, but the swelling is firm, inelastic, and does not pit on pressure. The skin is dry and rough. The wrinkles are obliterated, and this change in the face gives rise to a curiously stolid expression (Fig. 39, page 173). The features change, the nose, lips, and cheeks broaden, the mouth is enlarged, and the tongue appears too big. Red patches may mark the skin over the nose and on the cheeks. The hair is dry, stiff, and sparse. The muscles of the body become flabby, and a slow and hesitating gait develops.

Headache, defective memory, delayed mentality, an irritable and suspicious disposition, and finally delusions, hallucinations, dementia, and coma, follow the course of the disease within the brain. The thoracic and abdominal viscera remain normal, though the power of resistance to malign influences is diminished. Tuberculosis is therefore a common cause of death. The thyroid progressively diminishes in size, finally the glandular elements disappear, and only a shriveled and fibrous structure marks the location of the gland. Exophthalmic goitre sometimes develops coincidently with myxœdema, and the symptoms of the two conditions may be combined.

Diagnosis.—The diagnosis of uncomplicated myxœdema is usually easy if the causes and symptoms be carefully studied. Bright's disease is the ailment most commonly confounded with myxœdema and the urinary symptoms may be very similar in each affection. The solid character of the swelling and the fact that it does not pit on pressure as does renal œdema, the loss of hair, the dryness of the skin, and the peculiar mental state are the chief features by which the true nature of the lesion can be best determined.

Prognosis.—With proper treatment (thyroid extract) the percentage of recoveries has greatly increased within the past decade.

(IV) **Operative Myxœdema.**—As a result of operations in which the thyroid has been entirely removed a condition of myxœdema (cachexia strumipriva) develops in about one sixth of all cases. Allowing small portions of the gland to remain, or the presence of accessory glands (not infrequent), may prevent the occurrence of the characteristic symptoms, but when the gland is quite obliterated the myxœdematous cachexia develops with comparative rapidity.

XII. DISEASES OF THE THYMUS GLAND

Symptoms.—The functions of this gland are unknown, the disorders caused by its disease are rare, and the resulting symptoms obscure. Such symptoms as do arise develop, as a rule, before the age of puberty. If the gland persists after the 15th year instead of undergoing atrophy in the normal manner, or if the gland is unusually large (hypertrophy of the thymus), or if it be invaded by tuberculous, syphilitic, or neoplastic deposits, or if hemorrhage occurs or an abscess develops within the substance of the gland, and by any of these means the size of the gland be made disproportionate to its normal relations at the root of the neck, various symptoms due to pressure may develop. Pressure upon the trachea may cause dyspnœa; upon the adjacent nerve trunks, thymic asthma, spasm of the glottis, or laryngismus stridulus; if the large blood vessels are compressed, congestion or œdema may result.

Diagnosis.—Marked increase in the size of the gland may give rise to an increased area of dulness, on percussion, along the left sternal border from the 2d to the 4th ribs. The exact relationship existing between this purely mechanical pressure and thymic asthma, laryngismus stridulus, exophthalmic goitre, spasm of the glottis, or acute dyspnœa is so indefinite, that at the present time accurate diagnosis of thymic disorders is without any firm basis.

SECTION VI

DISEASES OF THE KIDNEY

Prepared by Henry Goodwin Webster, M. D.

(See also pages 511 to 516 and 681 to 712)

Movable Kidney.—*Symptoms.*—These are frequently lacking. In some cases there is dragging pain in the lumbar and sacral regions, occasionally colicky abdominal pain, intercostal and lumbo-abdominal neuralgia, and, rarely, the kidney itself is tender. Movable kidney is often associated with the multiple symptoms of hysteria, neurasthenia, and hypochondriasis, and serious mental anxiety often dates from the discovery of the condition. The various forms of nervous dyspepsia often coexist, and there may be prolapse of the stomach (gastroptosis), rarely dilatation, or descent of many abdominal viscera (splanchnoptosis) of which movable kidney is a part. Constipation is common, intestinal obstruction and jaundice (from pressure) are rare. The characters of the urine in movable kidney have been described elsewhere (page 709).

Attacks of severe abdominal pain with chills, fever, nausea, vomiting, and prostration (Dietl's crisis) may occur, probably from twisting or kinking of the ureter. The occurrence of oliguria during the attack, perhaps with swelling of the kidney (hydronephrosis), followed by an excessive flow of clear urine with subsidence of pain and swelling, renders this explanation plausible, at least in cases attended by the symptoms just described.

Differential Diagnosis.—The kidney may be *palpable*, or *movable* as far down as the navel, or *floating*, in which case it may be carried to various parts of the abdomen below the level of the umbilicus. The diagnosis depends upon the physical examination (page 513).

A movable kidney may be mistaken for an enlarged gall bladder, but the latter descends with inspiration and can not be moved except in the arc of a circle with its centre at the end of the 8th rib, whereas the movable kidney can be carried downward. Moreover, a distended gall bladder when pushed backward tends to return to its former position, while the movable kidney when reposited will frequently elude subsequent palpation. In rare instances a tumour of the intestine or the ovary may give rise to doubt, but a careful physical examination, together with the history, is usually sufficient to settle the question. In the large majority of cases movable kidney occurs in women, and it is the right kidney which is prolapsed.

II. **Renal Congestion** (*Hyperæmia*).—Presenting as the initial stage of nephritis, or in the course of the infectious and contagious diseases, or associated with the inorganic poisons, it is really a part of one or the other of these affections. An *acute* and a *chronic* hyperæmia are recognised.

(1) **Acute Congestion.**—This may occur after exposure to cold, after poisoning by turpentine, cantharides, and the like, and after operations upon the urethra, bladder, or kidney. The *symptoms* are diminution or suppression of the urine, which may contain blood, albumin, or casts, separately or together. More or less prostration is present. In severe cases, after nephrectomy, the patient may pass into the typhoid condition with delirium. After injury, collapse may supervene with suppression. A single attack of acute congestion is not in itself dangerous, but repeated attacks tend to induce nephritis. Where serious complications do not exist the outlook is good.

This condition is not likely to be mistaken, it being merely necessary to distinguish it from its accompanying conditions.

(2) **Chronic Congestion.**—Chronic hyperæmia of the kidney being due to chronic disease of the heart or lungs with vascular interference; or to pressure, as by tumours, ascitic fluid, pericardial effusion, pregnancy, and the like, presents, for the most part, the symptoms of its associated disease. Diminished secretion of urine is the most constant *symptom* (see also (3), page 710).

The *prognosis* depends largely on the causative condition, but from the tendency of chronic congestion to produce permanent changes in the kidney structure and so induce chronic nephritis, it is serious. Indeed, some writers do not attempt to separate this condition from diffuse nephritis.

III. **Uræmia.**—This name is given to a congeries of symptoms occurring in the course of acute or chronic nephritis, puerperal eclampsia, some cases of obstructed urinary excretion, and occasionally in patients with pronounced vascular changes alone.

Symptoms.—These, as usually described, include headache and sleeplessness, paralyses, amaurosis, convulsions, mania, vomiting, delirium, coma, increased arterial tension, and dyspnœa. Often there is marked fever. Sometimes muscular spasm is present. Many patients develop repeated acute attacks of uræmia of varying intensity, or the disease may pursue a chronic course. See *also* (17), page 711.

Mentioned somewhat more in detail, headache, usually occipital and generally severe, may be the only symptom, and may be continuous for a long period. Associated with sleeplessness it may lead to mania. Other nervous phenomena, such as itching, numbness, and cramps, may accompany it. It is often conjoined with high arterial tension. Paralysis may take any form, though hemiplegia, monoplegia, and aphasia are perhaps most frequent. The first and last are often associated with or succeeded by coma.

Amaurosis is not infrequently present in puerperal eclampsia. It appears suddenly and lasts for a short time only.

Convulsions, typical of eclampsia, may be mild or severe, single or multiple in type. In children they are usually dependent on acute Bright's disease and are not necessarily grave; in adults they are frequent in the later stages of chronic nephritis, when œdema has set in and the heart is failing; or they may appear suddenly at any time in the chronic interstitial form. These seizures may closely simulate epilepsy. The patient may complain of prodromal headache and restlessness, though often the attack occurs without warning. During the short intervals between the convulsions unconsciousness is the rule. The temperature varies. It may be subnormal during the seizure, but has a tendency to rise subsequently. Increased arterial tension with congestion is usually present. As already noted, children respond readily to treatment, so that the appearance of convulsions in such cases need not cause undue anxiety, but in older patients their occurrence is always grave.

Mania may appear suddenly in persons in whom nephritis is not suspected and who have never displayed indications of mental trouble. This is especially true of puerperal cases, in which attacks may accompany successive pregnancies.

Vomiting may be only a consequence of the general disturbance dependent upon the disordered vascular system, but is sometimes seen as the only marked symptom of an apparently slight nephritis. In such instances its onset is abrupt, its course intense, and its outcome not infrequently fatal. At times diarrhœa, or catarrhal or membranous colitis, may accompany such attacks. A form of stomatitis with furred tongue, foul breath, œdema, hyperæmia, and swelling of the lips and buccal mucosa, is described as peculiar to uræmia.

Delirium and coma are usually associated, and commonly appear toward the end of chronic nephritis. The onset is gradual, remissions may occur, but the course is toward a fatal termination.

Increased arterial tension of varying degree is very generally present in the uræmic state, many of the uræmic symptoms, such as headache, being associated with it. Dyspnœa may be due to mechanical causes, such as ascites, pleuritic effusion, pulmonary œdema, and the like, but that peculiar to uræmia is probably dependent on circulatory changes. It may simulate bronchial asthma in all but the characteristic "wheezing." It is paroxysmal at first, increases in frequency, and at last is continuous, the patient being unable to lie down. Toward the end the Cheyne-Stokes type of breathing may occur. The condition may persist for years, and is at first amenable to treatment directed to the circulatory trouble.

Diagnosis.—From alcoholic or opium coma, see pages 76, 77.

Cases of uræmic coma coming on slowly, with fever, and without convulsions, muscular spasms, and the like, may be mistaken for typhoid, but a positive Widal reaction may make the diagnosis clear.

Perhaps the most difficult differentiation is from cerebral disease. Uræmic hemiplegia and monoplegia occur, as previously noted, without cerebral lesions; but a suffused face, stertorous breathing, eyes turned toward the side of the hemorrhage, full, slow pulse, and hemiplegia should suggest apoplexy. In rare instances it is possible for meningitis to be confused with uræmia.

Prognosis.—This is always grave, but mild cases may recover and the patient survive for years.

IV. **Acute Bright's Disease.**—*Causes.*—Acute diffuse nephritis follows cold and exposure; the acute infectious diseases, especially scarlet fever, typhoid, diphtheria, and the like; the ingestion of poisons, such as arsenic, turpentine, cantharides, and carbolic acid; occurs sometimes as a result of extensive cutaneous burns, and more frequently in the course of pregnancy.

Symptoms.—The clinical symptoms include some or all of the following manifestations: More or less general serous effusion, anæmia, chills, pain, nausea and vomiting, sometimes fever, and, most characteristic of all, urinary changes.

The effusion may vary from simple puffiness about the ankles or the eyelids to general anasarca, or there may even be ascites or pleural effusion. The amount of fluid present is no indication of the intensity of the nephritis, as violent and rapidly fatal disease of the kidneys may be accompanied with little swelling. Pulmonary œdema is of not infrequent occurrence, and even œdema of the glottis has been noted. The skin, however, is dry. Anæmia appears early and is very generally present. Chills occur in some instances, particularly when the disease is dependent on exposure, but are by no means frequent—a statement which also applies to nausea and vomiting.

There is no temperature curve peculiar to acute Bright's disease. The fever may run high, especially in children, or there may be but little rise of temperature. The pulse may in some instances be hard, of increased tension and increased rapidity. Rapid cardiac dilatation with fatal issue has been reported. Uræmia is occasionally seen. Hemorrhagic retinitis occurs.

As a rule, the urinary symptoms (see (4), page 710) are the only ones which afford a clew to the real nature of the cases.

Regarding the *course* of the disease no fixed rule applies, as its duration and intensity vary greatly. The cases running the most acute course are usually those subsequent to scarlet fever, when suppression and œdema are the rule, although rapidly fatal cases may lack the latter, and hæmaturia may be the first cardinal symptom.

Differential Diagnosis.—Recognition of this condition should not be difficult. Of course the alert practitioner will be on the lookout for it as a sequel of scarlet and typhoid fevers, and as a manifestation in the course of pregnancy. Following exposure and the eruptive fevers, its onset is usually sufficiently marked to call attention to the real nature of the trouble, but in many cases of pregnancy, and when it appears in young children, it may begin so insidiously as to escape attention, and an eclamptic seizure may be the first symptom noted. It must be borne in mind that simple febrile albuminuria, with a few hyaline casts, does not constitute Bright's disease.

Prognosis.—This is always serious, a fatal issue sometimes ensuing as early as the 2d or 3d day. An increase in the dropsical symptoms, with small, rapid, low-tension pulse and increasing albumin, are grave symptoms, while increased urinary secretion and diminishing albumin are hopeful signs. The tendency to chronicity must not be forgotten. Scarlatinal nephritis should show an improvement in from 7 to 10 days, with complete recovery after 3 or 4 weeks in favourable cases. A much longer course—8 to 10 weeks—is not incompatible with a perfect restoration of structure and function.

V. **Chronic Bright's Disease.**—Different authorities recognise a number of sub-varieties of this disease, but for purposes of clinical diagnosis it is sufficient to describe a chronic *diffuse* or *parenchymatous*, and a chronic *interstitial* form. Amyloid degeneration is not a distinct form of Bright's disease, but an incident in either of the forms of chronic nephritis, or a manifestation of some one of the cachexias, such as that of tuberculosis, carcinoma, and the like. It will receive separate mention.

(I) **Chronic Diffuse Nephritis.**—Pathologically, the large white kidney—chronic diffuse or parenchymatous nephritis with exudation.

Symptoms.—As previously stated, an acute nephritis may merge into a chronic nephritis, or the chronic form may be primary and develop insidiously. If it follows the acute form the symptoms will be similar but of modified intensity. Developing as a primary affection, possibly dependent upon chronic alcoholism, syphilis, febrile diseases, and the like, its first indication may be a gastro-intestinal crisis. Moderate œdema of the feet or about the eyelids calls attention to a possible nephritis, and urinalysis makes the diagnosis certain. The cardinal symptoms of chronic diffuse nephritis are dropsy, anæmia, and the urinary changes. Associated with them may be uræmic symptoms, headache, nausea, and vomiting. A distinctive facies, due to the puffiness about the eyes and the marked anæmia, is insisted on by some writers as peculiar to this form of nephritis. Recurring attacks of bronchitis are of frequent occurrence. The tension of the pulse increases, the arteries gradually become stiff, and the left ventricle hypertrophied. There may be hemorrhagic retinitis. For the *urinary changes* see (5), page 710.

Prognosis.—This is always grave. Recovery may take place, especially in young subjects, but the course is for the most part toward a fatal issue, either from uræmia, œdema of the lungs, or intercurrent disease.

(II) **Chronic Interstitial Nephritis.**—Pathologically this is variously termed cirrhosis of the kidney, chronic productive nephritis without exudation, contracted kidney, etc.

Causes.—Chronic parenchymatous nephritis may merge into this form (small white kidney); it may result from arteriosclerosis, gout, alcoholism, or syphilis; or it may occur spontaneously.

Symptoms.—Its early stages pass unrecognised, though it may well be looked for in hearty eaters where a habit of high arterial tension exists. Although essentially a disease of middle life and old age, it is occasionally met with in young children. Its symptoms are manifested in the uropoietic, circulatory, respiratory, nervous, and digestive systems, and in the eyes, ears, and skin. The *urinary manifestations* are described in (6), page 710.

Of the early manifestations, increased arterial tension is the most important. It may be the only cardinal symptom in a congeries which includes headache, palpitation, bronchial cough, tinnitus aurium, muscæ volitantes, dizziness, malaise, anorexia, and a number of kindred subjective phenomena. Closely following it we find hypertrophy of the left ventricle, with eventual enlargement of the entire organ, and corresponding changes in the character of the apex beat. Reduplication of the first sound is not uncommon, while the second sound, as heard over the aortic area, is accentuated and has a peculiar ringing quality. A systolic murmur, heard at the apex and transmitted to the left, may develop later; and toward the end, when dilatation succeeds and compensation fails, any or all of the symptoms of chronic endocarditis and myocarditis may appear. The central nervous changes are those noted under uræmia. Retinitis, choked disc, and amaurosis are frequent ocular symptoms, and ringing in the ears and sudden deafness may occur.

Gastro-intestinal symptoms such as anorexia, nausea, vomiting, and diarrhœa are almost always present in greater or less degree. The tongue is generally coated. It may be red, dry, and cracked, or moist and glazed, or covered with a brownish scum, or furred and foul. Uræmic, and often cardiac, dyspnœa is of frequent occurrence; bronchitis is a very regular accompaniment; œdema of the lungs is often seen toward the last; and œdema of the glottis may occur. Eczema, dry and itching skin, "pins and needles," cramp, numbness, and other cutaneous and nervous manifestations occur, although œdema is rare. Where present it is generally merely a slight puffiness of the feet and ankles.

Differential Diagnosis.—The distinction must be carefully drawn between cases of diabetes insipidus, the "urina spastica" of neurotic and hysterical patients, and cases of interstitial nephritis with a large output of thin, clear urine. Daily examination of concentrated specimens of the latter will show sooner or later the casts and albumin which distinguish it. The character of the pulse also will throw light on the true nature of the condition. As the onset of the disease may simulate an attack of dyspepsia, gastro-enteritis, bronchitis, or even cerebral disease, the importance of careful urinalysis as a routine measure can not be underestimated.

Prognosis.—This is generally bad. While many sufferers from chronic interstitial nephritis go on in comparative comfort for many years, there is but one outcome. Eventually the arteriosclerosis determines cardiac disease, and the patient becomes subject to repeated cardiac or uræmic attacks of increasing intensity, or dies from apoplexy, œdema of the lungs, or intercurrent disease.

VI. **Amyloid Kidney.**—The cases where amyloid disease is manifested only in the kidney are very rare, and in these it is doubtful if the diagnosis can be made from the urinalysis alone. But occurring in association with the amyloid degeneration of the liver, spleen, and other organs which may follow the severe cachexias, or coming on during a chronic nephritis, it presents symptoms sufficiently marked to permit a diagnosis. Sequent to syphilis, general tuberculosis, osteomyelitis, or the cancerous cachexia, there may appear a condition characterized clinically by certain *urinary findings*, for which see (7), page 710. Amyloid disease of the kidney is seldom responsible for dropsical symptoms; nor are cardiac, arterial, and ocular symptoms common. The diagnosis rests upon the combination of polyuria with amyloid degeneration of other viscera.

VII. **Pyelitis.**—*Causes.*—Consequent upon renal calculus, local tuberculosis, the acute infectious diseases (diphtheria, typhoid fever, scarlet fever, gonorrhœa, and others), purulent cystitis, obstructed ureter, sarcoma of the kidney, pregnancy, and many other conditions, the pelvis of the kidney may become the seat of inflammation which may be limited to the pelvis alone (*pyelitis*); or to the pelvis and kidney substance (*pyelonephritis*); or the entire organ may be replaced by a single abscess cavity (*pyonephrosis*).

Symptoms.—These are separable into early and late, the former preceding suppuration, the latter accompanying it. At the inception of the disease, as in the course of typhoid fever, there may be moderate backache, tenderness to deep pressure over the kidneys, a marked rise of temperature, often decided chills and sweating. The urine remains acid, but becomes more or less turbid. For the *urinary characters* of the varieties of pyelitis see (8), page 710.

The milder cases of pyelitis, such as occur in fevers, often pass unnoticed, as they may give no decided symptoms, complete and spontaneous recovery occurring, but in those which proceed to suppuration the symptoms become more distinctive. A chill with a decided rise of temperature usually marks the establishment of the purulent process, while the increase of pus cells may in extreme cases render the urine almost milky. A septic temperature, with very marked accessions and remissions and repeated chills, is characteristic of the earlier stages, and persists in severe cases, but in those of less intensity the chart shows merely a small evening rise, dropping to normal the next morning. This is particularly the case when the pyelitis is tuberculous. After a longer or shorter time the effect on the general health becomes marked. The patient becomes anæmic, loses flesh and strength, his appetite is poor, he has night sweats, and usually runs a constantly elevated temperature. The disease may exhibit marked remissions and run a course of many years' duration.

In other more virulent cases the kidney substance becomes the seat of multiple pus foci, which may later coalesce, and the patient soon exhibits the effects of the septic process. Inspissation of the pus may occur, thus blocking the ureter, or occlusion may result from the presence of a calculus or other mechanical or inflammatory cause. The consequent accumulation of pus causes distention of the kidney into a tender tumour which may be readily perceptible in the loin. Irregular high temperature, delirium, and rigours mark such severe cases. Unless relieved, the patient succumbs to septic poisoning. In a few instances spontaneous cure arises through inspissation of the pus, whereby masses are found resembling putty, in which a deposit of lime salts may take place.

A train of symptoms referable to the central nervous system, including dyspnœa and other manifestations, which may simulate uræmia, is sometimes encountered.

Diagnosis.—This is not always easy, but the occurrence of chills and a rise of temperature in the course of the infectious diseases, with a close watch on the urine, should usually enlighten the practitioner. The differentiation between the calculous and tuberculous forms may often be made with accuracy by means of the Roentgen ray, and catheterization of the ureter is of great value for diagnosis as well as treatment. While tubercle bacilli may occasionally be demonstrated, the microscopical examination of the urine is more often negative than otherwise. Suppurative cystitis usually differs from pyelitis in the tenesmus and vesical pain and the alkaline condition of the urine ((18), page 711), the urine of pyelitis being more often acid, though cases may occur, especially in men, in which both conditions are present and differentiation is impossible. Lumbar pain and tenderness over the kidney are suggestive of pyelitis, although the symptoms are in some cases more vesical than renal.

Perinephritic abscess differs from pyonephrosis in that the pus does not escape through the urine, and œdema in the skin over the lumbar region and fluctuation may often be made out.

Prognosis.—Mild cases, especially those of febrile origin, usually resolve spontaneously. The suppurative varieties may occasionally be self-limiting, but more often are progressive and demand surgical relief. It must always be borne in mind that the condition may be bilateral, while it not infrequently happens that anomalies exist and death may result from removal of the only kidney.

VIII. **Hydronephrosis.**—*Causes.*—Dilatation of the kidney, its pelvis and calyces, may be unilateral, rarely bilateral; congenital, or due to obstruction of the ureter by kinking, valves, cicatricial bands, or calculi; may be continuous or intermittent, and present in varying degrees.

Symptoms.—It often exists for years without attracting attention, and at most simply presents a progressively enlarging tumour in the region of the kidney which may cause some dragging pain. When large, it may cause pressure symptoms. Bilateral cases are almost always congenital, and cause death from uræmia in a few days at most. The symptoms of the intermittent variety are quite distinctive. A large tumour is found, corresponding in position to the kidney, which suddenly disappears with the simultaneous passage of large quantities of clear urine. Such hydronephroses tend to reaccumulate, and the process may be repeated indefinitely.

Differential Diagnosis.—In young children sarcoma of the kidney and enlarged retroperitoneal glands closely simulate hydronephrosis, and exploration alone may demonstrate the true nature of the growth. Ovarian cystoma may be mistaken for hydronephrosis. Vaginal examination should help to make the difference plain; the ovarian tumour is more freely movable, fills the lower portion of the abdomen, and tends to push the intestines upward, while the ascending colon can often be made out passing over the hydronephrotic sac. From pyonephrosis the diagnosis may be made by the absence of constitutional disturbance. Rare instances occur of hydronephrosis so large as to be taken for ascites, while the combination of a movable kidney and hydronephrosis occurs. Should other means fail, puncture and the examination of the aspirated fluid ((10), page 717) may be of service, although (to be remembered) the characteristics of urine tend to disappear in collections of long standing.

Prognosis.—A majority of cases never cause trouble. A certain proportion are cured by spontaneous evacuation. In some instances, however, the growth of the tumour is such as to cause serious inconvenience, requiring surgical relief. A few cases of intra-abdominal rupture, or even evacuation through the diaphragm and lung, have been reported. Infection and transformation into pyonephrosis may happen.

IX. **Nephrolithiasis.**—Renal calculus may vary from a mere "infarct," so called, to a mass occupying the entire pelvis. The symptoms necessarily depend upon the size of the stone. The simple infarct gives no symptoms. Fine "gravel" may be passed by the patient for years and give no other symptom. The size of renal calculi passed *per urethram*, without discomfort, may be considerable. Such stones may appear but once, or may be passed at intervals for years. See *also* (10), page 711.

Symptoms.—Many patients, however, are not so fortunate, and attacks of *renal colic* result. The patient is suddenly seized with pain of an agonizing character, having its origin in the lumbar region, either anteriorly or posteriorly, and following along the course of the ureter. It is felt also in the testicle and down the inner side of the thigh, and is at times referred to the glans penis. Such an attack may last only a few minutes, ceasing as the stone enters the bladder, or it may last for hours, inducing nausea and vomiting, sweating, and even syncope and collapse. A chill may accompany the onset, the temperature is regularly elevated, and the pulse becomes rapid and feeble. There is often strangury. Some patients during an attack void large quantities of clear urine. It not infrequently happens that suppression, and even uræmia, supervene, although the opposite kidney is perfectly normal. Following the attack there is a period of prostration. Aching pain may persist in the region of the affected kidney for a considerable time after the stone has passed. It is rarely possible to demonstrate the condition by physical examination.

In the case of calculus too large to be voided by the ureter, its continued presence in the pelvis of the kidney may give rise to pain, usually a dull, boring backache, not always referred to the kidney, occasionally paroxysmal, and in certain cases simulating floating kidney; to hæmaturia, by no means a constant symptom, sometimes causing a smoky appearance, often discoverable only by the microscope, and frequently aggravated by exercise; to pyelitis either simple or purulent; and to pyuria. Non-suppurative pyelitis due to the irritation of a calculus is often recurrent, the backache, chill, fever, and high-coloured urine closely simulating malaria.

Differential Diagnosis.—Renal colic may closely resemble biliary colic (when occurring on the right side), intestinal colic, floating kidney, and sometimes vesical calculus. The direction and localization of the pain, which in renal colic is referred to the testicle, the latter becoming tender and retracted, the character of the urine, and the previous history, should distinguish this condition from the hepatic and intestinal varieties, while ammoniacal urine points to vesical calculus, and physical examination will often establish the diagnosis of floating kidney. In 80 cases with symptoms of calculus —58 typical, 22 indefinite—calculi were found, by X-ray, in but 23 (EASTMOND). It is therefore most probable that a positive diagnosis can be made only by the X-ray and a *competent* operator.

Prognosis.—Renal calculus may be present for years without danger. Repeated attacks of pain or the occurrence of pyelitis call for surgery.

X. **Tumours of the Kidney.**—*Symptoms.*—If benign, the growth may not be recognised until it has attained such a size as to cause discomfort by reason of its weight and the pressure exerted on surrounding organs, although it is rare to find non-malignant growths of large size. Malignant growths, on the other hand, forming a very large majority of kidney tumours, frequently attain enormous dimensions. This is especially the case with sarcoma, the form appearing most often in young children, in whom the tumour mass may occupy and distend the entire abdomen. The symptoms of malignant tumour of the kidney include hæmaturia, pain, and emaciation. The blood is frequently voided as clots, which often show as moulds of the ureter or even of the pelvis of the kidney, or it may be passed in the fluid state. It is claimed that detached portions of the growth may be detected in the urine. Pain is not always present, but the patient may complain of dull, aching, lumbar pain, sometimes radiating along the course and distribution of the genito-crural nerve. As in cancer elsewhere, there is progressive emaciation and loss of strength. In considering the question of the probable nature of the tumour, it must be remembered that cancer is most frequent before the 3d and after the 40th year.

When sufficiently large to be palpable, examination reveals a deep-seated mass in the lumbar region, which may be movable, may present a lobulated surface, and across which the colon may be shown to pass (Fig. 185, page 509). It does not move with respiration.

Differential Diagnosis.—From neoplasm of the retroperitoneal glands; this is more centrally placed and is quite immovable. The distinction may be impossible to make.

From enlargement of the spleen; the characteristic outline of the spleen should prevent confusion. It also moves with respiration.

From tumour of the gall bladder; the latter is more superficially placed, and jaundice is a prominent symptom.

From pedunculated fibroid and ovarian cystoma; the movability of these tumours is lateral and downward, and their pelvic origin is generally demonstrable.

Prognosis.—Benign neoplasms rarely cause trouble. The outlook in cancer is of the gravest. If early diagnosis can be made, extirpation affords the only chance of recovery. The percentage of successful cases is, however, small, as recurrences are frequent.

XI. **Cystic Disease of the Kidney.**—Of pathological rather than clinical interest, as the condition can rarely be diagnosed.

Four varieties occur: multiple small retention cysts incident to chronic nephritis; single larger cysts, probably of the same origin; combined cystic disease involving the liver and spleen in addition to the kidneys; and the congenital variety. While most of the latter cases die antepartum, or shortly after delivery, a few survive for years, the real condition being discovered post mortem. The cystic kidney is here bilateral, much distended, and presents a fluctuating tumour in both flanks. There is cardiac hypertrophy with high-tension pulse, and death may occur with the symptoms of chronic interstitial nephritis. The condition may simulate hydronephrosis, though the latter in its acquired form is rarely bilateral.

XII. **Perinephritic Abscess.**—*Causes.*—This disease has already been mentioned under pyelitis (page 1024). It may occur primarily as a result of traumatism; or secondarily as an extension from pyelitis, appendicitis, spinal caries, and empyema; or follow the acute febrile diseases; and a few cases seem to result from invasion by the common colon bacillus without traumatism.

Symptoms.—Following one of the conditions just mentioned, a chill, fresh rise of temperature, sweating, and deep-seated lumbar pain radiating into the thigh and testicle, with, later, the finding of a tender, fluctuating mass in the space between the last rib and the crest of the ilium, and œdema of the skin, point to perinephritic abscess. If the collection of pus lies anteriorly the patient is more apt to lie with the thigh flexed, and to complain of pain radiating into and about the hip-joint and the testis. Bending of the trunk toward the affected side is said to be a valuable diagnostic sign. The urine is clear unless a pyelitis coexists. The condition, instead of beginning abruptly, may have an insidious onset and a prolonged course with very moderate constitutional symptoms.

Differential Diagnosis.—From renal calculus, by the presence of constitutional disturbance and by physical examination. From appendicitis, by the history and position of the pain and swelling, which also holds for empyema of the gall bladder. From psoas abscess; the swelling and pain here are anterior to the anterior axillary line, and the pus tends to point in the groin. From pyelitis and pyonephrosis the diagnosis is sometimes difficult, as they may coexist. The presence of an indefinite fluctuating mass points to abscess. In doubtful cases the use of an aspirating needle would be justifiable. From spinal and hip-joint disease, by the characteristic deformities and limitation of motion. From lumbago, by the fever, tenderness and swelling, and lateral inclination.

Prognosis.—This is generally good provided free drainage is established. Some few cases of violent infection in patients already enfeebled by old kidney trouble die in spite of all treatment.

SECTION VII

DISEASES OF THE NERVOUS SYSTEM

(See also pages 517 to 595)

PREPARED BY SMITH ELY JELLIFFE, M.D., AND A. B. BONAR, M.D.

I. THE PSYCHONEUROSES: DISEASES OF UNDETERMINED PATHOGENY

A. SENSORI-MOTOR NEUROSES

I. **Epilepsy.**—*Causes.*—Epileptic attacks or epilepsy is a symptom, not a single disease. It may occur at any period of life. Infancy, puberty, the puerperium, and senility are periods of greatest frequency. The predisposing causes are: Injuries at birth, heredity, alcoholism, syphilis, the intermarriage of neurotic persons, and powerful emotions during pregnancy. Exciting causes are: Fright, injuries to the head, rickets at time of dentition, masturbation, sunstroke, syphilis, alcoholism, and infectious diseases, especially scarlet fever. Reflex causes (so-called) are worms, dyspepsia, lesions involving the peripheral nerves, and ocular, auditory, and dental irritations.

Symptoms.—In the *grande mal,* or major attacks, the patient may for a few hours, or perhaps a day before an attack, suffer from general malaise, vertigo, or irritability. For a description of the attack see page 80. There may be a temporary exhaustive paralysis immediately following the attack, with loss of knee-jerk. The dilatation of the pupils subsides and they often oscillate. Slight transient glycosuria or albuminuria may be present. The urea is not increased, but the earthy phosphates are. One attack may be followed by others, hour after hour—the *status epilepticus.* This condition usually lasts for less than 12 hours, but may last for 1 or more days.

Minor attacks (*petit mal*) may occur in which the patient suddenly stops whatever he may be doing, the features become fixed, the face is pale, the eyes are open and the pupils dilated, there is slight twitching of the muscles of the face or limbs, and a momentary loss of consciousness. The attack lasts only for a few seconds, and the patient may immediately continue his work or conversation from the point at which it was interrupted. He does not fall, and has amnesia for what has occurred but knows that he has had an attack. Occasionally there are forced movements, in which the patient turns around a few times, or runs, or takes a few steps in a confused automatic manner.

Psychical epilepsy may consist of sudden, violent automatic movements, or of sudden exhibitions of maniacal excitement following or replacing a minor attack. While in this condition crimes of violence may be committed. Rarely patients may go into a state of somnambulism, in which they perform automatically acts to which they are accustomed. This has been called somnambulic epilepsy, and when it occurs without a preceding minor attack it is by some considered a "psychical epileptic equivalent."

The aura may be a feeling of prickling or numbness which begins in the hand and goes upward until it reaches the head, when the patient becomes unconscious. A sensation of something passing from the epigastrium upward toward the throat is a common aura. Psychical auræ are not infrequent. (See also page 80.)

The most frequent form of epilepsy is that with severe attacks; next a combination of severe and minor attacks; then minor attacks alone; and, least frequent, the psychical form. The attacks may occur from once or twice a year to a number of times a day. Minor attacks may occur oftener, generally every day. It appears that attacks occur mainly in the waking hours. In most cases of epilepsy there is a gradual mental deterioration, which may be very slight. There is irritability of temper, inability to fix the attention or carry out a purpose, selfishness, and a weak memory. A lack of moral sense and vicious impulses may appear in children with epilepsy. Epileptics are not of robust constitution, are undersized, and for the most part present some stigmata of duration (page 541).

Diagnosis.—Epilepsy must be distinguished from hysteria and various symptomatic and toxic convulsions. The cardinal points are: The aura, the cry, the tonic convulsion, the sudden loss of consciousness, the biting of the tongue, the dilated pupils, and the emptying of the bladder. Hysterical patients do not, as a rule, bite the tongue or hurt themselves when they fall, and their movements are more co-ordinate. Especial pains should be taken to diagnose true epilepsy from many of the reflex epileptic phenomena (vertigoes, etc.) which occur in "nervous children." These cases are frequently reported as being cured by trifling surgical procedures, such as circumcision, cutting of eye muscles, *et al.* Jacksonian epilepsies are usually due to circumscribed local growths.

Prognosis.—A small number of epileptics recover. Dementia or insanity develops in about ten per cent, and these are incurable. In general life is somewhat shortened by epilepsy. Death may occur as the result of the ***status epilepticus.***

II. **Vertigo.**—See page 66.

III. **Migraine, Hemicrania** (*Sick Headache*).—This is best regarded as a constitutional disease. Begins usually at puberty, sometimes as early as the 2d year, and is most frequent in females.

It is occasionally hereditary in neurotic families with a history of gout, rheumatism, epilepsy, or neuralgic disorders. Overwork is an occasional cause in children. Autotoxæmia from unknown acids and intestinal poisons, errors in refraction, overwork, shock, injury, and exhausting disease are thought to be causative factors.

Symptoms.—There may be premonitory symptoms, a feeling of depression and general malaise, for a few hours or a day. The attack usually begins during the morning, in the forehead or occiput, on one side of the head, increasing in severity and extent until the whole head is affected. The pain is tense, throbbing, blinding, and increased by jars, noises, and light. Frequently there is dimness and restriction of the visual field, sometimes hemianopia, and flashes of light, or light or dark spots dancing before the eyes. Nausea and vomiting are usually present. Not infrequently a feeling of stupor, confusion of ideas, disturbances of memory, vertigo, and tinnitus aurium are present. These headaches are often called "bilious headaches" from the character of the vomited matter. Liver or gastric disturbances do not cause all migraines. The pulse is small, hard, and perhaps slow, and the patient's face is usually pale, rarely flushed. The length of an attack varies from 6 hours to 2 or 3 days, lasting, as a rule, from 6 to 24 hours. When the pain becomes less severe the patient goes to sleep, and usually awakes the next morning feeling well. The attacks occur periodically—weekly, fortnightly, or monthly—and, in women, especially at the time of the menstrual period. The severity and frequency of the attacks lessen at or about the time of the menopause, and generally then disappear altogether.

Rheumatism, anæmia, or dyspepsia may coexist, and neuralgia be added to the ordinary pain of the disease. When the pains are neuralgic there are usually no visual or aural disorders, as in migraine.

Diagnosis.—The cardinal points are: heredity; periodical character of the attacks, and their location; the visual and aural symptoms; and the nausea and vomiting. Renal or organic brain disease or neuralgia may coexist with migraine. Multitudinous abortive and mixed types occur. Some of these may be designated as *migrainoid* states. The *prognosis* for cure is not good.

IV. **Hysteria.**—*Causes.*—Occurs most frequently between the ages of 15 and 25 in females; at a somewhat later age in males; in children, between 11 and 15, perhaps as early as 8, years of age. Females are affected much oftener than men. The chief predisposing cause is heredity, with a history of the disease, or of some psychosis or neurosis in the parents. Exciting causes are: powerful emotions (especially fear), anxiety, excitement, worry, injuries accompanied by mental shock, imitation, and excesses. The hysterical reaction is a general indication of lessened mental resistance. It is not a disease *per se*.

Symptoms.—In **hysteria minor** the patient is very nervous, with hyperæsthesias, pains, and crises of an emotional character. Girls and young women are the usual subjects of hysteria minor. The patient becomes extremely sensitive, excitable, mentally depressed, and there is a marked loss of emotional control. She yields to impulses, and cries and laughs very easily. There may be spinal pains, and severe and chronic vertical headaches. When excited there may be sensations of tickling, fulness, or choking in the throat—the "globus hystericus"—and occasionally fleeting attacks of chilliness and trembling. More rarely there are vasomotor disturbances, resulting in cold extremities and flushings. Sleep is usually more or less disturbed. Attacks of headache or vomiting or of great mental excitement may occur. There may be somnambulism or, under excitement, attacks of cerebral automatism, in which she may perform acts of which she will be entirely ignorant after the seizure. After the crises there is usually a large quantity of light-coloured urine passed. In hysteria minor there are, as a rule, no anæsthesias, paralyses, or decided convulsions.

Between the crises of **hysteria major** the patient may be well, but generally there are characteristic *paralyses, contractures*, and *sensory disturbances*. Frequently there are anæsthesia and hyperæsthesia of the skin and mucous membranes, and anæsthetic disorders of the special senses. Anæsthesia of the skin occurs as an hemianæsthesia (most common), or a segmental or disseminated anæsthesia (Fig. 221, page 568). The pain sense is chiefly affected with a lesser impairment of the tactile and thermal sensibilities. The skin reflexes are very frequently absent. With hemianæsthesia there is usually some hemiplegia and occasional tremor, and in segmental anæsthesia often some paralysis of the part. There is often an anæsthetic condition of the retina, causing a concentric limitation of the visual field and a disturbance in the colour sense. Vision may be impaired in both eyes, or completely lost in one eye, the latter especially with hemianæsthesia, and on the same side the senses of hearing and smell may be impaired. The sense of taste is impaired or abolished, perhaps only on the back part of the tongue and palate.

Hyperæsthesia is found in small patches; in women, most commonly over the ovaries; in men, over the corresponding regions and on the scrotum. They are also found below the breasts, along the spine and in the epigastrium, and are sensitive to pressure, which causes various kinds of paroxysms—hysterogenic zones (Fig. 220).

Headaches, chiefly vertical or parietal, are frequent, and the pain is of a sharp, boring kind, at times very severe. Facial and intercostal neuralgias, migraine, and, more often, pains along the spine occur. There may be attacks of pseudo-angina.

Choreic and ataxic movements, tremor, contractures, amyosthenia, and paralyses occur in hysteria major. The paralyses are usually hemiplegias, paraplegias, and monoplegias. The onset of hysterical hemiplegia is sudden, and the left side is more often affected. The face usually escapes and the paralysis is not complete. When walking, the patient drags the affected leg after him, instead of swinging it around in a half circle as in organic hemiplegia. The knee-jerk is usually not exaggerated, and may even be absent for a time. The monoplegias rarely affect the face, usually an arm or leg, or the muscles of the larynx or of the eye. Electrical reactions are normal, and atrophy, if present, is slight. The adductors are involved in hysterical paralysis of the larynx, and the patient is unable to speak aloud (hysterical aphonia, usually sudden). Hysterical paraplegia is frequent, often with much pain in the back—the condition of "spinal concussion." The knee-jerks may be normal or increased, rarely lost. There may be a short or false clonus, and the sphincters are not involved. *Amyosthenia* is frequent, the patient's legs suddenly becoming weak or paralyzed, or the arm giving out when lifting some object. This symptom usually precedes a paralysis.

In some cases of hysteria contractures of the muscles may occur as the result of slight mechanical irritation. These may be temporary, or may last for a long time. The legs, arms, and facial muscles are most affected. Tremor of all varieties may be present.

In hysteria there is marked emotional instability and craving for sympathy, lack of self-control, and weakness of the will. The mood constantly changes; there is increased sensitiveness, and exaggeration of the Ego. There is a limitation of the field of consciousness as there is of the field of vision. The hysterical person can think of nothing but her personal feelings. There is an undercurrent of suggestibility in the mental state, through which ideas become fixed and patients become self-centered, and believe they have various ailments which have no objective existence.

Constipation, dyspepsia, anorexia, and sometimes regurgitation of food, or vomiting, occur. The specific gravity of the urine is often low, especially that of the large amount of light-coloured urine which is always passed after hysterical attacks. There may be retention of urine. The vasomotor symptoms consist of flushings and pallor, and sometimes coldness and œdema of the extremities. The œdema may be ordinary, or have a peculiar bluish tint ("blue œdema"), and not pit on pressure. The hands are usually affected, and their surface temperature is somewhat below normal. The condition resembles Raynaud's disease, but gangrene never occurs. Anæmia is usually present, and there may be irregular fever.

The crises of hysteria major are emotional, convulsive, neuralgic, or gastric; paroxysms of coughing, hiccoughing, or sneezing; or attacks of catalepsy, amnesia, cerebral automatism, trance, and lethargy. The first group are the most common. The emotional crises are similar to those which occur in hysteria minor. The "globus hystericus" is almost always present.

Two forms of convulsions in hysteria major are described: the *ordinary form*, which also occurs in hysteria minor, and the hysteroid attack, or *hystero-epilepsy*. In the former the patient suddenly falls, and various irregular movements of the body occur. In a more severe attack the hands, arms, and fingers are flexed and the legs and feet extended. Usually the eyes are closed. The pupils are dilated, and frequently there is convergence, or perhaps irregular movements, of the eyeballs. Sensation may be diminished over the body and on the conjunctivæ. The patient rarely bites her tongue or injures herself in any way, although she may bite her lips. She may scream or make noises during the attack. The attack may last from a half hour to several hours. The convulsion may consist simply of a chill-like tremor; in other cases simply a slight rigidity of the body, or a series of rhythmical movements of the limbs or trunk. Opisthotonus may be the chief manifestation; in others the patient falls and lies unconscious, as if sleeping, for some minutes, or it may be an hour. Marked mental excitement may accompany or follow the attack. During the attacks there is usually a consciousness of what is going on, and if spoken to sternly the patient will often respond. Pressure upon a hysterogenic zone may cause the attack to cease, especially in women.

Hystero-epilepsy is not epilepsy, but is a true hysteria. In this form of hysterical attack there is a prodromal stage; an epileptoid stage, lasting from 1 to 3 minutes; a stage of contortions and grand movements lasting 1 to 3 minutes; an emotional stage, lasting from 5 to 15 minutes; and, at times, a stage of delirium.

Diagnosis.—The cardinal points are: The hysterical temperament of the patient, the history of previous hysterical crises, the presence of some of the various stigmata of hysteria, the transient and varying character of the paralyses, anæsthesias, and other stigmata, and the condition of the deep reflexes. See also (2), page 81.

Prognosis.—Favourable in children. In hysteria minor the prognosis depends upon the severity of the disease. In hysteria major and hysteria associated with organic disease, the prognosis is very unfavourable. Hysterical attacks are usually curable. The tendency to recurrence is strong.

V. **Neurasthenia.**—*Causes.*—Occurs most frequently between the ages of 20 and 50, but may occur between the ages of 10 and 20, and also between 50 and 70. Men and women are equally affected. The disease is found more frequently in cities, and among the educated classes. The chief predisposing cause is the inheritance of a neuropathic tendency. The exciting causes are: severe shocks, with or without injury, as many cases of so-called "traumatic neuroses" are simply neurasthenia; overwork and worry; excessive use of stimulants and narcotics; sexual excesses; dietetic imprudences; and the infections of syphilis, malaria, typhoid, influenza, or other debilitating diseases.

Symptoms.—These are mainly subjective. The initial symptoms are usually headache or mental depression. The headache is more commonly occipital or diffuse in character and is chronic and persistent. It begins in the morning and lasts all day, but does not keep the patient awake at night. There may be a feeling of general mental depression, inability to concentrate the mind upon work, mental confusion, marked irritability, excessive sensitiveness and morbid reserve, or undefinable fears. Sleep is not good, and there may be persistent insomnia, or the sleep is not refreshing. There are various paræsthesiæ of the head, mainly feelings of constriction, tenderness, burning, or pressure. Paræsthesias of the hands and limbs occur, and there may be pain or a feeling of weakness in the back of the neck and along the spine. Slight but frequent vertigo is occasionally present, and there may be buzzings in the ears and head and spots before the eyes.

There is a general muscular weakness in a large proportion of the cases. The patient may not appear weak, but he tires quickly. There is often a tremor of the hands, tongue, lips, and eyelids, and sometimes twitching of the muscles of the face and tongue. The deep reflexes are usually much exaggerated, and the skin reflexes increased. The condition of the reflexes, however, varies greatly. The patient's eyes tire easily, although his vision is good. Astigmatism, hypermetropia, and asthenopias are frequent. The field of vision is not contracted. The pupils are frequently dilated, and in some cases are sluggish to light. Occasionally there is excessive mobility of the iris, inequality of the pupils; and hyperacusis or dysacusis. The sexual function is irritable and weak. Nocturnal emissions are frequent, and there may be partial or complete impotence. The pulse is often markedly accelerated on slight exertion.

Symptoms due to vasomotor disturbance are cold feet and hands, palpitation of the heart, hot flashes, pseudo-angina, dermographic skin, fulness and noises in the head, vertigo, a fluttering feeling in the abdomen, and paroxysms of profuse perspiration. In neurasthenics there is much stomach and intestinal indigestion and constipation. A "mucous enteritis" sometimes occurs. At times a temporary albuminuria or a transient glycosuria may be found.

Diagnosis.—The disease must be distinguished from hypochondriasis, melancholia, the initial stage of general paresis, hysteria (major and minor), simulation, and the effects of some other constitutional disease. The border line between neurasthenia and most of these conditions is not very distinct in the initial stages.

The following are some of the different forms of neurasthenia: Hystero-neurasthenia, traumatic neurasthenia, primary neurasthenia, acquired neurasthenia and lithæmia, climacteric neurasthenia, spinal irritation, neurasthenia with fixed ideas or anxiety psychosis, neurasthenia gravis, and angiopathic neurasthenia.

Prognosis.—The disease is usually chronic, and runs a course of from 1 to 8 or 9 years. Recovery is frequent, so also are relapses.

VI. **Traumatic Neuroses.**—*Causes.*—Trauma may be the exciting cause in the following conditions: (1) The nervous condition following railway and other injuries, especially when associated with fright. (2) Neurasthenia.—"Traumatic neurasthenia" or "neurosis," or "railway spine," does not differ from neurasthenia due to other causes except that coincident surgical troubles may be present. It is described under neurasthenia. (3) Hysteria.—"Traumatic hysteria" does not differ from hysteria due to other causes except by the presence of surgical troubles, and in its sudden appearance. It usually takes the form of hysteria major. (4) Trauma may also produce minute multiple hemorrhages throughout the nervous centres. The resulting organic symptoms are added to the hysterical and neurasthenic symptoms usually present.

Symptoms.—These are headache and vertigo, with lessened power of application, depression, irritability, and hypochondriasis. The sight is disturbed, and there may be a contracted field of vision and sometimes optic atrophy. Tremor, inco-ordination, numbness, prickling, and anæsthesia, not limited to one half of the body, may be present. The reflexes are apt to be exaggerated. The muscular movements are slow and weak. Control of the bladder may be impaired, and there may be some stiffness and pain in the back. The condition may resemble that of multiple sclerosis. Traumatism may be a contributory cause of tabes, insanity, and inebriety in those predisposed to these diseases, or of a cerebral tumour. The shock and the mental impression are more powerful factors in the production of functional neuroses than the physical injury.

Diagnosis.—Malingering must be guarded against. The essentials are the history of trauma and the points already given under the headings of neurasthenia and hysteria.

B. MOTOR NEUROSES

I. **Chorea** (*St. Vitus's Dance, Sydenham's Chorea*).—*Causes.*—Occurs most frequently between the ages of 5 and 15, but may appear after 20 or even in old age. Girls are affected more often than boys. It occurs with the greatest frequency in the spring, next in the autumn, winter, and summer. There is heredity in some. The exciting causes are mental worry, rheumatism, fright, and injury (seldom). Endocarditis frequently develops. Other causes are pregnancy; infectious fevers; reflex irritation from nasal disease, or sexual disorders; overstudy; and intestinal irritation from worms. Anæmia and malnutrition are frequent predisposing causes, and malaria may be a factor. The majority of ordinary choreas are dependent on antecedent infectious diseases.

Symptoms.—The onset is usually slow, lasting 1 or 2 weeks, but may be sudden. There are, at first, irregular twitchings of the hand of one side, and the patient may drop his knife or fork. Winking, grimacing, and twitching of the facial muscles become manifest. After a time the child's foot and leg are involved and he may stumble in walking. In a week or so the other side of the body is also affected, usually not to the same extent. The disease reaches its height in from 3 to 4 weeks. At this time the movements are nearly continuous, the child can hardly use his hands, and walking is difficult. The speech is indistinct, confused, and difficult, and the muscles of respiration may be affected. Usually the movements continue whether the muscles are at rest or acting. In some cases they are present only when the limbs are at rest, in others chiefly when volitional acts are attempted. During sleep the twitching generally stops. In severe cases there may be attacks of mental excitement or delirium. The most common mental impairment is irritability of temper and some dulness of intellect.

The general physical condition is impaired, there are anorexia, constipation, loss of flesh, and anæmia. Excitement or exertion increases the movements. The reflexes are diminished and the knee-jerk may be absent. There is no real paralysis, although the muscles are weak. Their electrical irritability is somewhat increased.

In *maniacal chorea* there is delirium with delusions and hallucinations. The mania after a week or so is succeeded by a condition of apathy and dulness. This form usually occurs in adult females and is serious. In *paralytic chorea* (usually in children) one arm suddenly becomes weak and powerless, perhaps with slight twitchings. In *chorea of adult life* and in *senile chorea* men are more often affected than women, and it is apt to become chronic.

The duration of chorea may be from 6 weeks to 6 months, but usually it lasts from 10 to 12 weeks. The disease may continue for years, each improvement being followed by a relapse. Relapses occur in nearly one half of the cases.

Diagnosis.—The peculiar twitching movements are characteristic. The disease must be distinguished from hysterical spasms, which include myoclonus, saltatory chorea, convulsive tic, and chorea major.

Prognosis.—Nearly all cases recover. In very few instances the disease in adults has a fatal termination.

II. **Huntington's (Hereditary) Chorea.**—*Causes.*—Occurs with about equal frequency in males and females, and usually between the ages of 30 and 50 years. The disease is always directly hereditary.

Symptoms.—Twitchings in the face are first noticed, then in the arms and legs. There is progressive mental impairment, a tendency to melancholy, and finally dementia. The disease is chronic, usually lasting from 10 to 20 years, and the *prognosis* is bad.

III. **Habit Spasm** (or **Tics**).—A condition characterized by the occurrence of co-ordinate movements. In different cases this movement may be a gesture, or a shrug of the shoulders, or a winking of the eyes, or a sniff, or some peculiar grimace. These movements are often seen in children, and may be the remains of an attack of true chorea, or may be a chronic convulsive tic from the beginning.

IV. **Saltatory Spasm.**—Occurs in both sexes and at all ages. The patients are hysterical or neurasthenic. The exciting cause is the weight of the body upon the feet. Violent contractions of the calf and hip muscles occur the instant the feet touch the floor, but all the muscles may be involved. The peculiar muscular contractions cause the patient to jump, and may be so violent as to throw him to the ground. The disorder is probably an hysterical spasm.

V. **Facial Spasm** (*Mimic Tic*).—Intermittent, involuntary twitchings of the facial muscles. Always chronic and usually unilateral. Occurs in middle and later life, more often in women. Predisposing cause is a neuropathic constitution. Exciting causes are injury, shock, anxiety, and exposure. Occasionally reflex, from irritation of the cervico-brachial nerves or some branch of the trigeminus. Some organic disease may cause a symptomatic tic, e. g., post-hemiplegic. *Symptoms.*—Onset slow. The orbicularis muscle and zygomatici are the first affected. The spasm is clonic, the muscles of the face being affected by a series of quick twitches, with intervals of rest. Occasionally the spasm is tonic and lasts for several seconds. There is no pain, paralysis, or atrophy, and no trophic or secretory symptoms. The spasm may become bilateral after a time. *Diagnosis.*—It is distinguished from organic spasm by the fact that in the latter there are always other symptoms, and it is usually unilateral, while habit chorea is bilateral. There is generally unconsciousness in spasm from cortical disease. The spasm is tonic when it follows hemiplegia, and in hysterical facial spasm. *Prognosis.*—Generally incurable. The outcome is better if the disease is due to a reflex cause.

VI. **Torticollis** (*Wryneck*).—A clonic or tonic spasm of the muscles supplied by the spinal accessory, sometimes of other muscles of the neck. (1) Congenital Wryneck.—Caused by injury to the sterno-cleido-mastoid during delivery, or due to intra-uterine atrophy. It most often follows foot or breech presentations. There is no spasm, but the neck is drawn to one side because of the shortness of the muscle. (2) Symptomatic Wryneck.—Appears usually as a symptom of rheumatic myositis, sometimes of tumours, adenitis, abscesses, or local syphilis. Occurs principally in children. Tenderness and pain are always present. (3) Spurious Wryneck.—An apparent or real spasm of the muscles of the neck. Usual cause is caries of the spine. (4) Spasmodic Wryneck.—This condition is purely nervous. There is spasm of the muscles supplied by the spinal accessory; sometimes of those innervated by the upper cervical nerves.

Causes.—It occurs in women more often than in men, and is a disease of early and middle adult life. Heredity and a neuropathic constitution are predisposing factors.

Symptoms.—There are primarily slight feelings of discomfort or pain in the neck, soon followed by spasm, which is at first clonic and intermittent. The sternomastoid and the upper fibres of the trapezius are oftenest affected. The head is inclined toward the affected side, the chin raised, and the head rotated to the opposite side. The superior obliquus and complexus are more rarely involved. The muscles of the opposite side may be affected, most commonly the splenius. The disease may start in one muscle and gradually involve others. The spasm usually becomes more and more constant, until finally it may be tonic. The pain gradually disappears. The unused muscles undergo atrophy, while the affected muscles hypertrophy. Facial asymmetry may or may not occur.

Prognosis.—Not fatal, rarely cured, but sometimes much improved. Usually reaches a certain stage and remains chronic.

VII. **Spasmus Nutans** (*Nodding Spasm*).— A disorder characterized by rhythmical nodding or oscillatory movements of the head.

Causes.—Occurs chiefly in poorly nourished and anæmic children. It may be due to digestive disorders, gross disease of the brain, basilar meningitis, or dentition. Is sometimes a habit chorea.

Symptoms.—Onset usually sudden. The attacks, sometimes rather violent, last for a few minutes, perhaps hours, or are constant, except during sleep. The movements of the head may be from 30 to 60 a minute, or more or less. The eyes and facial muscles may be affected. An epileptic attack may follow a paroxysm. The *diagnosis* is easy, and the *prognosis* depends upon the cause.

VIII. **Spasmodic Tic with Coprolalia** (*Gilles de la Tourette's Disease*).—Occurs in neurotic children, chiefly boys 6 to 16 years of age, with a neuropathic family history.

Symptoms.—There are attacks of irregular, perhaps violent, movements, involving at first the upper extremities, head, and face, later the whole body. The spasms can be sometimes controlled by the will, but are usually more severe afterward. They stop during sleep. After a while the patient makes inarticulate cries during the attacks, or he repeats words that he hears, or utters obscene or profane words or expressions. These utterances are made suddenly and automatically, accompanied by grimaces and contortions of the facial muscles. The disease is chronic and may last for years.

IX. **Paralysis Agitans** (*Parkinson's Disease, Shaking Palsy*).—*Causes.*—Occurs between the ages of 40 and 70, most frequently between 50 and 60. Less commonly it may occur in early life after puberty. Affects males more often than females. Predisposing causes are anxiety, prolonged overwork, and, rarely, hereditary influences. Syphilis, alcohol, and sexual excesses do not seem to enter into the etiology. The exciting causes are fright, acute mental suffering, exposure to cold and wet, injury, and, more rarely, sudden severe muscular strain, acute rheumatism, and fevers.

Symptoms.—There are at first aching pains in the arm, usually the left, and a tremor in the fingers of the same hand. The tremor gradually extends to the foot of the same side, then to the hand and foot of the other side. The face, tongue, and neck may be affected, but more rarely and only to a slight extent. There now develops a hypertonia of the muscles, usually in the flexors. This gradually brings about a characteristic attitude of the patient. The head and body are bent forward, the trunk is flexed on the thighs, the forearms on the arms, the fingers are straight, but are flexed as a whole on the metacarpus, and the knees are slightly flexed on the legs. He walks slowly with short and shuffling steps. It is difficult for him to rise from a chair, to start to walk, to stop, or to turn corners. There is often a feeling of propulsion or retropulsion, more rarely of lateropulsion. The voice is characteristically high-pitched, weak, and monotonous, with difficulty in beginning a sentence, but when started the patient talks rapidly.

There may be sensations of heat, cold, fatigue, nervousness, and restlessness. There are often aching pains, usually in the forearms or legs, more rarely neuralgic pains. Muscular weakness is marked, coming on early and slowly increasing. There are no paralyses. The deep reflexes may be exaggerated and there may be a clonus, but usually they are normal. Often there is profuse perspiration, and, as a rule, the face is red and flushed. The tremor increases in extent and the rigidity becomes more marked, until finally the patient becomes bedridden.

The *tremor* of paralysis agitans is peculiar in that it continues when the hand or limb is at rest, but ceases upon voluntary movements. In the hand, the tremor moves the fingers and thumb as a whole and they vibrate against each other (the "bread-crumbling" movement). Tremor of the head and face muscles occurs, but the trembling of the head usually seen is communicated from the body. The lips and neck muscles are sometimes the seat of tremor.

The *rigidity* begins early and increases until the patient is helpless. The flexors are chiefly affected. The muscular movements are slow. The muscles of the face are stiffened, and there is a peculiar expressionless, masklike appearance. The patient is usually emotional. There may be polyuria. The disease may affect one limb, or those of one side only, or rigidity may be the sole symptom.

Diagnosis.—The cardinal points are: the tremor, ceasing on voluntary movements, the rigidity of the back, neck, and limbs, the masklike face, monotonous voice, and the characteristic position of the hands. The disease must be distinguished from multiple sclerosis, senile tremor, posthemiplegic tremor, and wryneck affecting the extensors bilaterally. In multiple sclerosis the tremor is jerky and "intentional"; there are scanning speech, nystagmus and other eye troubles, and often apoplectiform attacks and paralyses. In senile tremor the head is affected first and most, and the patients are older. In posthemiplegic tremor there is a history of hemiplegia, and the increased reflexes, paralysis, and tremor are unilateral. The neck muscles and frontalis only are involved in retrocollis.

Prognosis.—Successful treatment as yet unknown. Chronic course.

X. **Occupation Neuroses.**—The most common, writer's cramp, occurs in men oftener than in women, most frequently between the ages of 25 and 40, seldom before the age of 20 or after 50. The predisposing causes are a neuropathic constitution, heredity, alcoholism, worry, and other weakening influences. The chief exciting cause is excessive writing, especially if done under a strain. Other causes are lead-poisoning, exposure to wet and cold, albuminuria, and local injuries.

Symptoms.—Onset usually slow. At first there is a slight stiffness in the fingers, or occasional jerky, uncertain movements of the pen when writing. This condition may last for months or years. The patient may be depressed and fearful of paralysis. Finally, writing becomes impossible. If the attempt is made there are spasmodic contractions of the fingers and even of the arm. Usually all other complex movements can be performed. The arm aches and occasionally is tender to pressure, but there is no active paralysis and no anæsthesia. Numbness and prickling are present in many.

In the *spastic* form of the disorder there are muscular cramps in half the cases. The muscles most affected are those of the thumb and first three fingers, the flexors in writers, the extensors in telegraphers. The forefinger, thumb, or little finger alone may be involved; frequently also the supinators and pronators. The spasm is usually tonic, and there is inco-ordination for writing movements. The *neuralgic* form often accompanies the spastic form, with the addition of severe pain and fatigue when writing. Rarely a trembling movement of the hand and arm develops on an attempt at writing (an "intention" tremor), and stops when the attempt ceases. In the *paralytic* form, also rare, an overpowering sense of weakness and fatigue develops in the fingers and hand as soon as the patient begins to write. The pen may drop from the fingers. The arm aches, and may become painful if the attempt is continued.

The patient may be emotional, nervous, and at times mentally depressed. There may be vertigo, insomnia, sensations of numbness, prickling, weight, pressure, and constriction, and pains and weariness, perhaps with some hyperæsthesia, and occasional headaches. When the nerves are involved there may be local sweating, dryness of the skin, cracking of the nails, or a passive congestion of hand and arm. In severe cases the condition of the fingers is suggestive of chilblains. The electrical reactions are variable. In the early stage the irritability is increased, in the later stages it is decreased, to both forms of current.

Diagnosis.—The history of excessive writing and the symptoms presented make the diagnosis simple. The *prognosis* is not favourable, although with prompt treatment recovery may result. The disease is chronic and progressive. If the left hand is used, that, too, is apt to become affected (three fourths of all cases).

Other *occupation neuroses* of similar character are musician's cramp, in which are included pianist's, violinist's, clarionet- and flute-player's cramp; telegrapher's cramp; sewing spasm, affecting tailors, seamstresses, and shoemakers; smith's spasm, affecting those making penknife blades, scissors, and the like; and driver's spasm, milker's spasm, and ballet-dancer's cramp.

Occupation neuroses also affect billiard players, hide dressers, dentists, painters, weavers, pedestrians, artificial flower makers, stampers, turners, type-writists, etc.

XI. **Tetany.**—*Causes.*—This disease, characterized by bilateral tonic spasms, intermittent or continuous, and affecting the extremities, is of various origin. The largest number of cases are found in connection with rickets, or debility due to chronic diarrhœa or lactation, or during pregnancy; also as a result of acute (especially typhoid) fevers. It may be a sequel of parathyroidectomy; and has occurred acutely in Europe as epidemic (or rheumatic) tetany. Gastric tetany, i. e., that associated with pyloric stenosis (usually due to ulcer, at times to malignant disease) with stasis and hyperchlorhydria is not uncommon. The disorder seems to bear some relation to the calcium metabolism of the body.

Symptoms.—The arms are folded, the elbows and wrists flexed, the thumb bent into the palm, the fingers close together and extended except at the metacarpo-phalangeal joint. In the lower extremities the toes are adducted and the feet extended. The muscles of the face, jaw, neck, and abdomen may be involved in the severer cases; so also those of the thorax, causing cyanosis from interference with the respiration.

During the attack the paroxysms may be produced (Trousseau's symptom) by pressure on the affected parts in the direction of their principal nerve trunks; or along the blood-vessels so as to hinder the circulation. The electrical and mechanical excitability of the motor nerves is greatly increased. A light blow over a nerve will cause active contraction of the muscles supplied by it (Chvostek's symptom). Pressure on a sensory nerve will induce paræsthesia over its peripheral origin. In acute cases the pulse may be rapid and fever present. Gastric tetany is preceded by prickling, numbness, and formication in the extremities; often also, in the severe cases, by intractable vomiting. The tetanic contractions are usually paroxysmal, and last for varying periods. The attacks continue from a few hours in the slighter cases to two weeks or longer in the aggravated forms, particularly gastric tetany, and may run for many years.

Diagnosis.—In tetanus a trauma precedes, and the contraction begins in the jaw-muscles; in tetany, in the extremities, and the attitude of the hands is quite different. Carpo-pedal spasms in rickety children, or in those with severe gastro-intestinal catarrh, are slight and transient, and may be considered as abortive tetany.

Prognosis.—The simpler forms recover. Severe gastric tetany is a very fatal disease, occurring mainly in cases where there is a grave mechanical obstruction to the passage of food from stomach to intestine. Under medical treatment the mortality reaches 88 per cent; under surgical measures (gastro-enterostomy) it is but 37.5 per cent.

XII. **Paramyoclonus Multiplex.**—A large group of borderland choreiform affections. In general defined as a motor neurosis choreic, or tic-like, in character, usually involving large muscular movements, notably of the trunk. The sharp, lightning-like contractions are usually symmetrical. The movements are not purposeful in the truer types of the disease. They usually disappear during sleep and during co-ordinated actions, but are rendered more energetic under emotional excitement. Nothing is known of the cause of certain types.

Symptoms.—At least two types stand out more prominently from the mass of hysterical and tic-like relatives, *viz.*, Friedreich's paramyoclonus and Unverricht's progressive paramyoclonus, with or without epilepsy as a complication. In the Friedreich type the movements involve more often the supinators, the biceps, and the quadriceps femoris. The muscles involved are not systematized groups, and both sides of the body are usually implicated, but the contractions in symmetrical groups may not be synchronous. There is hypertonus, but no electrical change. The contractions are rapid, usually occur in periodic attacks, with good days, or hours, and then recur.

In Unverricht's syndrome there is, as a rule, an hereditary element, although Unverricht's first cases did not show this, and the attacks are usually complicated with epileptic symptoms. In his patients the muscular movements partook of the same lightning-like character, were non-synchronous, and frequently individual muscle-bundles in a muscle would show the signs of non-volitional irritability of some type. Good and bad days were also apparent; the entire musculature might be affected, even the diaphragm, causing grunting; and on severe days epileptic attacks would supervene. The disease may appear as early as 10 years or as late as 70 years.

Diagnosis.—Undoubtedly many conditions of motor anomalies may be grouped under these heads, in which it is difficult to exclude hysteria; there may even be accompanying hysterical manifestations thus clouding the diagnosis. Chronic chorea and chronic hereditary chorea are differentiated with difficulty, particularly Unverricht's type.

The lack of direct heredity and appearance in childhood are important points of difference. Myotonia or Thomsen's disease is at times difficult to exclude, but the muscular fatigue of this latter is different.

The *prognosis* is bad.

C. TROPHONEUROSES

I. **Raynaud's Disease.**—Rare, and occurs usually in children and young adults, and in women more often than men. Predisposing causes are anæmia, chlorosis, and neurasthenic conditions. The chief causative factors are the acute infectious fevers, menstrual disturbances, malaria, fright, and occupations that lead to exposure. Syphilis and diabetes may be causes.

Symptoms.—The onset is sudden. Two or three fingers of both hands are usually involved. At first, or in a mild form, there is coldness, numbness, and waxy pallor of the fingers. They feel as if dead, the skin appears shrunken, and there is slight anæsthesia. This condition disappears, but returns again, and may become almost constant. All the fingers may be affected, more rarely the toes, tip of the nose, and the ears. This mild form (*digiti mortui*, dead fingers, local syncope) is due to a slight exposure to cold.

In the more severe form the fingers are blue and swollen. There are burning sensations and much pain, without anæsthesia. Gangrene follows this "local asphyxia." In the gangrenous stage the tips of the fingers become the seat of small blisters, which fill with bloody serum and then dry up. Beneath the scab thus formed a shallow ulceration begins, which soon heals and leaves a scar.

Occasionally hæmaturia may be present. The condition of *digiti mortui* may last a few days or weeks, perhaps months. The gangrenous stage usually continues for about three weeks.

Diagnosis.—This disease must be distinguished from senile gangrene, frostbite, ergot poisoning, alcoholic neuritis, endarteritis, and obstruction of the nutrient vessels. The *course* is long and the disease is liable to recur, but most cases recover.

II. **Erythromelalgia** (*Red Neuralgia of the Feet, Congestive Neuralgia*).—*Causes.*—Commonly affects men in middle life. The usual causes are severe physical exertion afoot and fevers. It occurs in diabetes and in the gouty.

Symptoms.—There are continuous burning pains in the ball or the heel of the foot, and all of the plantar nerve distribution on the sole of the foot is soon involved. The pain is usually worse at night. Walking and standing are painful. On exertion there is flushing of the feet, and in bad cases the parts affected present a continuous dusky, mottled redness, with some swelling. The hands may be slightly involved. Blisters and ulcerations may result from slight injuries. Lying down usually relieves the congestion and also the pain. In warm weather the symptoms are aggravated.

This disease must be distinguished from alcoholic and gouty paræsthesias, reflex pains, podalgia, and local disease of bone and ligaments. It is not dangerous to life, but is very chronic.

III. **Angio-neurotic Œdema** (*Circumscribed Œdema*).—*Causes.*—Occurs most frequently between the ages of 20 and 30, but is met with before and after this period. Males are affected oftener than females. The predisposing causes are exhausting occupations, occasionally hereditary influences. The disease appears oftenest in winter. The exciting causes are fright, anxiety, grief, sudden exposure to cold, certain kinds of food, and slight traumatisms.

Symptoms.—The onset is sudden. A circumscribed swelling appears within a few minutes, or an hour or two, upon the face or arms or hands. The swelling may measure from ½ to 2 or 3 inches across. It may be pale and waxy, or rosy, or of a dark reddish colour. There is slight, if any, pitting on pressure. There is no pain, but there may be sensations of itching, burning, scalding, or tension and stiffness. The face is most often affected, then the hands and extremities, the body, the larynx and throat, and the genitals, in the order mentioned. There is usually only one swelling at a time, but there may be several. This œdema lasts from an hour to 2 or 3 days, and often returns at intervals varying from 3 or 4 weeks to several months. If the disease affects the larynx and throat, serious dyspnœa and even death may result. The patient is well between the attacks. An hereditary form has been described.

Diagnosis.—The essential points are: the sudden appearance of the circumscribed œdema, the absence of pain, and the recurrence of the swelling at intervals. The *prognosis* is usually good as regards life, but not very satisfactory with reference to cure.

IV. **Progressive Facial Hemiatrophy.**—This begins usually between the ages of 10 and 20 years, and is more frequent in females. Occasional causes are injuries and infectious fevers.

Onset is slow. The skin loses its pigment, and the hair falls out in patches. Occasionally the periosteum and bone are involved. The disease affects the subcutaneous tissue most, the muscles least. There is atrophy of the bony parts, the lower jaw becoming perhaps two thirds of its normal size. The eye sinks in, the lid becomes narrow, and the pupil is dilated. The secretion of perspiration may be increased, but that of sebum ceases. Occasionally there may be some pain. Anæsthesia is rare. Slight spasms of the muscles of mastication may occur. The tongue may also be affected. The diagnosis is easy (Fig. 40, page 174). The disease must be distinguished from infantile hemiplegia with atrophy, atrophy from gross nerve lesions, and congenital asymmetry. The disease is rapidly progressive at first and then stationary. It is not curable, but does not affect life.

V. **Acromegaly** (*Marie's Disease*).—Occurs equally in males and females, and usually begins between the ages of 18 and 26 years. Many cases are associated with disease of the *pituitary body*.

Symptoms.—There is a gradual enlargement of the head, feet, and hands, accompanied by headaches, malaise, dulness, slight rheumatic pains, general weakness, anæmia, polyuria, and dryness of the skin. The sexual power diminishes in men and menstruation ceases in women. The bones and the soft parts are both hypertrophied, the enlargement being in width rather than length. The hands and feet, and the tongue, lips, and nose are enormously hypertrophied. The lower jaw is often more involved than the cranium. The sternum is hypertrophied and the chest bulging. The pelvis may be enlarged, but the bones of the thigh and leg usually escape. The arms and the shoulder girdle, except the clavicle, are not usually much affected. The face (see (*e*), page 172) is heavy and massive, the hair coarse and dry, and the skin frequently pigmented. There is a cervico-dorsal kyphosis. The voice is altered and the speech is slow, guttural, and embarrassed, apparently as a result of the enlarged tongue. The vision may be affected, and hemianopia may be present, especially if the tumour of the pituitary body presses on the optic tracts. The muscles, at first hypertrophied, afterward atrophy. Paralyses and anæsthesias do not occur.

Diagnosis.—The disease must be distinguished from osteitis deformans, congenital enlargements, and the so-called giant growth which affects only single members. The disease, as a rule, can not be cured, but it may become stationary. Its course is chronic, and it persists from 10 to 20 years.

II. DISEASES OF THE PERIPHERAL NERVOUS SYSTEM

A. DISEASES OF THE PERIPHERAL SENSORY NEURONES

I. **The Neuralgias.**—(I) **Coccygodynia.**—Neuralgia affecting the lower posterior branches of the sacral nerves. It occurs most frequently in women, and is due mainly to labour, injury, and exposure. Coccygeal pains occur in spinal irritation and also reflexly from pelvic disease. The disease is painful and interferes with walking and sitting. There is tenderness on pressure in the parts and also pain at stool.

(II) **Tarsalgia** (*Policeman's Disease*).—This is a neuralgic affection, of which incipient flat foot and stretching of the plantar ligaments is the most common cause. It occurs in people who have gone barefoot for some time, and who have then been obliged to stand or walk a great deal, as in the army or among the police.

(III) **Morton's Neuralgia.**—This disease affects the metatarso-phalangeal joint of the 3d and 4th toes. It occurs generally in women. It is due to flattening of the anterior transverse (metatarso-phalangeal) arch of the foot, and is analogous to flattening of the longitudinal arch (flat foot). Is increased by trauma and tight shoes.

(IV) **Sciatica** (*Neuralgia of the Sciatic Nerve, Sciatic Neuritis*).—*Causes.*—Men are more often affected than women. The disease occurs most frequently between the ages of 30 and 50, and in the fall and winter. The predisposing causes are occupations that lead to exposure and strain, the arthritic and gouty diathesis, and a neurotic constitution. The exciting causes are pressure from hard seats, constipation, pelvic diseases, exposure, and muscular strain from heavy work. Injury to the nerves, inflammation, the pressure of pelvic tumours, vertebral and spinal disease, may cause symptomatic sciatica. It may occur in phthisis and in diabetes.

Symptoms.—Onset usually sudden, with pain in the back of the thigh, running down the course of the nerve. It may extend up into the lumbar region, but is most marked in the thigh. Motion increases it, and consequently the pelvis tilts up toward the sound side and the trunk leans over toward the affected side (*sciatic scoliosis*). The pain is dull and almost continuous, with paroxysms in which it is sharp, lancinating, burning, and of great severity. There may be sensations of tingling, numbness, and a sense of weight and coldness in the affected limb. There are tender points at the sciatic notch, the middle of the hip, behind the knee, in the middle of the calf, behind the external malleolus, and on the back of the foot. Rarely there is anæsthesia along the course of the nerve. There may be weakness and muscular atrophy in chronic cases, and occasionally a partial R. D. The duration of the disease is usually about 2 or 3 months, although it may last for a year or more.

Diagnosis.—Sciatica must be differentiated from organic disease of the cauda equina or cord, hip-joint disease, muscular pains in the hip or leg, and from pains caused by tumours. If the leg is extended and the thigh at the same time flexed upon the abdomen, a sharp pain occurs at the sciatic notch which is diagnostic of sciatica. Recovery occurs in the majority of cases within 6 months.

(V) Plantar Neuralgia.—Very rarely the pain of sciatica is limited to the plantar nerves. In this condition there is paræsthesia and sometimes anæsthesia.

(VI) Lumbo-abdominal Neuralgia.—Affects the upper lumbar nerves. *Causes.*—Occurs usually in women after the 30th year. In addition to the usual causes of neuralgia, straining, pelvic disease, and constipation are found to be causative factors. Diseases of the hip or of the internal genitals cause reflex pain. Myalgic and reflex pains from uterine disorders are common. True essential neuralgias are infrequent.

Symptoms.—The chief symptom is pain in the back (frequently bilateral), loins, and buttocks, which extends down to the hypogastrium or genitals on one side. Occasionally painful points may be found. *Femoral* or *crural* neuralgia is a form in which the long lumbar branches are affected. *Meralgia* is a condition in which numbness and pricking is felt along the thigh. It is due to a lesser irritation of the long lumbar branches, and is a mild form of femoral neuralgia. The pain in true femoral neuralgia is in the front of the knee and the anterior and outer parts of the thigh. There is no pain below the knee.

Diagnosis.—The cardinal points are: the unilateral pain, with its distribution and paroxsymal character, the presence of tender points, the absence of organic disease, and of marked discomfort on motion or pressure. Lumbago has a sudden onset, a history of exposure, and is limited to a single group of muscles which are painful on deep pressure. In lumbar sprain there is a history of injury, a sudden onset, and marked local tenderness.

(VII) Mammary Neuralgia (*Mastodynia*).—True mammary neuralgia is due to injury, pendent breasts, anæmia, and pressure from badly fitting corsets. It also occurs in hysterical women and sexually precocious girls, and in pregnancy and during lactation. It is also caused by local tumours. Local disease of the breast causes many mammary pains. This form of neuralgia is unilateral and very severe. It may cause mental depression from fear of cancer.

(VIII) Herpes Zoster (*Shingles*).—A dermatitis secondary to an acute hemorrhagic inflammation of the posterior ganglia. The most frequent *causes* are infections, wounds, rheumatic, syphilitic, and gouty poisons, emotional influences, and the morphine habit. The *onset* is gradual, and characterized by pain and a herpetic eruption. The lower dorsal nerves are often affected, and the eruption follows the course of the nerve. The attack is usually over in a few weeks, but in some cases the pain may persist for long periods.

(IX) Intercostal Neuralgia (*Side Pains*).—This affection is common and occurs more frequently in women than in men, usually in the winter, and between the ages of 20 and 35. The chief causative factors are neurasthenia, hysteria, anæmia, childbearing, pelvic disease, heart disease, dyspepsia, lead-poisoning, and malaria. Very rarely exposure and muscular strain are exciting causes.

Symptoms.—The onset is sudden. The pain is sharp and stabbing, and but slightly increased by respiratory movements. There may be tenderness over the seat of the pain, over the exit of the dorsal or the anterior branch of the nerve. The left side is affected more often than the right. The duration of the disease is usually from 2 to 6 weeks, but it may last months.

Diagnosis.—This neuralgia must be distinguished from myalgic pains by the character of the latter, the history of rheumatism and of the cause, by the pain on deep inspiration, and by the tenderness on pressure. The important points in diagnosticating intercostal neuralgia are the presence of rheumatic and reflex causes, the character of the pain, the presence of tender points, and the exclusion of pleurisy. The prognosis is good. The disease occasionally becomes chronic, especially when due to a degenerative neuritis.

(X) **Cervico-brachial Neuralgia.**—Not frequent, and occurs in early adult and middle life, mainly in women. Caused by rheumatism, gout, and overuse of the arm in anæmic and neurasthenic patients. It occurs as a symptom of tabes and other cord diseases, and also reflexly from uterine disease and caries of the teeth.

Symptoms.—Onset is gradual, with aching pains along the course of the nerves and in the neck, shoulder, and axilla. One arm only is affected. The pains are worse at night, and are increased by exposure and use of the arm. There may be painful or tender points in the axilla, over the deltoid, at the lower end of the scapula, over the lower part of the radius, over the ulna near the wrist, and occasionally on each side of the lower cervical vertebræ. Paræsthesias and feelings of numbness are present. Vasomotor disturbances, herpes, anæsthesia, and muscular weakness and atrophy may appear.

(XI) **Digital Neuralgia.**—A single finger may be affected. Neuritis or a local injury is the usual cause. Occasionally the pain may be reflex from some distant trouble (e. g., uterine). The main point is to find the cause of the neuralgia, and to exclude organic disease. The prognosis is good unless the neuralgia is complicated with a neuritis, when it is more serious. If the neuritis, however, is not marked, or is rheumatic or gouty, or is secondary to injury, the prognosis is still good.

(XII) **Cervico-occipital Neuralgia** (*Neck Pains*).—*Causes.*—These pains occur in migraine, hysteria, neurasthenia, and spinal irritation;

as a true neuralgia; as a result of eye strain, and as a symptom of brain tumour, meningitis, and rheumatic inflammation of the nerves and muscles of the neck. True cervico-occipital neuralgia occurs more commonly in women and between the ages of 20 and 35. Pelvic disease often causes it reflexly.

Symptoms.—In the typical form the pain is paroxysmal, unilateral, and sharp, sometimes intense, and there are tender points over the nerves. The duration of the disease is about 5 or 6 weeks, but it may become chronic if due to a reflex cause. When due to hysteria or spinal irritation the disease is central and there is a sharp boring pain below the occiput. Symptoms of cerebral congestion or anæmia, and vertigo and faintness, may be present, but no vomiting. Pain of a boring character points almost positively to spinal irritation. The pain in neurasthenia is more like a tired ache.

(XIII) **Neuralgias of the Trigeminus.**—(1) *Symptomatic Form.*—This is the most frequent, and includes supraorbital, infraorbital, or supramaxillary, inframaxillary or dental, and mixed forms. Supraorbital neuralgia is the commonest variety. It occurs in females oftener than in males, usually in the first half of life. The left side is generally affected. The causes are dental disorders, exposure, anæmia, childbearing, disease of the eyes or nose, syphilis, gout, rheumatism, diabetes, epilepsy, malaria, and trauma.

The pains are very sharp and severe, with exacerbations and remissions. They may last for days and then be absent for a long time, but they come back unless the cause is removed. There may be œdema of the eyelids in supraorbital neuralgia. There are tender points along the course of the nerve involved. The pain may radiate from some point, as the ear or occiput. The pupil is occasionally dilated, and there may be a reflex facial spasm.

(2) *Tic Douloureux.*—This form of neuralgia occurs in middle or advanced life, and is very severe. It is due to disease of the Gasserian ganglion. Exposure, overwork, and depressing influences are accompanying factors. The chief symptoms are the intense, unilateral, darting pains, which radiate from the side of the nose or the upper lip, through the teeth, or into the eye, or even the brow and head. The pains come in paroxysms, lasting for a few minutes, during which the face is flushed and the eyes and nose run. There is an expression of agony on the face. The patient is seldom free from some pain, and a breath of cold air, speaking, eating, or putting out the tongue may bring on a paroxysm. These pains may come on for a certain length of time each year. There are often spasmodic movements of the face, tongue, or jaws. Occasionally there is anæsthesia. This disease is very difficult to cure permanently without surgery.

(3) *Trigeminal Paræsthesia.*—A thrilling, formication, or numbness along the course of the nerve. There is no pain. It occurs in anæmic, nervous, and hysterical people.

B. DISEASES OF THE PERIPHERAL MOTOR NEURONES

I. **Multiple Neuritis.**—Onset acute, but running a subacute or chronic course, and characterized by weakness or paralysis of all four extremities, accompanied by atrophy, pain, and tenderness.

(1) *The Motor Type.*—This is the commonest form. It is a disease of early adult life, and occurs in women more often than in men. Occasionally it occurs in children. Predisposing causes are exposure to cold and wet, excessive tea-drinking, anæmia, sexual abuse, and tuberculosis. The exciting causes include nearly all the infectious fevers and malaria, arsenic, alcohol (most common), copper, phosphorus, lead, rheumatism, and diabetes.

There may be numbness, slight pains and general weakness, especially in the legs, for a few weeks before the attack. The onset is usually sudden, with pains and tenderness in the legs and feet and a fever for a day or two. The pains and weakness increase and the muscles and nerves become very tender. The arms and hands are also affected, but not as severely. The skin becomes reddened or slightly edematous. The muscles of the legs grow weak, and in a week or so there may be a complete paralysis of the anterior muscles of the legs and a corresponding loss of power in the extensors of the hands, but to a less degree. There may be a loss of co-ordination. The pains are severe. Nearly all the muscles of the legs and forearms become more or less involved, and atrophy begins. Rarely the motor cranial nerves are affected. Slight nystagmus is common. The condition soon develops into that of a paraplegia with foot- and wrist-drop. The knee-jerk and elbow-jerk are absent, except in rare cases. There is some diminution or delay in temperature and pain sensibility. Tactile anæsthesia is present, often associated with hyperalgesia. The muscular and articular senses are usually impaired. There is R. D. in the muscles, varying in degree in different muscles and nerves. The sphincters are rarely involved, the bladder only being occasionally affected for a short time. In alcoholic cases there may be mental symptoms, usually consisting of a low muttering delirium. An acute confusional insanity may develop. There is often marked prostration. In diphtheritic neuritis some of the throat and eye muscles are involved, with few sensory symptoms and less involvement of the extremities. In the less affected muscles contracture and shortening may occur. Sensory symptoms consist of numbness, hyperæsthesia, severe pains, marked tenderness and burning sensations, and tactile anæsthesia. They occur in the hands, legs, and feet.

(2) *Sensory or Pseudo-tabetic Form.*—Diabetes, and infectious and metallic poisons are the causative factors in this form more often than alcohol. The course is similar to that of the motor form, although the paralysis is usually less marked and the sensory symptoms are more prominent. The muscular and articular senses are lost. Inco-ordination is a prominent symptom, hence the resemblance of this form to tabes dorsalis.

(3) *Epidemic and endemic forms* are due to acute infection and malaria (see Beri-beri, page 830).

(4) *The acute pernicious form* comes on suddenly. The course is rapid, and death follows in a few weeks. The cardiac and respiratory nerves are involved. The cause seems to be some septic organism. Landry's, or acute ascending, paralysis is another form in which acute pernicious multiple neuritis appears. In this type there are no electrical changes or atrophy and few sensory symptoms.

Differential Diagnosis of Multiple Neuritis.—The cardinal points are: alcoholic history, acute onset, gradual paralysis of legs and hands; foot-drop and wrist-drop; severe pains and tenderness; early loss of faradic and change in galvanic excitability; the absence of sphincter involvement; and loss of the knee- and elbow-jerks.

It is distinguished from anterior poliomyelitis by the persistence of pain and other sensory symptoms, the tenderness of the nerve trunks, the early loss of electrical excitability, the history of the case, and the age of the patient. In anterior poliomyelitis the paralysis is not symmetrical, and the loss of electrical excitability occurs after weeks or months. In myelitis the bladder is affected, while in multiple neuritis it is seldom disturbed. The onset and progress of myelitis are more rapid, the muscular atrophy less, and the knee-jerks are increased. It is distinguished from tabes by its more rapid onset, the presence of paralysis, atrophy, and R. D., and the absence of involvement of the organic centres and pupils. In spinal hemorrhage there is usually pain in the back. Neuralgia is unilateral, while the pains of multiple neuritis are bilateral. The persistence of tenderness and hyperæsthesia would also indicate multiple neuritis.

The *prognosis* of multiple neuritis is good in all forms except those due to alcohol or toxæmia. The majority of cases recover almost entirely. In alcoholic cases death is frequent, although often not due to the neuritis, but to alcoholism or to phthisis.

II. **Symmetrical Spontaneous Ulnar Neuritis.**—Probably a degenerative neuritis and due to some infection. The exciting cause is unknown. A neuropathic tendency is the predisposing cause. The onset is gradual, with pain and paræsthesia along the ulnar nerve of one side. There are weakness and atrophy of the muscles supplied by the ulnar. Anæsthesia develops also. The other hand then becomes affected. The disease is chronic.

III. **Migrating Neuritis.**—This is a rare disease, and usually follows an operative or other wound of a nerve, the neuritis extending up the arm. Pain, paralysis, anæsthesia, and atrophy are the chief symptoms. The course of the disease is chronic.

IV. **Acute Ascending Paralysis.**—(*Landry's Paralysis*).—A rapid paralysis, involving first the legs, then the trunk, arms, respiratory and throat muscles, generally ending in death. It usually affects men between the ages of 20 and 40. The causes are exposure, syphilis, and the acute infectious fevers.

Symptoms.—Slight prodromal symptoms may be present for a few days, consisting of general malaise, slight fever, numbness in the limbs, and pain in the back or legs. When these are absent, weakness in the legs is the first symptom. The paralysis rapidly extends and soon affects the arms. Finally, the medulla is involved, respiration becomes difficult, and the patient may be unable to talk or swallow. There is almost no pain or disturbance of sensation, although there may be slight anæsthesia. Atrophy, R. D., and secretory or vasomotor disturbances are absent. The bladder and rectum are rarely affected, and sometimes there are facial and eye palsies. The disease *may* begin in the cervical region and descend.

Diagnosis.—The cardinal points are: the acute ascending course of the paralysis, the *absence* of fever, anæsthesia, decubitus, sphincter involvement, and, especially, R. D. Acute myelitis, acute multiple neuritis, and acute poliomyelitis are the diseases from which it must be distinguished. It should be borne in mind here that there is good reason to regard Landry's paralysis as an extensive anterior poliomyelitis. Whether the central or peripheral lesion is primary is difficult to decide. An alcoholic history and the age of the patient must be taken into account. The *prognosis* is bad. The disease usually runs its course within 7 days and ends in death. If it does not extend into the medulla, and the patient is paralyzed only below his neck, improvement begins, and in 1 or 2 years a partial recovery of muscular power may take place.

V. **Paralysis of the Spinal Part of the Accessory.**—*Causes.*—Caries of the vertebræ, chronic meningitis, progressive muscular atrophy, injuries, and all forms of spinal disease which extend high up in the cervical cord.

Symptoms.—If one nerve is paralyzed there is inability to rotate the head perfectly, but it can still be held straight. There is atrophy of the sterno-mastoid. If the trapezius is involved there is a depression in the neck, seen best on deep inspiration. It is difficult to raise the arm. When both nerves are paralyzed there is marked difficulty in rotating the head or lifting the chin. Paralysis of both sterno-mastoids tends to cause the head to fall backward; of the upper parts of both trapezii, to drop forward. The paralysis is followed by atrophy and R. D. When both parts of the spinal accessory are affected there are also dysphonia, dropping of the palate, and rapid pulse. The characteristic features are marked.

VI. **Paralysis of Phrenic Nerve.**—Due to injury or disease of the cervical cord, peripheral disease, hysteria, mediastinal tumours, rheumatic and toxic influences, pleurisy, and peritonitis. The most common causative factors are, however, tabes dorsalis, acute ascending paralysis, spinal-cord disease, and surgical injuries. In bilateral paralysis of the diaphragm there is no movement of the abdomen or the epigastrium, and the hypochondrium is drawn in (pages 417, 418). Dyspnœa and rapid breathing follow slight exertion. If the diaphragm alone is affected the trouble is in the trunk of the nerve.

VII. **Combined Paralysis of the Brachial Nerves.**—Occurs oftenest in men. It is not rare in infants as a result of obstetrical injuries. Exciting causes are injuries, tumours, dislocation of the shoulder, neuritis, and crutch and other compressions. There are also functional palsies from hysteria and overwork.

According to the degree of compression or injury to the nerve, there are pain, tenderness, paralysis, atrophy, electrical changes in the muscles, anæsthesia, or trophic, vasomotor, and secretory disturbances. If the musculo-cutaneous is chiefly affected the patient can not flex the forearm. If the musculo-spiral is affected the patient can not extend the arm. If the circumflex or the lower cervical and upper dorsal nerves and the posterior thoracic are involved the patient can not elevate the arm outward.

Diagnosis.—Can be made by ascertaining the special nerves involved and the nature and seat of the lesion. Distinguish between (1) a *total-arm palsy*; and (2) an *upper-arm type* (*Erb's palsy*), in which the deltoid, the biceps, brachialis anticus, supinator longus, and sometimes the supinator brevis are involved. The arm hangs by the side and the forearm can not be flexed. Erb's palsy is frequent in infants and is one of the obstetrical palsies. There is also (3) a *lower-arm type*, in which the triceps, the flexors of the wrist, the pronators, the flexors and extensors of the fingers, and the hand muscles are involved. The hand is useless and the extension of the forearm is impossible. Combined nerve palsy due to a primary brachial neuritis is differentiated from progressive muscular atrophy by the pain, anæsthesia, and electrical reactions. The *prognosis* is usually good. Nearly all cases recover.

VIII. **Paralysis of Single Brachial Plexus Nerves.**—(1) *Posterior Thoracic.*—Causes are sudden strains or injuries in the neck. Occurs rarely, usually in adult males. May be involved in progressive muscular atrophy. The nerve supplies the serratus magnus. There may be pain, and the course of the paralysis is often long. There is difficulty in raising the arm above the horizontal position, and the movements of the shoulder are impaired. The posterior edge of the scapula recedes from the thorax when the arm is put forward. At the same time the scapula is rotated, its lower angle going inward and upward (Fig. 82, page 302).

(2) *Circumflex.*—Often paralyzed. It supplies the deltoid, teres minor, third head of the triceps, and shoulder joint. It is *caused* by injuries or falls, dislocation, and rheumatic inflammation. The *symptoms* are anæsthesia, atrophy, and occasionally pain. The arm can not be rotated outward or elevated.

(3) *Suprascapular.*—Rare. The teres minor, spinati muscles, and the shoulder joint receive this muscle. There is an impairment in elevation of the shoulder and also in rotation.

(4) *Musculo-spiral* (*Wrist-drop*).—Frequently paralyzed. Pressure on the nerve during sleep, fractures, wounds, crutch pressure, tumours, lead, arsenic- and alcohol-poisoning, and multiple neuritis are common causes.

Symptoms.—There is an inability to extend the wrist and fingers, and "wrist-drop" results. The first finger is least affected. There may be atrophy and R. D. Numbness may be present, and also some anæsthesia over the radial nerve distribution on the hand. The course of the disease is short if due to pressure, but longer if due to lead-poisoning, neuritis, or severe injury of the nerve. When lead-poisoning is the cause the supinator longus is usually unaffected, the paralysis comes on more slowly and affects both arms, and there is seldom any pain or anæsthesia. In paralysis due to compression there is often an involvement of the supinators and the triceps.

Diagnosis.—Easy. Differentiate between lead palsy, neuritic palsy, and compression palsy. Find out the cause of the paralysis. Exclude progressive muscular atrophy. The *prognosis* is good.

(5) *Median.*—Rarely affected alone, but may be due to injuries or neighbouring lesions. Abduction and flexion of the thumb and flexion of 1st and 2d fingers are impaired. The grip is weakened. There may be atrophy of the thenar eminence. There is anæsthesia.

(6) *Ulnar.*—Frequently paralyzed. Early involved in progressive muscular atrophy, but rarely affected in lead-poisoning. Injuries

are the most common cause. It may be the seat of a primary degenerative neuritis.

Symptoms.—The ring and little fingers are especially weak, and the hand can not be closed tightly. There is a condition of "*main en griffe.*" There is some anæsthesia over the distribution of the nerve. Pain and tenderness may be present.

IX. **Paralyses of the Lumbar Nerves.**—Paralyses and spasmodic troubles of these nerves are usually symptomatic—e. g., of caries of the spine, psoas abscess, impacted fæces, pelvic tumours or injuries, hip disease, obturator hernia, or pressure of the fœtal head.

If the 2 upper lumbar nerves are affected only sensory symptoms occur. If the next 2 are affected there is trouble in extending the leg and flexing the hip on the trunk. If the *obturator nerve* is paralyzed the thigh can not be adducted or the leg crossed, and outward rotation of the thigh is weak. If the *anterior crural* is paralyzed the anterior thigh muscles are weak and the leg can not be extended. There is anæsthesia or pain over the crural area. If the *posterior branches of the lumbar nerves* are paralyzed there is weakness or paralysis of the erectors of the spine. This occurs in progressive muscular atrophy.

X. **Peripheral Leg Palsies.**—May be combined or single.

(I) *Combined Palsies.*—Due to dislocation, injury, hip disease, tumours, and neuritis. There may be an hysterical sacral palsy. The foot can not be moved nor the leg flexed. The thigh can not be drawn back or rotated freely. Pain, anæsthesia, atrophy, and vasomotor and secretory disturbances are present. The *diagnosis* depends on the history, the area of anæsthesia, and the extent of paralysis. It is differentiated from spinal-cord disease by the unilateral symptoms, the absence of sphincter trouble, and the atrophy and sensory disturbances.

(II) *Single Nerve Sacral Palsy.*—The sciatic is oftenest affected, especially its anterior tibial branch. *Symptom.*—" Foot-drop."

XI. **Facial Palsies.**—The most common variety is here considered (see also pages 185 to 190).

Peripheral Facial Palsy (*Bell's Palsy*).—*Causes.*—Infection, exposure, and rheumatism. Occurs between the ages of 20 and 40, more frequently in males. A neuropathic constitution predisposes. It may occur in tabes and multiple neuritis, or follow injuries to the petrous bone, ear disease, or swelling in geniculate ganglion.

Symptoms.—Sudden onset. Is usually complete in a few hours. There may be some pain about the ear. For a description of peripheral paralysis, see page 185. If the paralysis is not complete,

latissimus dorsi. Some atrophy is often present in the muscles of the upper arm, but those of the face, neck, and forearm are rarely affected. There may be hypertrophy of the tongue.

Together with the pseudo-hypertrophy there is an atrophy of certain groups of muscles. After a time the pseudo-hypertrophy disappears and the atrophy increases. In the lower limbs the atrophy affects chiefly first the flexors of the hips, then the extensors of the knee and hip. The calf muscles become atrophied, then the anterior tibial groups. The gait becomes more waddling; there is great difficulty in going upstairs, and the patient can not get up from the floor. This disability is due to the weakness of the extensors of the knees and hip and the flexors of the hip. For the same reason there is a condition of lordosis, which disappears when the patient sits. There may be also some lateral curvature of the spine. As a result of the weakness and contractures of the leg muscles a talipes equinus appears early. Later the forearms may become flexed on the arms, and the legs on the hips. There is no R. D. The knee-jerks and elbow-jerks gradually diminish and are finally lost. There are no fibrillary twitchings, pain, or sensory disturbances. There is no mental impairment or involvement of the organic spinal centres. The parts affected feel cold and have a reddened appearance.

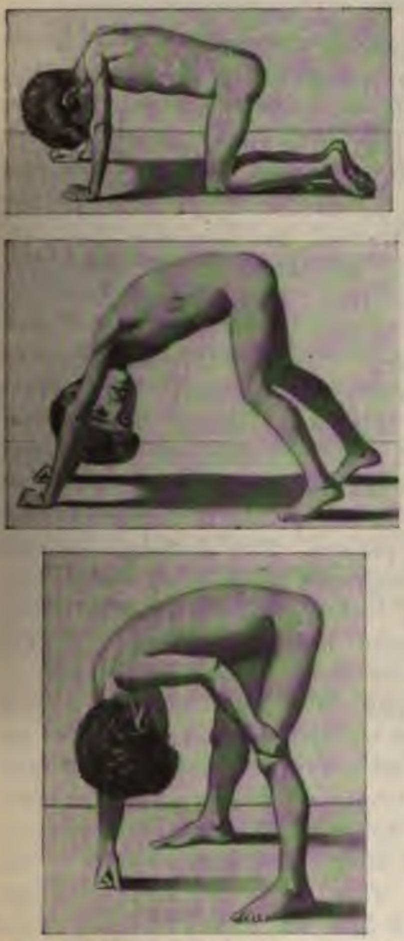


FIG. 271.—Mode of rising from floor in pseudo-muscular hypertrophy. Read from above downward.

Diagnosis.—The chief diagnostic points are: the peculiar gait, and the mode of rising from the floor (Fig. 271); the age of the patient; the progressive character of the impairment; the enlargement of the calf muscles; the combination of enlargement of the infraspinatus with a wasting of the latissimus dorsi and lower part of the pectoralis. In congenital spastic paraplegia the knee-jerk is exaggerated, there are spasms of the legs, the resulting contractures of which can be overcome, and the patient does not rise from the floor in the same way as in pseudo-hypertrophic paralysis.

Prognosis.—The disease may last for from 10 to 25 years or longer. Its progress at first is slow, but the prognosis is serious, and the patient does not live long after becoming bedridden.

(II) **Juvenile Dystrophy of Erb** (*Scapulo-humeral Form of Dystrophy*).—This form of dystrophy begins in childhood or early youth, a little later than pseudo-hypertrophy. The muscles of the shoulder girdle are first attacked, then those of the arm. The legs and forearms are not involved until late in the disease. The muscles affected are parts of the pectoralis and trapezii, the latissimus dorsi, the rhomboid, upper-arm muscles, and supinators. The supraspinati and infraspinati are not usually involved. Fibrillary twitchings and R. D. are absent. False and true hypertrophy may occur.

(III) **Facio-Scapulo-Humeral Form** (*Landouzy-Dejerine Type, Infantile Progressive Muscular Atrophy of Duchenne*).—This occurs usually in the 3d or 4th year of childhood. It may develop later. There is first atrophy of the facial muscles, causing the "myopathic face." The oral muscle becomes weak and the lips protrude ("tapir mouth"). There may be atrophy of the muscles of deglutition and mastication, and also of those of the eye. The shoulders are next attacked, and then the disease progresses as in other dystrophies. The age of onset, the hereditary history, and the location of the initial atrophies are the cardinal points in diagnosing this form. The *prognosis* is bad. In some cases the disease becomes stationary.

(IV) **Arthritic Muscular Atrophy.**—The most common cause is rheumatic arthritis. The muscles oftenest affected are those of the shoulder girdle. The extensors are always first and most affected, whichever joint is attacked. Atrophy is greater in the muscles above the joint than in those below it. The atrophy advances rapidly at first, but more slowly later. The whole length of the affected muscles becomes wasted. There are no fibrillary twitchings, no R. D., no pain, tenderness, or anæsthesia. The irritability of the muscles may be increased, and there may then be exaggerated reflexes and perhaps a clonus. The *prognosis* is good. The muscles recover when the arthritis becomes well.

(V) **Occupation Muscular Atrophies.**—Constant overuse of certain muscles is the cause of this form of atrophy. The atrophy occurs most frequently in the small muscles of the hand. Usually the atrophy disappears when the muscles are given rest, but it may pass over into progressive muscular atrophy. It is distinguished from the latter by the absence of pain and vasomotor symptoms.

III. DISEASES OF THE SPINAL CORD AND MEDULLA

The nosological grouping of the diseases of the spinal cord and medulla is one of great simplicity, from the point of view of the anatomical arrangements of the cells and fibres. Figures 195 *a* and *b*, pages 524, 525, also Figs. 193, 194, page 523, show these topographic relations, and an understanding of the anatomy of the cord and the medulla renders the interpretation of the clinical picture seen in spinal cord and medullary affections a comparatively definite affair.

It is essential to bear in mind, however, that clear-cut types of disease are the exception rather than the rule. In spinal-cord diseases, as in affections of other parts of the body, writers are accustomed to describe *typical forms*. These are in reality the well developed and completely rounded out clinical groupings, and are rare cases rather than the common ones—the word *typical* should read *well developed.* Thus it is found that the clinical groups which are dealt with in this section represent the well-rounded classical types, those founded on the anatomical configuration of the structures affected. In practice it will be found that the disease processes will be found incompletely developed and the types will merge one into another. It should be added, however, that in no other part of the body can the clinical picture and the anatomical substratum be brought into quite such a definite correlation.

Diseases of the spinal cord and the medulla may thus be arranged under eight anatomically comprehensive, and clinically most helpful, groups, as follows:

(1) Anterior horn motor nuclei group.—Anterior poliomyelitis type. Syndrome of muscular paralysis, lost reflexes, atrophy.

(2) Anterior horn nuclei and lateral column group.—Amyotrophic lateral sclerosis type. Syndrome of muscular weakness, spasticity, atrophy.

(3) Diffuse disease spinal cord.—Myelitis type. Syndrome of spastic paraplegia with or without sensory or nucleus changes according to site.

(4) Central gray substance group.—Syringomyelia type. Syndrome of pareses, atrophy, dissociation of heat, pain, and tactile sense.

(5) Antero-lateral column group.—Lateral sclerosis type. Syndrome of paralysis, spasticity, increased reflexes.

(6) Posterior sensory column group.—Locomotor ataxia type. Syndrome of ataxia, lost reflexes, sensory changes.

(7) Secondary degenerations group.—Hemiplegic type. Multiple sclerosis. Syndrome of paralysis, spasticity, unilaterality.

(8) Combined scleroses group.—Posterior and lateral column. Two general syndromes. (*a*) Spastic spinal paralysis type; (*b*) ataxia with increased reflexes.

ANTERIOR HORN NUCLEI GROUP

I. **Acute Anterior Poliomyelitis.**—This is a disease of the anterior horns of the spinal cord (Fig. 195) with sudden paralysis of one or more limbs. It occurs at all ages, but the average age at the time of the attack is 2 years. Occurs more often in males and in the hot summer months. It is usually epidemic. Infectious fevers (especially measles), falls, injuries, overexercise, and chilling of the body when heated appear to be contributory causes.

Symptoms.—The onset is sudden, with slight fever (100° to 102°) lasting from 1 to 4 days, sometimes with vomiting, diarrhœa, delirium, and convulsions. Within 24 hours in acute cases, or a week in the subacute, paralysis appears, most commonly a paraplegia, or in one leg, or a diplegia, or simultaneous monoplegias. After the acute symptoms subside there may be pain in the back and limbs for a few days. Rarely there may be retention of urine. In infants the respiratory, laryngeal, and ocular muscles are not involved. The paralysis increases for from 1 to 4 days, remains stationary for from 1 to 6 weeks, and improves for from 6 to 12 months. Within 2 or 3 weeks there may be some atrophy in the affected limbs. The paralysis gradually disappears from the limbs least affected, but leaves one or both legs, generally one (and that the right) permanently paralyzed. The affected limb is cold, mottled, and reddish purple in colour; the muscles are flaccid, become atrophied, R. D. is present, reflexes absent. In the leg the anterior tibial group of muscles are oftenest affected and foot-drop results. The paralyzed limb does not grow as rapidly as the unaffected limb, but remains smaller and shorter, and may develop contractures and various deformities—e. g., in the leg, talipes equinus, varus, and valgus, and contraction of the plantar fascia. Lateral curvature of the spine and a deformed knee may ensue. In adults the facial nerve may be attacked. Rarely the nuclei of the ocular muscles are involved (polio-encephalitis superior) or the lower cranial nerve nuclei (polio-encephalitis inferior).

Differential Diagnosis.—The cardinal symptoms are: infancy, sudden onset, immediate and profound paralysis; the absence of spasticity, pain, anæsthesia, and vesical or rectal symptoms; the presence of R. D., the subsequent improvement, and the arrested development of the permanently paralyzed limb. In *cerebral palsy* there is spasticity, the reflexes are exaggerated, the paralysis is unilateral, and there is no atrophy or R. D. In *myelitis* and *spinal-cord hem-*

orrhage there are disorders of sensation, vesical symptoms, and bedsores. *Multiple neuritis* and *progressive muscular atrophy* rarely occur in children, and their onset is not so abrupt.

Prognosis.—Good as to life, but recovery is rarely complete, one or more muscle groups remaining paralyzed.

II. **Chronic Anterior Poliomyelitis.**—This disease is rare. Adult males are chiefly affected. The principal causative factors are exposure, syphilis, and lead-poisoning. One or more, sometimes all, the extremities gradually become paralyzed, and atrophy with R. D. quickly follows. There are no vesical symptoms, and only slight pain and sensory troubles. The presence of pain, tenderness, and anæsthesia in *multiple neuritis* distinguishes it from this disease. The rapid onset, the paralysis quickly followed by atrophy the early R. D. and the absence of fibrillary twitchings distinguish it from *progressive muscular atrophy.* It is impossible to differentiate a central from a peripheral atrophy, and the distinction is artificial.

In some cases, after the paralysis reaches its height an improvement sets in which may be nearly complete. In others the disease progresses until a condition similar to progressive muscular atrophy exists, and after a short time, or perhaps 1 or 2 years, death ensues.

III. **Progressive Muscular Atrophy** (*Progressive Spinal Amyotrophy, Duchenne-Aran's Disease*).—May occur between 14 and 70 years of age, usually between 25 and 45, and is more frequent in males. The chief causes are traumatism, exposure, excessive use of certain groups of muscles, great mental strain, childbirth, syphilis, acute infectious diseases, especially typhoid fever, cholera, and measles, acute rheumatism, and lead-poisoning (frequent) (Fig. 195).

Symptoms.—The first symptoms are slight rheumatoid pains in the arm or shoulder, with some numbness and a feeling of weariness. Atrophy next appears, usually in one hand (Fig. 270) the adductor longus pollicis, the thenar, and the interossei muscles being the earliest affected. The wasting extends without regard to the nerve distribution, although the muscles supplied by the ulnar nerve are most affected. The different motions of the fingers become impaired according to the muscles involved. The wasting passes gradually up the forearm to the arm and shoulder. The flexors and extensors are alike affected. The hand is now flattened and thin, there is inability to flex the wrist or extend the fingers, and the characteristic condition of *main en griffe* or claw hand is the result. The other hand usually becomes affected within from 3 to 9 months. Occasionally the shoulders and arms are first affected ("upper-arm type"), the atrophy appearing in the deltoid, triceps, and biceps, and extending downward to the hands.

The wasting usually progresses, passing from the shoulder muscles to the deep muscles of the back, thence down to the hip and thigh muscles. In this region the glutei, the crural abductors and extensors are most often affected. The muscles of the legs are not usually involved, although they may be. The wasting in the trunk extends to the intercostals. The disease may ascend the neck, resulting finally in paralysis of the diaphragm, or a bulbar palsy may begin. Exceptionally the atrophy may begin in the legs and ascend.

From the beginning there are weakness and paralysis corresponding to the degree of wasting. This paralysis follows and is due to the atrophy. There are fibrillary twitchings, and the myotatic irritability is marked. Usually the muscles are flaccid, the deep reflexes, knee-jerk, and arm-jerk are early lost, and the condition is one of "atonic atrophy." Contractures may result and a pseudo spasticity be apparent.

The electrical irritability of the muscles is at first only diminished, but later there may be partial R. D. Paræsthesias and rheumatoid pains may occur. There is no anæsthesia in uncomplicated cases. Often there are vasomotor disturbances, excessive sweating, and congestion in the parts involved. One or both sides of the face may be affected. The pupils may be unequal in size. The optic nerve is never involved. The sphincters are not affected. The sexual power may be impaired.

Diagnosis.—The disease must be distinguished from the progressive muscular dystrophies, syringomyelia, neuritis, neuritic family atrophy and from spinal syphilis. The muscular dystrophies offer a history of heredity; the disease begins in childhood; the progress is slower; there is a more frequent involvement of the lower limbs; and an absence of R. D. and of fibrillary twitchings. In syringomyelia there are peculiar trophic and sensory disturbances. In neuritis there is the history of the case, the involvement of the extensors of the forearms chiefly, and no progressive tendency. Occasionally lead palsy develops into a true progressive muscular atrophy. The onset of multiple neuritis is rapid, and pain is a prominent symptom. The onset of chronic anterior poliomyelitis is sudden and the disease does not progress after it has reached its height. The wasting follows the paralysis. The muscles which are affected are physiologically related, not merely anatomically, as is true in the case of progressive muscular atrophy. In the hereditary or "leg type" of progressive muscular atrophy the legs are first affected, there are marked sensory disturbances, a hereditary or family history, and typical R. D.

Prognosis.—The disease usually progresses with remissions until it is well developed and then remains stationary. Its duration is from 2 to 30 years, with an average of 10 years. Death is usually caused by lung disease due to paralysis of the respiratory muscles, sometimes by involvement of the deglutitory and laryngeal muscles.

IV. **Progressive Hereditary Muscular Atrophy of Leg Type** (*Charcot-Marie-Tooth Type*).—Occurs in males somewhat oftener than in females, usually before the age of 20. It is a family or hereditary muscular atrophy of a central or neuritic origin.

Symptoms.—The muscles of the leg, not the foot, are primarily affected, the order of involvement being first the peronei, then the extensors of the toes, then the calcaneal muscles. The thighs are not affected until later. The small hand muscles and those of the upper extremities become involved after some years; still later the arm muscles, except the supinator longus. The trunk, neck, and shoulder muscles are not affected. There may be some fibrillary twitchings. R. D., varying in degree, is always present. There is no anæsthesia, but there may be some pain and numbness. Occasionally there may be spasmodic contractions, especially of the thigh muscles. The *course* of the disease is long, and there may be remissions, but it is incurable.

V. **Glosso-Labio-Laryngeal Paralysis** (*Progressive Bulbar Paralysis*).—Occurs usually between the ages of 40 and 70, more frequently in men. A neuropathic heredity is occasionally found. The chief causes are syphilis, lead-poisoning, exposure, excessive use of the muscles in talking, debilitating influences, and mental strain.

Symptoms.—The disease first affects the tongue, which can not be elevated or protruded, and appears wrinkled and scarred. The patient does not talk distinctly (see (2), page 263), and is unable to pronounce the lingual consonants, *l*, *n*, *r*, and *t*. Next, the lips become weak, and there is inability to articulate the consonants *m*, *p*, *b*, or the vowel *o*, or to whistle. Difficulty in swallowing, and dribbling of saliva from the mouth soon begin. Hard solids, and later fluids, are the most difficult to manage, semi-solids being taken more easily. The lips finally become so paralyzed that the patient can not shut the mouth, and the lower part of the face becomes motionless and expressionless, while the upper part manifests great suffering and anxiety. Sometimes the facial nerve becomes a little involved. Articulation is at last almost entirely impossible. Paralysis of the palate gives the voice a nasal twang.

The patient's throat feels tired with a sense of dryness and stiffness. There may be some impairment in the sense of taste. Pain and anæsthesia are absent. In the latter part of the disease there is partial R. D., but at first the electrical reaction is normal. There is a weakness of the laryngeal reflex, and of the adductors, but abductor paralysis is uncommon. There is no mental disturbance, but often there is some emotional weakness. The disease may be the final stage of spinal muscular atrophy, or it may be associated with the latter, or with ophthalmoplegia, or amyotrophic lateral sclerosis.

Diagnosis.—The cardinal points are: slow onset; progressive course; the bilateral character; absence of involvement of sensory nerves and R. D This disease must be discriminated from polio-encephalitis inferior, tumours, bulbar apoplexy, softening, multiple sclerosis, chronic lesion of the cerebral hemispheres causing pseudo-bulbar paralysis, and asthenic bulbar paralysis. There is marked paralysis in asthenic bulbar paralysis, but no typical atrophy.

The *course* of the disease is progressive, often with remissions of a few weeks or months. The duration of the disease is from 3 to 4 years. Death results from inanition, or pulmonary disease.

VI. **Asthenic Bulbar Paralysis and Asthenic Bulbo-spinal Paralysis.**—Occurs usually under the age of 30 years, but may occur later. Sometimes profound anæmia is a causative factor, also overwork and mental strain.

Symptoms.—The onset is gradual. The muscles of the throat, face, and eyes are oftenest affected. Ptosis of one or both eyes is a common symptom. Weakness of the muscles of mastication, defective articulation, and difficulty in swallowing follow. The voice is nasal. The condition resembles that of glosso-labio-laryngeal paralysis. Some ophthalmoplegia develops with great exhaustion and marked weakness of the extremities. The patient becomes almost helpless, perhaps unable to lift his arms or legs.

Diagnosis.—This disease is distinguished clinically from true bulbar paralysis and progressive muscular atrophy by the fact that there is no true atrophy of the muscles of the face, tongue, or extremities, and no fibrillary twitchings, and that its course is irregular, with remissions. The disease is of long *duration*, and its rate of progress varies. The patient may be almost moribund, then pick up, become worse again, and so on for years. He may die within 6 months, or after a number of years, or may recover. The cause of death is exhaustion.

VII. **Progressive Ophthalmoplegia** (*Progressive Upper Bulbar Palsy*).—This is a degenerative disease of the nuclei of the motor nerves of the eye, occurring between the ages of 15 and 40. Onset slow. Lead, diphtheria, syphilis, and traumatism are the causes. It occurs in locomotor ataxia, and progressive muscular atrophy.

Symptoms.—Often not noticed until the condition is well advanced. The mobility of the eyeball gradually becomes limited.

Then there is a slight strabismus, usually divergent, and drooping of the eyelids. The eye cannot follow the finger beyond a certain limit. The peculiar expression of the face is known as the "Hutchinson face." Reactions of the pupil to light and accommodation are not usually impaired. There may be diplopia. The *course* of the disease is slow, sometimes almost stationary, except when complicated with progressive muscular atrophy, in which case it is more rapid, and death results in 2 to 3 years, from the latter disease.

ANTERIOR HORN NUCLEI AND LATERAL COLUMN TYPE

VIII. **Amyotrophic Lateral Sclerosis.**—(*Spastic Form of Progressive Muscular Atrophy, or Charcot's Disease*).—Occurs usually between the ages of 35 and 50, more often in women. It is not caused by lead-poisoning, syphilis, or acute infectious diseases, and is considered to be a disease of involution—that is, due to an inherently deficient vitality. Cold, trauma, and shock seem to be precursors in some cases. (See also Fig. 195, page 525.)

Symptoms.—The disease in its typical form combines the symptom complexes of anterior poliomyelitis, spastic spinal paralysis, and bulbar palsy. Usually the initial symptoms are referable to the medulla, but the arms may be primarily affected, less often the legs. There is first a difficulty in swallowing or speaking. There may be an occasional spasm of the tongue, and the lips or cheek may feel stiff. Then the legs and arms become weak and stiff. The progress of the symptoms is slow. There are disturbances of speech, and swallowing is difficult. The arms atrophy and become stiff and rigid, producing characteristic deformities. The reflexes are markedly exaggerated; there is ankle clonus; the arm reflexes are increased, and there is an active jaw-jerk. The jaw is stiff. Slight pain may be present. Anæsthesia and sphincter involvement do not appear until late in the disease. The patient may become quite helpless and be confined to bed within a year, because of the rigidity and contractures in both arms and legs. In some bulbar symptoms are marked.

Diagnosis.—The disease must be distinguished from multiple sclerosis, transverse myelitis, and the other forms of progressive muscular atrophy. The chief points of diagnosis are: the marked and progressive wasting; the rigidity and contractures; the exaggerated reflexes; and the absence of sphincter or sensory disturbances. The disease differs from ordinary bulbar paralysis in that there are stiffness, cramps, increased reflexes, and rigidity in the muscles supplied by the facial, trigeminal, glosso-pharyngeal, 10th, 11th, and 12th nerves. The *prognosis* is always bad. Duration, 2 to 3 years.

DIFFUSE DISEASE OF SPINAL CORD

IX. **Myelitis** (*Inflammation of the Spinal Cord*).—May be acute, subacute, or chronic. When both white and gray matter are affected it is called *diffuse myelitis* and *transverse myelitis*. (See Fig. 194, page 523.) If the periphery is involved the condition is called *marginal* or *annular myelitis*. If the inflammation occurs in isolated parts of the cord it is called *disseminated myelitis*, and when it surrounds the central canal, *periependymal myelitis*.

(I) **Acute Transverse Myelitis.**—This is usually an acute softening of certain parts of the cord followed by secondary degeneration. The lesion commonly involves 1 or 2 inches of the length of the cord, and as it affects both white and gray matter it is often called diffuse myelitis. Disseminated myelitis sometimes occurs in an acute form.

Causes.—It occurs in early adult life, chiefly in males. Predisposing causes are a neuropathic tendency, exposure, and muscular strain. Exciting causes are injuries to the cord, muscular strains, exposure, extension of surrounding inflammation, alcoholism, syphilis, and micro-organisms. Injury and syphilis are commonest.

Symptoms.—Very rarely there are paræsthesias or pain in the back and limbs, or a chill, as prodromal symptoms. At first the feet and legs feel heavy, weak and numb, occasionally with some pain in the back. Spasmodic twitchings in the limbs or even a general convulsion may occur. Walking soon becomes difficult, and the legs begin to feel stiff. A paraplegia develops in a day or so, perhaps within a few hours. There is some anæsthesia. The arms are also paralyzed, sometimes before the legs, if the lesion is in the cervical cord, and the intercostal muscles may be affected. There may be a febrile movement. The sphincters are disturbed, and very early there is retention or incontinence of urine, and constipation. At the height of the disease the legs can be moved slightly, but the patient is unable to walk or stand. The flexors of the feet are more affected than the extensors. There is no pain or tenderness in the legs, but they feel numb and heavy. There is often a girdle sensation at the level of the spinal lesion. Upon the limbs and as high up on the body as the level of the lesion there is anæsthesia to touch, pain, and temperature in varying degrees. At the level of the lesion there is usually a zone of hyperæsthesia. When the anæsthesia is not total, the loss of tactile sensibility is most complete, that of tenderness next, and that of pain least. There is an anæsthesia of the bladder, with retention of urine; also anæsthesia of the rectum and constipation, and the patient does not feel the fæces when his bowels move. The sexual power is abolished when the lesion is in the lumbar cord, but there may be strong erections without the patient's knowledge if the lesion is in the dorsal or cervical region. The bladder may also contract automatically and forcibly expel the urine when the lesion is above the lumbar region. The temperature of the limbs is at first slightly elevated, but later it is subnormal. In a few cases there may be a body temperature of 102° to 104°, under which circumstances the prognosis is bad. On the buttocks and heels bedsores may appear early in the disease. They are due to trophic disturbances and pressure. The skin may be dry, or there may be hyperidrosis. When the lesion is in the lumbar cord the skin and tendon reflexes are diminished, the paralysis is flaccid, the muscles tend to atrophy, and there is R. D. More commonly it is dorsal, and the reflexes become exaggerated; spasms and contractions appear, and deformities result.

The reflexes will be absent if the lesion extends entirely across the cord, although there may be still some excessive muscular tension. The arms are generally affected to a greater extent than the legs when the lesion is in the cervical cord. The pupils may be dilated and unequal because of the involvement of the cilio-spinal centre. If the cervical lesion is extensive, paralysis of the intercostal muscles and disturbance of the action of the heart may ensue.

The disease reaches its height in a few days, or at the most within 2 weeks, remains stationary for some weeks, and then, in non-fatal cases, a gradual improvement begins. Within the first 6 months there is a return of sensation, and within 18 months some return of motion accompanied by spasms and contractures due to a descending degeneration. If the patient has enough strength to walk, there is apt to be some ataxia and also a little anæsthesia left, which will leave him in a condition of "ataxic or spastic paraplegia."

Differential Diagnosis.—This must be made from hemorrhage, acute embolic or thrombotic softening, multiple neuritis, meningitis, acute ascending paralysis, meningeal hemorrhage, and hysterical paralysis. Spinal hemorrhage is sudden in its onset and usually there is no fever. Acute softening is the initial lesion in many cases of acute myelitis and therefore can not be differentiated from it. The onset of multiple neuritis is slower; pain, local tenderness, and sensory disturbances are more severe, and the sphincters are rarely involved. Acute ascending paralysis is progressive, and disturbances of sensation, atrophy, and changes in electrical irritability are absent or slight. Pain and tenderness in the back and limbs are present in meningitis, and there are rigidity, cramps, and slight paralysis, but the bladder is not affected. In hysterical paraplegia there are the various signs of hysteria, little spasm or rigidity, and no marked atrophic or electrical changes. There may be some characteristic changes in sensation, and if the knee-jerks are exaggerated it is but slightly. The diagnosis of the *location* of the lesion is made by studying the various symptoms presented. When the disease goes entirely through the cord the reflexes are absent.

Prognosis.—Depends on the extent and severity of the motor paralysis. If there is no improvement in 6 months, the outlook is unfavourable; so is the presence of bedsores and fever.

(II) **Subacute Myelitis** is that form in which the onset lasts from 2 to 6 weeks. The course and symptoms are otherwise identical with the acute form.

(III) **Chronic Myelitis.**—Usually occurs as a transverse myelitis, perhaps as a disseminated or a diffuse myelitis. Occurs commonly in early and middle adult life, and is more often secondary than primary. It is really only the later stage of acute myelitis, softening, injury, or hemorrhage. Chief causes are shock, alcoholism, exposure, infectious fevers, lead-poisoning, syphilis, and the gouty diathesis.

Symptoms.—In *chronic primary myelitis* the legs feel heavy and tire easily; there are numbness and prickling in the feet; perhaps a feeling of constriction around the waist. The reflexes are exaggerated. The legs stiffen, but atrophy is slight. There are retention of urine and constipation. After a few months the condition is one of partial paraplegia with exaggerated reflexes, rigidity of the legs, anæsthesia, and sometimes slight pain, chiefly in the back. The muscles show little change in electrical excitability, but become somewhat atrophied. Retention of urine becomes marked. The gait is stiff and shuffling. When the paraplegia becomes complete the patient is bedridden, the atrophy increases, contractures occur, the rigidity and anæsthesia increase, and cystitis and nephritis develop. If the arms are involved, weakness, stiffness, some atrophy, anæsthesia, and pain appear.

Chronic secondary myelitis (most common) presents the same symptoms as the primary form, but they are worse at first, then improve, or remain stationary for some time before finally growing worse. The dorso-lumbar regions of the cord are usually affected. In lumbar lesions, the paraplegia, atrophy, and involvement of organic centres are more marked.

Compression myelitis is due to compression of the cord, usually from caries of the vertebræ, tumours, or aneurism. The onset is slow, and the initial symptoms are those of spinal irritation. The final condition is one of spastic paraplegia following weakness, spasms, and contractures.

Differential Diagnosis of Chronic Myelitis.—Must be differentiated from tabes dorsalis, pachymeningitis, spinal tumours, multiple sclerosis, brain palsies, amyotrophic lateral sclerosis, and progressive muscular atrophy. In *tabes dorsalis* there are ataxia and sensory disturbances, with little motor paralysis. In *pachymeningitis* there is a history of injury; the cervical region is usually the seat of the lesion; the pain and anæsthesia are marked, and the sphincters are not involved. The symptoms of *spinal tumours* come on more slowly, are more localized, and there is usually much more pain. In *multiple sclerosis* there are speech disturbances, intention tremor, and nystagmus. In *brain palsies* the paralysis is unilateral, spastic, and without pain or disturbances of the visceral centres. *Progressive muscular atrophy* presents atrophy with no sensory or sphincter disturbance. The *prognosis* is not favourable, although patients may live for twenty years or longer.

X. **Spina Bifida** (*Rhachischisis Posterior*).—Congenital hernia of the spinal membranes, sometimes the cord, through a cleft due to the absence of some of the vertebral arches. Occurs in 1 child out of every 1,200, oftener in females. Hydrocephalus and other developmental defects are often associated.

Forms.—(1) Spinal meningocele—the spinal membranes alone protrude into the sac. (2) Spinal meningo-myelocele—the cord and membranes both protrude. (3) Syringo-myelocele—the fluid is in the central canal, and the inner lining of the sac is formed by the meninges and thinned-out spinal cord.

Symptoms.—Because the lumbar and sacral laminæ are the last to solidify, spina bifida almost always occurs in these regions. Two or three vertebræ are usually involved. The skin is often glossy, tough, thickened, or ulcerated. The tumour may be pedunculated or sessile, and varies in diameter from 1 to 6 inches. The subjects are feeble, badly nourished, and poorly developed mentally. Paraplegia occurs in a great number. In some there is sphincter trouble and anæsthesia. Talipes is frequent. The *diagnosis* is easy. Congenital tumours must be excluded. Important to determine whether the cord is in the sac or not. Marked paraplegia, sphincter troubles, or anæsthesia indicate its presence. Most of the cases die, but the *prognosis* is best for meningocele.

XI. **Spinal Hemorrhage.**—(I) **Spinal Meningeal Hemorrhage.** —*Hæmatorrhachis* (hemorrhage into the membranes of the cord, usually outside the dura) is the most common form. Occurs in the newborn and in adults, in men rather than women. Injuries, fractures of the spine, and falls are the most common exciting causes; also severe convulsions from strychnine poisoning, epilepsy, tetanus, chorea, eclampsia, or severe muscular exertion. More rarely the bursting of an aortic or vertebral aneurism, cerebro-spinal meningitis, or malignant infectious fevers are responsible.

Symptoms.—Slight hemorrhages may be latent. In larger hemorrhages there are sudden severe pains in the back and limbs corresponding to the seat of the lesion, with numbness, tingling, hyperæsthesia, and muscular spasms, chiefly of the back muscles. Very shortly weakness or paralysis, anæsthesia, and disturbance of the visceral centres may follow. The symptoms reach their height in a few hours, followed by slow recovery, or by chronic meningitis.

Diagnosis.—The cardinal points are: a history of injury or childbirth; sudden pain, and symptoms of irritation, which rapidly subside. There is less pain in hæmatomyelia (see (II), following) and injury of the cord, but more paralysis and anæsthesia. In tetanus the symptoms develop more slowly, and trismus is present.

Prognosis.—In severe cases, bad, but if the patient lives 3 or 4 days the prospect is good for a partial or nearly complete recovery.

(II) **Hemorrhage into the Substance of the Cord** (*Hæmatomyelia*).—Not very infrequent. Occurs as a primary condition, usually in males between 20 and 40, sometimes in infants, from vascular disease or purpura hæmorrhagica; or, secondarily, after myelitis and tumours. Overexertion, exposure, injuries, excessive coitus, vascular syphilis, convulsions, old age, and arteriosclerosis are causes.

Symptoms.—Onset rapid. For 1 or 2 hours there may be numbness or weakness, followed by a sudden paraplegia with ataxia or anæsthesia, or both, perhaps with transient loss of consciousness. The sphincters may be paralyzed. At first the reflexes may be absent, but they soon return and become exaggerated. There is often much pain in the back. If the lesion is high up, the thorax and arms are involved. In about 10 days the symptoms subside, and a condition similar to chronic myelitis ensues; otherwise acute myelitis and death follow.

Diagnosis.—Meningeal hemorrhage is more painful, with less paralysis, more spasm, and a better recovery. Sudden onset, absence of fever, and gradual recovery are points of use in the diagnosis. Initial fever suggests myelitis.

Prognosis.—Often serious as to life, always as to health, depending on the location and extent of the lesion; better in dorsal than in cervical hemorrhages.

(III) **Caisson Disease** (*Diver's Paralysis*).—This is a condition of more or less complete paraplegia, occurring in persons who work in compressed air, and is brought about by sudden changes of atmospheric pressure.

Causes.—The disease is therefore found in men who are employed in caissons (whence its name), or who work in deep mines, so deep that the atmospheric pressure is very high; or those who descend to considerable depths in diving bells, or in the ordinary diver's outfit.

The paralytic symptoms, and the accompanying pains, appear upon coming out from the high pressure. The organism apparently accommodates itself to high pressures without much difficulty, but it is the sudden change from a high to a low pressure which is responsible for the occurrence of the disease.

As a means of prevention, in modern engineering practice, several chambers are provided between the high-pressure room and the outer air, in which successive lower pressures are maintained. The workman, having finished his shift, passes from one to another, spending perhaps an hour before he reaches the outside atmosphere. It is found that if these precautions are observed the disease does not occur, or its frequency is reduced to a very small percentage.

Men employed in caissons or diving-bells are usually under a pressure of from 1 to 4 atmospheres, which is equivalent to from 15 to 60 pounds to the square inch. Some men are more susceptible than others. This condition is not usually brought about unless the pressure is greater than 3 atmospheres. Those unused to the work are more apt to be attacked. Small fissures, lacerations, and hemorrhages have been found in the spinal cord. There is a hypothesis that bubbles of oxygen, previously absorbed by the blood under high pressure, are liberated in the substance of the cord; or that emboli, composed of gas, are formed in the capillaries; or that the multiple hemorrhages which are found are due to acute congestion or thrombosis.

Symptoms.—These usually appear immediately after the patient comes out of the caisson, but may not do so for several hours. The most common symptoms are pains of great severity about the joints, the knees and elbows especially, usually without swelling, muscular pain and swelling, and vomiting, with pain in the epigastrium. Less commonly there are vertigo, headache, and paraplegia; rarely hemiplegia and monoplegia. In some cases apoplectiform attacks occur, followed by coma and death within a few hours. The disease lasts from a few hours to several weeks.

Prognosis.—Death may follow in the severest cases; complete recoveries are not infrequent; the paraplegia sometimes disappears, but if organic changes (softening) occur, there is a persistent paraplegia.

XII. **Tumours of the Spinal Cord.**—Comparatively rare, and occur usually between 30 and 50 years, more often in males. Tubercle appears between 15 and 35 years; lipoma is congenital, Predisposing causes are syphilis, tuberculosis, and cancer. Exciting causes are injuries and exposure.

Symptoms.—Depend upon the location, character, size, and rate of growth of the tumour. Pain is early and constant, usually continuous and severe, and ordinarily referred to nerves orginating in the region of the tumour. Girdle sensation, numbness, hyperæsthesia, and, after a time, anæsthesia may follow, with spinal tenderness and rigidity. The sensory symptoms are generally more marked on one side. Subsequently there are spasm, contractures, and exaggerated reflexes involving one leg, or both, or an arm and a leg. Paraplegia, sphincter disturbance, atrophy, bedsores, and death from exhaustion follow. With cervical tumours there may be a gradual involvement of all the extremities and the trunk, cervical rigidity, and optic neuritis. When lower down a hemiparaplegia, or a paraplegia with increased reflexes, occurs. If in the lumbar region the sphincters are early involved and the reflexes are sooner abolished.

Brown-Séquard paralysis is a hemiparaplegia with paralysis of motion and muscle sense on the side of the lesion, and paralysis of cutaneous sensation, especially of pain and temperature, on the opposite side. The reflexes are exaggerated. There is frequently hyperæsthesia on the side of the lesion, with a band of anæsthesia at the level of the tumour. The growth may be outside or inside the dura and the symptoms vary accordingly. Cancer, lipoma, sarcoma, and gumma are the most common *extradural* tumours. In these the sensory and motor irritation is more marked; there is usually some malignant tumour elsewhere; there may be some symptoms of disease of the vertebræ, and the paralysis does not usually take the form of hemiparaplegia. Tubercle and glioma are the common forms of medullary or *intradural* tumours. In these hemiparaplegia is more common, while early in the disease spasm, rigidity, and pain are less common. There may be a secondary myelitis. Spinal tumours occur most frequently just below the mid-cervical, and in the upper and the lower dorsal regions. Hemorrhages, softening, secondary degenerations, and inflammatory reactions may result.

Diagnosis.—Transverse myelitis, caries of the vertebræ, and hypertrophic pachymeningitis must be eliminated. The points in which tumour differs from caries are the absence of any external swelling or kyphosis, or of a tuberculous diathesis, the slight amount of rigidity and tenderness, and the age of the patient. Hypertrophic pachymeningitis frequently can not be distinguished from spinal tumour. From myelitis the disease can be differentiated by the history of early pain, followed by motor and then sensory paralysis, by the localization of the symptoms, and by the progressive course of the disease. In middle life the tumour is probably a sarcoma or a glioma. Tubercle is rare. The average duration of the disease is from 2 to 3 years, but it may last for 5 years. The *prognosis* is bad. Tubercle may become stationary, and syphiloma may be amenable to treatment. Surgery occasionally saves life.

DISEASE IN CENTRAL GRAY SUBSTANCE

XIII. **Syringomyelia.**—A disease of the spinal cord in which there is a development of gliomatous tissue in the central parts with the formation of cavities (Fig. 194, page 523). It is comparatively rare; occurs in men more often than in women, and develops between the ages of 15 and 25. Hand workers are especially affected. Pregnancy, trauma, and infectious diseases seem causative.

Symptoms.—Onset gradual, with paræsthesias in the hands and some aching pains in the neck and arms, followed by muscular wasting of both hands, which increases and extends gradually toward the trunk. There may be fibrillary twitchings and a partial R. D. There is usually a marked anæsthesia of the hands and arms to temperature and pain, but not to touch. When the legs are involved (late in the disease) there is generally spastic paraplegia. There is a scoliosis of the spine, as a rule, in the dorso-lumbar region. Rarely the throat and face are involved. Vasomotor, secretory, and trophic symptoms are marked—e. g., sweating or dryness of the skin; red, congested, or edematous hands; skin eruptions, such as herpes, eczema, and bullæ; painless whitlows, erosions, and ulcerations of the terminal phalanges, and the nails may become dry and brittle and drop off; and arthropathies or spontaneous fractures. The pupils may be unequal. In the late stages of the disease the bladder, rectum, and genital centres are affected, and the medulla becomes involved.

There are 5 types of the disease (DANA): (1) The disease may be latent, with few or non-characteristic symptoms; (2) there may be a period of irritation and pain in the extremities followed by paraplegia, with few sensory troubles, the course suggesting a chronic transverse myelitis or a Brown-Séquard paralysis; (3) a type in which bulbar symptoms develop early, but differing from ordinary bulbar paralysis in the involvement of the trigeminus and other cranial nerves not commonly attacked; (4) a form characterized by a rather rapid ascending paralysis; and (5) a type characterized by the symptoms of muscular atrophy with analgesia and felons (Morvan's disease).

Diagnosis.—The distinguishing points are: its beginning during adolescence, the progressive muscular atrophy combined with the peculiar dissociated disturbances of sensibility, and the scoliosis and trophic disturbances. It must be distinguished from progressive muscular atrophy and dystrophy, hypertrophic cervical pachymeningitis, amyotrophic lateral sclerosis, chronic transverse myelitis, anæsthetic leprosy, and Morvan's disease. In progressive muscular atrophy there are no sensory or trophic disturbances, or scoliosis. The disease can not often be differentiated from Morvan's disease, although whitlows are rare in the ordinary forms of syringomyelia. Morvan's disease begins in one hand and slowly extends to the other, and there is generally loss of tactile as well as thermic and pain sense. Dissociation of the sensory symptoms is not present in leprosy, and the anæsthesia is found along the course of the nerves or in sharply defined patches. The *prognosis* is bad. The duration of the disease is, however, from 5 to 20 years, with periods of remission.

IV. SYSTEMIC CORD DISEASES

POSTERIOR COLUMN GROUP

I. Locomotor Ataxia (*Posterior Spinal Sclerosis, Tabes Dorsalis*). —A chronic progressive disease in which primarily the posterior spinal ganglia or analogous neurones are involved. (Figs. 194, p. 523; 225, p. 576.)

There is a common, a paralytic, and a neuralgic form, and one in which optic atrophy is one of the first symptoms. It may also be complicated with muscular atrophy, general paralysis, and other scleroses.

Causes.—Occurs between the ages of 30 and 50, oftenest between 30 and 40, perhaps as early as 10 or as late as 60, more often in males. Exposure to cold and wet, combined with muscular exertion and excessive railroad travelling, dancing with exposure, or sexual intercourse, are important causes. Heredity is indirect only, and very unimportant. The most important single factor is syphilis, which does not act directly, but if followed by excesses and exposure tends to produce the disease. A syphilitic history is found in from 60 to 90 per cent of all cases. Acute infectious diseases, depressing emotions, prolonged lactation, difficult labour with hemorrhage, excessive smoking, and injuries with shock are also influential.

Symptoms.—Locomotor ataxia may be divided into 3 stages: the initial or *pre-ataxic*, the *ataxic*, and the *paralytic*. In actual practice the cases are very variable.

(1) The *initial stage* may last for a few months or several years. Neuralgia-like pains often precede by two or three years. First noticed are slight uncertainty in walking (especially in the dark), occasional darting pains in the legs or rectum, sensations of numbness in the feet, and attacks of double vision and vertigo. Vesical control is impaired and sexual power diminished. The knee-jerk is lost. The patient feels profoundly fatigued without exertion. Areas of tactile anæsthesia may be discovered on the trunk.

(2) The *ataxic stage* lasts for several years. The patient's unsteadiness increases, so that, when walking, he uses a cane and watches the ground and his feet. He cannot stand with eyes closed without swaying or perhaps falling. The numbness in his feet is

such that he feels as if walking on a thick carpet. There are areas of anæsthesia on the legs or the feet and toes. The leg pains are lightning-like and occur in paroxysms. Sexual power may be lost. Vesical control is impaired, and there are constipation, girdle sensation, attacks of epigastric pain with vomiting, and perhaps a causeless diarrhœa. The pupils become small or unequal and react to accommodation, but not to light. Later the ataxia, pain, and anæsthesia may affect the arms.

(3) The *paralytic stage*. The power of walking is entirely lost, although the muscular strength of the legs is fairly good. With closed eyes the patient does not know where his legs are, the ataxia and anæsthesia are marked, and he does not feel a touch or the prick of a pin. The ataxia, anæsthesia, and pains in his arms have increased, but never become as bad as in the legs. The pains, although present, are not as severe. The bladder is paretic, anæsthetic, and has to be catheterized. Mental involvement may or may not be present.

Description of Symptoms.—Ataxia, both static and motor, is present very early in a moderate degree, due to a beginning anæsthesia of the joints and tendons. The knee-jerk is early lost—an important diagnostic sign. The station (III, page 35) and gait ((1), page 36) are characteristic; so also are pains of a lightning or lancinating type, usually in the legs, along the course of the nerves, or in small areas on the leg, foot, or thigh, so sudden and severe that the patient involuntarily jumps or jerks the limb. An early symptom may be a severe rectal neuralgia. The pains may be nearly continuous, or may occur every few days or only once or twice a month. Sometimes they are entirely absent for several months, only to return with renewed vigour, but usually persist throughout the disease.

Areas of tactile anæsthesia on one or both sides of the chest, and in the back at the same level, are early and diagnostic symptoms. Anæsthesia affects first the feet, then the legs and thighs, fingers and hands, later extending from the thighs to the trunk. The anæsthesia is greatest to pain, but also affects the tactile and temperature senses. There are often delayed sensation and polyæsthesia.

Optic atrophy (6 per cent of all cases, more often in left eye) appears in the pre-ataxic stage, and if the patient has reached the second stage without it, he will probably escape it altogether. It begins with flashes of light, *muscæ volitantes*, and increased sensitiveness to light. Vision fails, and there is often a disturbance of colour sense, and always an irregular contraction of the field of vision. The atrophy progresses until in about 3 years blindness results. Disorders of hearing are frequent, usually due to middle-ear disease.

The eye muscles are nearly always implicated. The pupils are small, sometimes uneven, and react to accommodation but not to light (Argyll-Robertson pupil). At the beginning the light reaction may be merely sluggish. The ocular skin reflex is early abolished. Sometimes light and accommodation reflexes are both lost, more frequently in exudative brain syphilis.

Not infrequently there is early a slight drooping of one or both lids—sympathetic nerve ptosis. Of the external eye muscles those most often affected are the external rectus, levator palpebræ and internal rectus, one or several. These paralyses occur most often in syphilitic cases, and may be transitory or permanent. Early paralyses are usually transitory.

Arthropathies (especially) and spontaneous fractures of bones occur in from 5 to 10 per cent of all cases. The knees, ankles, and hips are most often affected, but the shoulder, elbow, wrist, and finger joints may be involved. Fractures occur oftenest in the shaft and neck of the femur. In arthropathies a sudden painless swelling of the joint develops in from 24 to 48 hours. An osseous hyperplasia of the joint occurs and it becomes much enlarged. If the swelling is due simply to synovial distention and enlargement of bones, as in some mild cases, it soon goes down and the joint returns to nearly its natural size. In more severe cases the disturbance progresses, the ligaments become relaxed, the bones of the joint are freely movable, luxations are easily produced, and the limb is almost or quite useless. Arthropathies may occur in the early stages and often are not recognised.

Spontaneous fractures are usually due to a slight fall or injury, but violent muscular efforts may cause them. They are painless, and often heal well, sometimes with unusual rapidity.

Trophic disturbances of the skin are numerous and usually appear late. Herpes and lichen are the most common. A round perforating ulcer may form on the sole of the foot, sometimes following the cutting of a corn. Teeth and nails may fall out in rare cases.

"Crises" of various kinds occur in tabes. Gastric crises are the most common. They consist of attacks of severe epigastric pain with vomiting, sometimes diarrhœa, and may last for 2 or 3 days. Laryngeal crises are sudden attacks of spasm of the adductors or paralysis of abductors, with noisy, croupy respirations, cough and struggling for breath. Vertigo may be present and cause the patient to fall. The pulse may be very rapid. The attack lasts from a few minutes to several hours, and is distressing but not dangerous. Simple aphonia may be present. Cardiac crises consist of a sudden dyspnœa with rapid heart action and a sense of suffocation. Great weariness and heaviness in the limbs is an early, characteristic, and constant symptom.

Muscular atrophies sometimes occur. There may be a true progressive muscular atrophy, with ophthalmoplegia, bulbar paralysis, and spinal amyotrophy; localized muscular atrophies, with wasting of certain groups of muscles in arms or legs; or general wasting. Hemiplegia and acute paraplegia are found rarely. Cerebral symptoms sometimes occur in tabes, mainly insomnia and vertigo.

The *course* of the disease is not always progressive. Its duration varies from 1 to 30 years. The 1st and 2d stages may last between 5 and 20 years each.

Diagnosis.—The cardinal points are: loss of knee-jerks; lightning pains; Romberg symptom and ataxic gait; Argyll-Robertson pupil; numbness of the feet; a history of syphilis; and the slow onset of the disease. Lightning pains, loss of knee-jerks, and Argyll-Robertson pupils are usually sufficient to assure a diagnosis. The disease in the first stage must be distinguished from multiple neuritis, hereditary ataxia, spinal syphilis, spinal tumour, chronic myelitis, neurasthenia, and general paresis.

Prognosis.—In a few cases the disease may be stopped in the 1st stage and the patient may get practically well. After the 2d stage a cure is impossible, but the patient may be made comfortable and his condition somewhat improved. Death is rarely due to the disease or its " crises."

ANTERO-LATERAL COLUMN GROUP

II. Spastic Spinal Paralysis (*Lateral Spinal Sclerosis*).—Two varieties are here considered: Little's disease and Erb's syphilitic form.

Little's Disease.—A spastic spinal paralysis. Always congenital, presumably due to developmental defects in the pyramidal tracts of the cord (Fig. 194, page 523). It may be hereditary.

In family types the disease may not appear until the 5th year or even later, but is usually manifested within the 1st year. The *symptoms* are those of ordinary cerebral diplegia or birth palsy, except that there are no marked mental defects; the child is not microcephalic and idiotic; there are no hydrocephalus, cranial nerve palsies, or epilepsy. Children suffering from this disease walk " cross-legged," the legs crossing one in front of the other. The legs are more affected than the arms. The facial and throat muscles may be involved. Pain is absent. Occasionally the disability increases as the child grows older, rigidity and contractures occurring in the legs and arms. At puberty mental impairment and epilepsy may appear.

The absence of epilepsy, microcephalus, and mental defects distinguishes the disorder from cerebral diplegia. The involvement of the arms and the absence of pain and sphincter disturbance are the points in which it differs from myelitis due to compression. Hereditary spastic paraplegia makes its appearance at about the 5th year, is found in succeeding generations of a family, and involves chiefly the legs. In mild cases the patient may learn to walk and use his hands, and may slowly improve and live to a good age.

Syphilitic Spastic Spinal Paralysis.—This disease, differentiated by Erb from lateral sclerosis, is (W. Browning) of much more common occurrence than the primary form. Its onset is slow and usually within 5 years of the initial lesion. The majority of cases develop between 20 and 40 years of age.

The symptoms are the gradual oncoming of rigidity and stiffness of the muscles of the legs, with cramps, muscular twitchings, tremor, increased reflexes, and ankle clonus. The gait is dragging, shuffling, and cross-legged (see page 1080). The muscles are in a state of tension and resist passive movement. The Babinski reflex (page 572) is an early symptom. The disease may at first be unilateral, but soon involves both lower extremities. It affects primarily the muscles of the thigh and leg, later those of the hip. The patient may be finally confined to bed, and there are contractures of the affected muscles whereby the knees are drawn up to the abdomen and the heels to the buttocks, resisting all efforts at extension. Muscular atrophy from disuse follows. The upper extremities are often not involved, but may become affected toward the latter part of the disease. This form of the malady, as distinguished from Little's disease and other spastic paraplegias, is characterized by the presence of rectal and bladder disturbances, and occasionally by pain and other disorders of sensation. It is an essentially chronic disorder extending over many years, and is usually progressive. It is at times arrested or much ameliorated by treatment.

POSTERIOR AND LATERAL COLUMN GROUP

III. The Combined Scleroses.—This term relates to certain forms of degenerative sclerosis in which both the posterior and lateral columns of the spinal cord (Fig. 194, page 523) are involved.

The extent of the sclerosis (Putnam, Starr) depends largely upon the duration of the disease. In some instances the lateral columns are first and more deeply involved; in others the posterior; in time the entire length of both columns.

The combined scleroses have been studied in this country, especially by Putnam and Dana, and particularly with reference to the variety of the disease which occurs in profoundly anæmic and toxic conditions.

Of all the combined scleroses, there are, according to Dana, but three which possess a practical clinical interest. They are, in his classification, as follows:

1. Combined scleroses of profoundly anæmic and cachectic or toxic states (Putnam's type).
2. Hereditary spinal ataxia (FRIEDREICH'S).
3. Hereditary ataxic paraplegia.

(1) *The Combined Scleroses of Pernicious Anæmia and Cachectic States* (*Putnam's Type*).—Occurs more often in women between the ages of 45 and 65. A neuropathic constitution predisposes. Active causes are severe malarial toxæmia, pernicious anæmia and its factors, disease of the suprarenal capsule, and perhaps lead-poisoning.

Symptoms.—The initial symptom is usually numbness in the extremities, followed by increasing weakness, and, finally, paraplegia. Emaciation and anæmia are marked, and obstinate diarrhœa may be present. There is no paralysis until the paraplegia appears. The common symptoms are spasticity, exaggerated knee-jerk, ankle clonus, perhaps ataxia, and some anæsthesia. Girdle sensation and lightning pains are rare. The arms are involved, but not so much as the legs. Speech, vision, and other special senses are not affected. The sphincters are not involved until late. Finally there may be some mental disturbance. In *diagnosis* the cardinal points are: the age; the presence of profound anæmia and perhaps of a malarial history; paræsthesia; slight ataxia; marked and progressive weakness and emaciation; obstinate diarrhœa and rather sudden paraplegia. The *prognosis* is not good.

(2) *Hereditary Spinal Ataxia* (*Friedreich's Ataxia*).—The chief predisposing cause is an inherited lack of development of the cord, more especially of its posterior columns and pyramidal tracts (Fig. 194, page 523). Other predisposing causes are neuroses, syphilis, and habitual intemperance in the parents. The disease develops between 6 and 15 years of age, sometimes earlier or later, usually about the 12th year, more often in males, in children of the labouring and farming classes, and seems to follow the infectious fevers.

Symptoms.—There is at first some ataxia and weakness in the legs, which gradually increases and after 5 or 6 years extends to the arms. Walking is seriously interfered with, and movements of the arms are impaired. Within the first year the knee-jerks are abolished. There may be bulbar symptoms, such as thick or scanning speech, and frequently nystagmus. Headache and vertigo are often present. There are no vesical or rectal disturbances. Talipes varus, dorsal flexion of the toes, or some other deformity of the foot, and lateral curvature of the spine often appear. The legs become weaker, until finally paraplegia, with contractures and atrophy, begins. Oscillation of the head and choreiform or inco-ordinate movements of the extremities may appear. The progress of the disease is slow. Less common symptoms are: tremors, spasms, decreased electrical irritability, vasomotor paresis, polyuria, muscular atrophy, glycosuria, fibrillary tremor, attacks of choking, anæsthesia, ptyalism, diplopia, strabismus, blepharospasm, ptosis, sluggish pupils, impotence, tachycardia, incontinence of urine, fragilitas ossium, and profuse sweats.

Differential Diagnosis.—The cardinal points are: ataxia, beginning in legs and extending to arms and tongue; peculiar rolling, ataxic gait; disturbance of speech; spinal curvatures and talipes, gradual development of paraplegia; loss of knee-jerk with Babinski's sign; absence of cutaneous anæsthesia, bladder disturbance, severe pains, and eye troubles, except nystagmus; and the development of the symptoms at about puberty.

Prognosis.—Although progressive, the disease may be stationary for a long time. Its average duration is between 15 and 20 years.

(3) *Hereditary Ataxic Paraplegia.*—Occurs in females between the ages of 12 and 16 (DANA). The predisposing cause is a neurotic heredity. No exciting cause is known. The ***symptoms*** are weakness and stiffness of the legs with marked ataxia, occasionally more cerebellar than spinal. The reflexes are much exaggerated, with ankle clonus, and some paræsthesia, but no pains, painful spasms, or anæsthesia. The arms may be slightly, but the legs are chiefly, involved. The face, cranial nerves, and sphincters escape. The health is otherwise good. The ***diagnosis*** must be made from the gradual onset and slow progress of the disease, the age at the onset, the hereditary and family history, the ataxia, and the paraplegia. The *course* is slow and long, and, as far as life is concerned, favourable.

V. INFLAMMATION OF THE SPINAL MENINGES

(I) **External Meningitis** (*Pachymeningitis Externa*).—Affecting outer surface of dura mater. Rare and nearly always secondary to tuberculosis, caries of spine, psoas abscess, sacral bedsores, peritonitis, pyæmia, or purulent pleurisy.

Symptoms.—Local pain in back, radiating pains, tenderness, hyperæsthesia, paresis, twitching, paraplegia, exaggeration of reflexes, sphincter involvement, and sometimes anæsthesia.

Diagnosis.—Radiating pains, tenderness, kyphosis, and the presence of the local disease, with the motor and sensory irritation and paralysis, are the diagnostic points. ***Prognosis*** usually bad.

(II) **Internal Spinal Meningitis** (*Pachymeningitis Interna*).—Affecting inner surface of dura mater. Two varieties are recognised, hemorrhagic and hypertrophic. Usually occurs in adults, sometimes in children, more often in males. Exciting causes are alcoholism, exposure, syphilis, and trauma (most important).

Symptoms.—Pain (shooting to occiput and back) and stiffness in the neck, with numbness, prickling, pain, and perhaps stiffness and cramps, in the arms, generally more in one than the other, and worse at night. There may be nausea and vomiting. After 6 months paralysis begins, with weakness, atrophy, rigidity, and contractures in the arms. Anæsthesia, hyperæsthesia, and trophic changes occur. Paraplegia follows, with rigidity and increased reflexes, and the patient finally dies of exhaustion. *Diagnosis* must be made from tumour, Pott's disease, myelitis, wryneck, and progressive muscular atrophy. The important points are: history of injury; slow progressive course; the localization of the symptoms and their bilateral character; and pain. The majority of cases die. Some are cured, others remain stationary for long periods.

(III) **Acute Spinal Leptomeningitis** (*Inflammation of the Pia Mater of the Spinal Cord*).—Frequently occurs in connection with disease of the cerebral pia mater, seldom alone. Children are most often affected, and among adults, men. Alcohol predisposes. Always secondary to an infection, with or without traumatism. Exciting causes are tuberculosis, syphilis, typhoid fever, rheumatism, insolation, exposure, and surgical operations.

Symptoms.—Pain in the back and along the nerves, with some fever and an initial chill. The pain increases, and there is dorsal tenderness, and rigidity of the muscles of the back, sometimes amounting to opisthotonus. There is constipation, occasional retention of urine, and hyperæsthesia of the skin. The reflexes at first are increased. Paralysis comes on after a time, and there are retention of urine, anæsthesia, and atrophy. The weakness increases, bedsores may occur, and death from exhaustion ensue. The symptoms come on more slowly in the tuberculous form, and with greater severity in the septic form.

Differential Diagnosis.—This must be made from myelitis, rabies, tetanus, gonorrhœal rheumatism, rheumatism of the dorsal muscles, and strychnine poisoning. In myelitis there is not much pain and a great deal of paralysis. In tetanus there is trismus, no fever, and a history of trauma.

Prognosis.—Not good, especially in cases with high fever, severe pains, and early paralysis, and in tuberculous meningitis. The acute form may subside and become chronic.

(IV) Chronic Spinal Leptomeningitis and Meningo-Myelitis.—Rare, always secondary, generally to cerebro-spinal meningitis, syphilis, or chronic alcoholism. Occurs oftenest in male adults.

The *symptoms* are the same as those of the disease to which it is secondary. Pain in back, radiating to trunk and limbs, tenderness and stiffness of spine, twitching and spasms in limbs with weakness, and later, paralysis, atrophy, anæsthesia, and weakness of the bladder, are the usual symptoms. The course of the disease is irregular.

The *differential diagnosis* must be made from tabes dorsalis, myelitis, spinal irritation, vertebral caries, and tetanus. In tabes dorsalis the knee-jerk is absent and there is ataxia, but no paralysis and no tenderness along the spine. In vertebral caries there is deformity, the pain and tenderness is more localized, and there is spasmodic fixation of the trunk. In spinal irritation there is neurasthenia or hysteria, and no rigidity, radiating pains, twitching, atrophy, or paralysis. The disease is seldom fatal.

VI. INFLAMMATION OF THE MEMBRANES OF THE BRAIN

(I) Serous Meningitis (*Alcoholic Meningitis*, "*Wet Brain*").—An acute toxæmia of the brain rather than a true meningitis. Occurs most frequently in males, usually after 8 or 10 years of drinking, and between the ages of 30 and 40. The persistent use of chloral, cocaine, or morphine may lead to the same condition. The exciting cause is usually a continuous drinking spell of 2 or 3 weeks, ending perhaps in delirium tremens.

Symptoms.—If the patient has had delirium tremens, he sinks, after 2 or 3 days, into a semi-coma with muttering delirium, delusions, and hallucinations. The temperature is normal, or slightly above, with rapid pulse. There is hyperæsthesia of the skin, and pain upon pressure on the muscles of the arms, legs, or abdomen. The pupils are small. In a few days the stupor becomes more complete and the patient hardly can be aroused. Arms, legs, and neck are stiff, and the latter is somewhat retracted, and painful if moved. The reflexes are exaggerated, and the abdominal walls retracted. The eyelids are closed, the pupils small and do not react well to light. The tongue is dry and coated. There may be involuntary evacuations of the rectum and bladder. The extremities become cold and stiff, the pulse fast and weak. The coma deepens, and the fever may rise to 104°. Pneumonia may be present near the end. Some cases do not go into this last stage, but improve and recover.

Diagnosis.—Must be distinguished from acute encephalitis, ordinary suppurative meningitis, and acute serous meningitis caused by infection, but, as the symptoms of all these are very similar, the alcoholic history usually decides the diagnosis. *Prognosis* bad after marked rigidity and coma have begun. In most cases when the neck is stiff the patient dies; if not, the prognosis is better.

(II) **Inflammation of the Dura Mater** (*Pachymeningitis Externa*).—Common causes are injuries, caries of the petrous bone in mastoid disease, or the ethmoid bone in ozena, necrosis, erysipelas, and syphilis. The disease may be acute or subacute. The *symptoms* are fever, local headache, delirium, and occasionally convulsions and paralysis. The *diagnosis* is made by finding the local cause.

(III) **Inflammation of the Pia Mater** (*Leptomeningitis*).—The following varieties are recognised:

(1) *Acute Simple Leptomeningitis.*—Occurs more frequently in males, especially in the young. Trauma and acute alcoholism predispose. The disease is always the result of an infection, reaching the membranes usually from without, sometimes through the blood. Disease of the middle ear and mastoid cells is the most frequent cause. Others are operations upon or disease of the frontal sinuses and upper nasal passages; disease, injuries, and fractures of the cranial bones; pneumonia, septicæmia, pyæmia, scarlet fever, variola, rheumatism, empyema, typhoid fever, mumps, measles, and, more rarely, endocarditis and brain abscess. Sunstroke alone does not seem to be a potent factor.

Symptoms.—Prodromal symptoms are languor, malaise, headache (most prominent), irritability, vertigo, loss of appetite, and vomiting. In the second or *irritative* stage, headache, delirium, rigidity of the neck, hyperæsthesia of the skin, vomiting, retraction of the abdomen, irregular fever, contracted and often unequal pupils, and occasionally optic neuritis or retinitis are the chief symptoms. Headache is persistent with severe exacerbations. There is an early muttering delirium, with perhaps alternating stupor and violence. Vomiting, when present, is of an explosive (projectile) character. The neck is retracted and rigid. General rigidity, resembling catalepsy, may appear. If the skin is scratched a red line appears (*tache cérébrale*). The abdomen is "boat-shaped." There is photophobia, usually with contracted and uneven pupils. Convulsions and cranial-nerve paralysis (ptosis, strabismus, facial palsy) may ensue. Respiration is rapid and uneven; the pulse usually arrhythmic or intermittent (50 to 70). There is irregular fever (101° to 103°), with constipation and oliguria (sometimes albuminuria).

In the *paralytic* stage there is stupor or coma. Some rigidity persists, so also the scaphoid abdomen. The pupils may become dilated, the skin is moist, and urine and stools may be passed involuntarily. Usually death occurs within a day or so after this. The disease generally lasts from 1 to 2 weeks, but it may begin suddenly with coma and the patient die within 1 or 2 days.

Diagnosis.—The symptoms and the history of the exciting cause usually make the diagnosis easy. The chief difficulty is to distinguish it from cerebro-spinal fever or tuberculous meningitis. The *prognosis* is bad, not so serious as in tuberculous meningitis, more so than in cerebro-spinal meningitis.

(2) *Epidemic Cerebro-spinal Meningitis* (*Spotted Fever, Cerebro-spinal Fever*).—See page 737.

(3) *Tuberculous Meningitis.*—See page 788.

VII. DISEASES OF THE CEREBRAL SUBSTANCE

I. Apoplexy from Intracranial Hemorrhage (*Cerebral Hemorrhage, Hemiplegia*).—There may be: dural or pachymeningeal; pial or subarachnoid; or central hemorrhages; and hemorrhage into the medulla, pons, and cerebellum.

(1) Central Hemorrhage.—Most common, and due to rupture of the blood vessels supplying the internal capsule, the great basal ganglia, and the white matter. Occurs somewhat more frequently in males, and, in four fifths of all cases, after the age of 40. The predisposition increases yearly from 40 to 80.

Heredity, marasmic conditions, infective fevers, chronic alcoholism, chronic kidney disease, gout, rheumatism, and syphilis predispose. Heart and arterial disease and miliary aneurisms are determining causes. Other factors are scurvy, purpura, leucocythæmia, and the apoplectic habit. Exciting causes are sudden physical exertion, coitus, passion or excitement, excessive eating and drinking, straining at stool, or a cold bath.

Symptoms.—The discussion of the symptoms of apoplexy involves a complicated series of anatomical data. Only the more ordinary classical types are here touched. Very rarely, except in syphilitic cases, there are prodromal symptoms, vertigo, "full" feelings or pain in the head, numbness of one hand and foot, loss of memory for words, and bad dreams. The heart action may be irregular and nose-bleed may occur. The attack is sudden with convulsions and coma; coma alone; or no loss of consciousness at all. Convulsions rarely occur with the attack; are unilateral or partial, but may be general (due to meningeal hemorrhage).

The attack usually begins with sudden vertigo and unconsciousness (see Coma from Apoplexy, page 77). Retention or incontinence of urine and fæces may occur. The sp. g. of the urine is high, and it may contain albumin. In severe cases the temperature may be subnormal during the first 12 hours. Usually the temperature of the paralyzed side exceeds that of the other by 1° or 2°. In rapidly fatal cases the coma persists, the pulse increases in rapidity, there is Cheyne-Stokes breathing, the temperature rises to 102° or 103° until shortly before death, when it may fall again, speech and swallowing become difficult, hypostatic pneumonia develops, and the patient dies in from 2 to 4 days. There are fatal cases which run a slower course of 2 to 3 weeks; in these consciousness is partially regained, and the patient is stuporous or mildly delirious, with restlessness and headaches. The temperature remains normal, until after 2 or 3 weeks, when it rises, pneumonia sets in, the patient becomes unconscious and dies.

The majority of cases are not fatal. In these the coma disappears within 5 or 6 hours, the patient is weak and may be mentally confused, with some disturbances of speech. The condition is now one of hemiplegia, the arm and leg being affected most, the face least. Of the facial nerve only the lower 2 branches are involved and the eyes can be closed. The tongue protrudes toward the affected side. Anæsthesia is sometimes present on the paralyzed side, and occasionally there may be hemianopia and disturbances of hearing. According to the site of the lesion there may be motor or sensory aphasia (page 269). The paralyzed limbs are at first usually flaccid, but rigidity may begin early. The temperature usually rises to 100° or 102° on the 2d or 3d day, and then gradually falls to the normal about the 10th day. A continued rise of temperature after the 4th or 5th day indicates an inflammatory reaction or more hemorrhage.

The *chronic stage* begins when the fever and signs of cerebral irritation have disappeared, usually in about 4 weeks. The leg and arm can be moved somewhat, the sensory symptoms are less marked, the mind is clear, and there is no headache. The face is least affected, the leg next, and the arm most.

The rigidity, beginning about the 2d week, gradually increases until there are contractures of the affected limbs, involving, in the foot, the extensors more than the flexors. For the gait, see (4), page 37. The shoulder is adducted and the forearm flexed, and the fingers are flexed into the palm. The face muscles are slightly contracted and draw to the affected side. There is no muscular atrophy.

The reflexes, at first diminished or absent, reappear, the knee, elbow, and wrist jerks are much exaggerated, and there is ankle clonus and Babinski. Electrical irritability is unaltered. In the paralyzed limbs there may be tremor, ataxia, associated, continuous or athetoid, choreic, and spastic movements; also burning, cramping pains, joint affections, vasomotor disturbances, sweating, and skin eruptions. Paræsthesias are common. The patient cries easily, is irritable, and, in general, emotional, and his memory and power of attention are affected. Epilepsy and insanity may develop.

(2) Meningeal Apoplexy.—Due to rupture of the middle meningeal artery, or vein, or their branches. Obstetrical and other injuries to the head, insanity, and alcoholism are causes. Distinct cerebral symptoms usually appear within a few hours after the accident, but may be delayed for periods of a few hours to 2 months. Partial or complete hemiplegia appears on the side opposite the clot, with increased reflexes, some rigidity, and irregular spasmodic movements of the muscles of the affected side. The pupils are contracted and unequal. The eyes usually look toward the affected side, i. e., away from the lesion. Stertorous breathing is rare, the pulse is full and slow, and there may be aphasia. The temperature may be normal or several degrees above. The breathing becomes stertorous, the pulse rapid and feeble, and death ensues.

(3) Pial Apoplexy.—Rare, and usually caused by trauma, associated with alcoholism and syphilis. There is a sudden incomplete hemiplegia with spasmodic movements.

(4) Pons Apoplexy.—There is a sudden loss of consciousness, occasionally with spasmodic movements of the limbs. There is rigidity on both sides of the body, and both pupils are minutely contracted. Temperature always rises, perhaps to 104°. The facial or ocular nerves may be involved, with some hemiplegia.

(5) Cerebellar Apoplexy.—There may be a headache for several days, or a sudden coma may occur with stertorous breathing, perhaps vomiting, and hemiplegia. The hemiplegia is on the side of the lesion. The respiration is especially affected. Death is inevitable.

Diagnosis of Hemorrhagic Apoplexy.—To be distinguished from uræmic (page 77), opium (page 76), or alcoholic (page 76) coma, epilepsy (page 78), hysteria (page 78), and acute softening from embolus and thrombus. Following are the distinguishing points between hemorrhage and acute softening (DANA):

HEMORRHAGE—Age, 30 to 50	ACUTE SOFTENING—Earlier (in embolism) or later age (in thrombus)
History of arterial disease in family.	History of infectious disease or syphilis.
Sudden onset, with coma and paralysis occurring together, the coma deepening.	Premonitory symptoms and more gradual onset (in thrombus), more transitory coma or absence of coma.
Initial and early rigidity.	Initial convulsive movements.
Very unequal pupils.	Presence of weak heart (in thrombus).
Stertorous breathing and hard, slow pulse.	Presence of endocarditis (in embolism).
Peculiar alternating conjugate deviation.	Slight hemiplegia with anæsthesia.
Early rigidity.	The puerperal state (in embolism).
Peculiar disturbances of temperature.	

Prognosis of Hemorrhagic Apoplexy.—Most cases recover, seldom completely, from the first attack. A 2d attack is liable to occur within 5 years, from which the minority recover. Later attacks fatal.

II. **Acute Softening of the Brain** (*Embolism, Thrombosis*).—Thrombosis is more frequent in men, embolism in women. Embolism usually occurs between the ages of 20 and 50, rarely in children; thrombosis between 50 and 70. The predisposing causes in *embolism* are infectious fevers, acute or recurrent endocarditis, blood dyscrasias, pregnancy, and profound anæmia; in *thrombosis*, fatty heart, blood dyscrasias, and syphilitic, lead, or gouty arteritis.

Symptoms.—In *embolism* the onset is sudden, with convulsive twitchings, then hemiplegia, and a *temporary* loss of consciousness. Fever develops after a few days.

In *thrombosis* prodromal symptoms are frequent. There may be cranial-nerve paralyses and headaches when syphilis is present; in other cases drowsiness, numbness in the hand and foot, transient hemiplegia, vertigo, and temporary aphasia. The onset is slow, the hemiplegia taking hours to become complete, during which time the patient gradually becomes comatose; sometimes more sudden, without loss of consciousness; or it may come on during sleep. There may be an initial fall in temperature followed by a rise.

The hemiplegia due to embolus or thrombosis is apt to improve rapidly within a few days or weeks, but the chronic stage resembles that of hemorrhage. There is usually more mental impairment in thrombosis than in embolus.

Diagnosis.—Distinguished from hemorrhage by a syphilitic history, and the earlier or later age of its occurrence; by the presence (in thrombosis) of a more gradual onset, premonitory symptoms, and a weak heart, and (in embolism) of endocarditis, the puerperal state, initial convulsive movements, and slight hemiplegia with anæsthesia. The *prognosis* is better than in hemorrhage, and the recovery is more complete.

III. **Polio-encephalitis** (*Acute Exudative Encephalitis of the Gray Matter*).—The symptoms are those of *acute* glosso-labio-laryngeal palsy (page 1047), or of ophthalmoplegia (pages 214, 218, and 1049), according as the disease is inferior or superior.

IV. **Hæmorrhagic Encephalitis** (*Acute Exudative Encephalitis with Hemorrhage*).—Occurs most frequently in females, and, when due to infection, usually under the age of 20. Exciting causes are infectious fevers, such as cerebro-spinal meningitis, typhus, and typhoid, influenza, and malignant endocarditis; the puerperal state, sunstroke, and acute alcoholism.

Symptoms.—Onset sudden, with severe headache, and fever (perhaps to 105°). Vomiting, vertigo, photophobia, and delirium may be present, followed by semi-coma or stupor. The pulse is rapid and weak, breathing shallow and frequent, deep reflexes diminished, the sphincters perhaps involved. After a few days or 2 or 3 weeks the coma may change to restlessness and irritation, or the patient may gradually improve and eventually recover. In the early stage there may be epileptoid convulsions, hemiplegia, paralysis of an arm or leg, aphasia, hemiataxia, hemianopia, impairment of speech and deglutition, eye palsy, nystagmus, or optic neuritis.

Diagnosis.—Must be distinguished from meningitis by the sudden onset with coma; the absence of stiff neck, pinhole pupils, rigidity of the limbs, projectile vomiting, and hyperæsthesia; and the presence of local paralysis or hemiplegia, or the occurrence of an epileptic attack.

Prognosis.—Serious, but patients often recover. Mild cases run a course of 2 to 3 weeks; in severe cases the patient soon dies of exhaustion, but the disease may last for weeks or months.

V. **Abscess of the Brain** (*Acute Suppurative Encephalitis*).—Occurs between the ages of 1 and 50, especially between 10 and 30. More frequent in males. Due primarily to microbic infection, although the microbe and mode of entrance vary. Chief exciting causes are disease of the nose, ear, or cranial bones, suppurative processes in general, injuries, tumours, and infectious fevers. The most common cause is chronic disease of the middle and internal ear, especially when the mastoid cells and tympanum are affected; next are injuries or chronic disease of the cranial bones, empyema, tuberculosis of the lungs, fetid bronchitis, pyæmia, typhus and typhoid fevers, smallpox, diphtheria, influenza, and erysipelas.

Symptoms.—In *acute cases* the onset is rapid, and the symptoms may be divided into 3 groups: 1. Those due to pressure: vomiting, persistent and severe headache, vertigo, mental dulness, perhaps delirium, and finally coma. The temperature and pulse vary, but are both usually normal or subnormal. The pupils may be irregular, and optic neuritis may occur. 2. Toxic symptoms, as in any septic poisoning, i. e., anorexia, emaciation, prostration, irregular fever, and mental and sensory disturbances. 3. Those due to local irritation or destruction: paralysis, usually hemiplegia; epileptoid convulsions (rarely); aphasia, and some cranial-nerve disorders. The final stage is coma and death from exhaustion.

In *chronic cases* the onset is exceedingly slow; perhaps with no active symptoms for months or even years, during which there may be depression, mental irritability, headaches, vertigo, and convulsions. The symptoms may at times be greatly increased in severity, with vomiting, intense pain, and perhaps delirium or convulsions, which subside and leave the patient in fairly good health. The final stage may begin with symptoms like those in the acute form. In other cases sudden coma, or epileptic or apoplectic attacks may occur, which are rapidly fatal. The disease (especially if traumatic) is frequently complicated by meningitis, or, when caused by ear disease, by phlebitis of the superior and lateral sinuses.

Brain abscesses occur more frequently in the cerebrum (right side, frontal and temporal lobes especially) than in the cerebellum, and are rare in the medulla and pons, depending upon the anatomical relation of the cause of the abscess to the temporal lobe and the cerebellum. The course of an acute brain abscess is from 5 to 14 days, in rare cases a month. In chronic brain abscess the latent period may last from a few weeks to months, or even 1 or 2 years, the terminal symptoms only a few days.

Diagnosis.—The cardinal points are: a history of ear or nose disease, injury, or remote suppuration; the presence of septic symptoms, headache, vomiting, local tenderness and increased temperature of the scalp, normal, subnormal, or irregular temperature, slow pulse, stupor, optic neuritis, lessened urinary chlorides, rapid emaciation, and delirium. The diagnosis of the location of the lesion must be made from a history of the cause, the presence of tenderness and increased temperature over a local area of the scalp, local convulsions, and hemiplegia. Brain abscess must be distinguished from meningitis, brain tumours, and phlebitis of the sinuses. The *prognosis* is always bad without surgical interference.

VI. **Chronic Hydrocephalus.**—Usually (80 per cent) begins at birth or within the first 6 months. Predisposing causes are lead-poisoning, tuberculosis, alcoholism, or syphilis in the parents, rachitis, and some unknown family taint. Late childhood or adult cases are usually due to tumour or inflammation obstructing the venæ Galeni and the Sylvian aqueduct.

Symptoms.—Usually the child's head (see page 170) begins to increase in size soon after birth, or may be much enlarged at birth. The infant becomes irritable and restless; its general nutrition is impaired; it does not grow as do normal children, although the appetite may be good; does not develop mentally; and generally does not learn to walk. Strabismus, and occasionally optic atrophy, are present. Within 2 or 3 years vomiting, coma, and convulsions appear, and death from exhaustion occurs.

Diagnosis.—The disease must be distinguished from rickets by the shape of the head (see page 171) and the presence of bone changes in the latter (*q. v.*). Cases which develop after birth may live for about 5 years. If mild, the process may stop and the patient live a fairly healthy life. Congenital cases may live from 2 to 3 years, but usually die within a few months.

VII. **Infantile Cerebral Palsies.**—(1) *Infantile Hemiplegia.*—Occurs slightly oftener in males, and in the great majority during the first 3 years of life. In congenital cases due to emotional disturbances, injuries, and perhaps diseases, affecting the mother during pregnancy; in cases occurring at birth, the use of forceps, tedious labour, or other conditions involving injury to the child; in cases occurring after birth, injuries, infectious fevers (especially pertussis, measles, pneumonia, scarlet fever), epileptic convulsions, cerebro-spinal meningitis, and rarely syphilis.

Symptoms.—A general, perhaps unilateral, convulsion, which may last for hours, initiates about one quarter of the cases, and is accompanied by fever which persists for several days. During this period, or after the acute symptoms have disappeared, the arm, leg, and face of one side, or of both sides, are found to be paralyzed. This paralysis gradually improves, the face first and most, then the leg, and the arm last and least. The growth of the affected side is retarded, and eventually the leg or arm may be 1 or 2 inches shorter than the other. The paralyzed limbs are cold, there are vasomotor disturbances, and rigidity, with contractures of the flexors and adductors. The most common contractures are of the heel, causing talipes equino-varus, or equino-valgus; and of the forearm, wrist, fingers, and the adductors of the thigh. Various motor spasms develop, e. g., ataxic, choreic, and athetoid (most common) movements, also associated movements and tremors. The reflexes are exaggerated and clonus is usually present. The mental development suffers. Feeble-mindedness, imbecility, and complete idiocy each claim an equal number. About one fourth of all cases preserve fair intelligence. Nearly one half of the cases have epilepsy; and a microcephalic or macrocephalic skull, asymmetry of the skull and face, imperfectly developed teeth, a high palatal arch, and prognathism are common. As a rule the skull on the side of the lesion is flattened. The special senses are sometimes defective. The chronic stage begins a few months after the attack. After the first amelioration there is little change until after puberty, when the general physical condition usually improves.

(2) *Diplegias or Birth Palsies.*—Due to injuries received at birth, or (more commonly) to intra-uterine disorders. At birth there may be prolonged asphyxia or convulsions. Some weeks later it is noticed that the child does not use its arms or legs. More convulsions occur, and a double hemiplegia becomes distinctly evident, with marked mental impairment. Epilepsy is common.

(3) *Spastic Cerebral Paraplegia* (*Little's Disease*).—See page 1080.

Diagnosis of Cerebral Palsies.—Must be distinguished from spinal palsies, in which there is an absence of rigidity, the reflexes are not exaggerated, there is marked atrophy and shortening of the limbs, and R. D. is present. The mode of onset and the distribution of the paralyses in cerebral palsies are characteristic. In mild cases the hemiplegia may almost disappear; with marked epilepsy and mental impairment they rarely reach adult life, otherwise they may improve and live long.

VIII. **Tumours of the Brain.**—The commonest forms are the sarcomatous type, tubercle, gumma, and infectious granulomata. Occur at all ages up to 50, one third under the age of 20, more often in males. Predisposing cause is perhaps heredity; occasional exciting causes are injuries to the head. In childhood, tubercle is the most common form, next glioma and sarcoma; after 20 years, gumma, glioma, and sarcoma; in middle age and late life, sarcoma, gumma, and cancer.

Symptoms.—The *general symptoms* are: persistent intense headache with marked exacerbations; vomiting, convulsions, general or local; paræsthesias; vertigo, impaired eyesight, and perhaps mental dulness or slowness. Weakness and emaciation follow the vomiting and intense pain. Paralyses and blindness ensue, convulsions occur more frequently, the patient becomes bedridden, and after from 1 to 5 years dies of exhaustion.

Head pain occurs in over one half of all cases, is very severe (lancinating, boring), and may be in the forehead, occiput, or the whole head. It may be periodic, quotidian, or tertian, as if of malarial origin. Pain occurs most frequently with tumours of the cerebellum or the cerebral hemispheres and midbrain; less often if in the peduncles and at the base. There may be tenderness of the scalp and cranium, especially over the tumour. Vertigo is present in nearly one half of the cases, either severe (especially with cerebellar tumours) and accompanied by forced movements, or slight. Vomiting, frequently "projectile" and with little or no nausea, occurs about as often as headache, with rapidly growing or cerebellar tumours. Optic neuritis (four fifths of all cases, usually in both eyes) is most frequent in tumours of the cerebellum, midbrain, and great basal ganglia; rare in tumours of the medulla, infrequent in slow-growing tumours; and usually ends in optic-nerve atrophy.

In about one fourth of the cases general convulsions occur, more frequently when the tumour is in the cerebral hemispheres and cortex. Apoplectiform attacks, more rarely true apoplexy, may occur. There is almost always some mental impairment, e. g., attacks of somnolence, mental slowness, weakened memory and power of attention, and sometimes silliness, childishness, or peculiar mental irritability. In tumours affecting the pons and medulla and the origin of the cranial nerves, there is frequently a speech disturbance consisting of a running together of the syllables of words. The cranial temperature is somewhat higher than under normal circumstances.

Tumours cause, besides general symptoms, *focal symptoms*, which depend upon the site of the tumours. For convenience the brain is divided into 12 parts or areas, as follows:

1. *Frontal Area.*—Often cause no distinct localizing symptoms. In a goodly number of cases there are peculiar mental disturbances consisting of childishness, silliness, mental hebetude, and a tendency to emotional instability. There may be optic neuritis, hemianopia, and anosmia. Involvement of the orbit results in ocular paralyses and exophthalmos. Backward growth causes irritation of the motor centres, with convulsions, spasms, and paralyses. 2. *Motor Region.*—Tumours here first irritate the motor centres, causing Jacksonian epilepsy (page 550). Sensory symptoms often precede or accompany the spasms, i. e., prickling, numbness, or slight hemianæsthesia. As the tumour grows the convulsions become general, and hemiplegia may appear. There may be some impairment of the muscular sense. Agraphia and motor aphasia may also be present. 3. *Parietal Area.*—The most characteristic symptoms, which may be slight, are disorders of muscular sense and word-blindness. When the tumour is near the longitudinal fissure the leg muscles may be involved. 4. *Occipital Lobes.*—Tumours, situated in the cuneus and first occipital convolution, cause homonymous hemianopia (page 222). Mind-blindness (page 267) may result if the other parts of the occipital lobe are involved and without serious injury of the cuneus. There may be word-blindness, with some hemianopia, if the tumour grows toward the angular gyrus. Hemianæsthesia and perhaps hemiplegia may occur when the tumour encroaches forward upon the parietal area. 5. *Temporal Area.*—Tumours on the right side produce few symptoms; on the left side word-deafness, and attacks of vertigo or forced movements. There may be disturbances of taste and smell if the hippocampal convolution and the uncus are involved. 6. *Corpus Callosum.*—Tumours are rarely found here. The focal symptoms are a gradual hemiplegia followed by paraplegia, with drowsiness, mental dulness, and stupidity. The presence of apraxia is significant. 7. *The Great Basal Ganglia and the Capsule.*—The symptoms are similar to those of tumours in the corpus callosum. There may be less stupidity. There is a progressive hemiplegia, sometimes with anæsthesia or choreic movements. Involvement of the posterior part of the optic thalamus will cause hemianopia with hemiopic pupillary inaction (page 222). 8. *Corpora Quadrigemina, Deep Marrow, and Pineal Gland.*—Inco-ordination, forced movements, and oculo-motor palsies are characteristic. There may also be blindness or hemianopia. 9. *Crura Cerebri.*—Tumours here are very rare, and cause a crossed paralysis. 10. *Pons and Medulla.*—The symptoms vary according to the size and location of the tumour. If high up in the pons there will be 3d-nerve paralysis on one side, hemiplegia on the other; if lower

down 7th-nerve paralysis on one side, hemiplegia on the opposite side. When large, hemianæsthesia may exist with the hemiplegia. A tumour in the medulla causes hemiplegia and hemianæsthesia, with paralysis of the hypoglossal, or some other cranial nerve on the same side. If both sides of the medulla are involved the symptoms of progressive bulbar paralysis may appear. Pons tumours sometimes cause a conjugate deviation of the eyes toward the side opposite to the lesion. 11. *Cerebellum.*—If the tumour is in the middle lobe there is cerebellar ataxia (see page 565), and there may be severe forced movements. There are often secondary symptoms from pressure on the medulla when the tumour is in the middle lobe. Cranial-nerve disturbances and glycosuria are usual. In the lateral lobes there are no localizing symptoms until the tumour presses upon the adjacent parts. Late in the disease hemiplegia, paraplegia, bulbar symptoms, and perhaps hydrocephalus, may develop. 12. *Base of the Brain.*—The symptoms of tumours in the anterior fossa are very similar to those caused by tumours in the prefrontal area. There are, however, anosmia, and perhaps involvement of the optic and oculo-motor nerves. Tumours of the middle fossa may involve the hypophysis. The symptoms of tumours of this region and of the interpeduncular space are those of pressure on the optic chiasm, viz., early optic neuritis and hemianopia.

It must be borne in mind in localizing brain tumours that a certain proportion of them are multiple.

Diagnosis.—The cardinal symptoms of brain tumour are: vomiting, headache, vertigo, optic neuritis, mental disturbances, and the progressive course. Other points are the history of an injury, local tenderness, or tuberculous or syphilitic disease. Brain tumour must be distinguished from brain abscess, meningitis, hysteria, paretic dementia, lead-poisoning, and cerebrospinal syphilis.

Prognosis.—Serious. The disease may last for from 1 to 18 years; usually death occurs within 3 years; rarely it may remain stationary.

IX. **Multiple Sclerosis.**—Occurs usually between the ages of 20 and 30, more often in males, also, rarely, in infants and children. There is often an inherited neuropathic tendency. The most important causative factor is infection—e. g., malaria, typhoid fever, the exanthemata, pneumonia, rheumatism, pertussis, diphtheria, dysentery, cholera, and erysipelas, especially typhoid and malarial fevers. Slow poisoning by some of the metals, and exposure to cold, more rarely sunstroke, fright, shock, and trauma, are causes.

Symptoms.—Onset slow and gradual. First noticed are heaviness and weakness in the legs, perhaps with some numbness; or a tremor in the hands. Soon follows ataxia of the legs, which, with the weakness and rigidity, increases the difficulty of walking. There may be some incontinence of urine. The tremor in the hands, which may not have appeared until now, is "intentional." The speech is slow, syllabic, and scanning in character. Deglutition may be impaired.

Occasional pains may occur in the joints and extremities, with some numbness or slight tactile anæsthesia in the limbs. The gait is awkward and stiff, and there is, perhaps, slight hemiplegia. There is marked atrophy of the limbs, without R D. The ataxia is due mainly to the inability to co-ordinate and control the movements, weight and pressure senses being unimpaired. The jerky tremor of the hands may be so marked that the patient finds it difficult to dress himself, or to carry a glass of water to his mouth without spilling it. There may be tremor in the muscles of the neck, causing a constant movement of the head, or in those of the face. The tongue is protruded in a jerky manner, and the monotone speech may be unintelligible. There are exaggerated knee-jerks, Babinski and ankle clonus. Nystagmus is present, perhaps only to be seen when the eyes are turned far to one side, but may be manifest when looking directly at an object, perhaps with diplopia. The pupils react normally. Optic atrophy may be present, chiefly in the temporal half of the disc, which may become complete later in the disease, followed, as results, by weakness of vision, contraction of the visual fields, and scintillating scotomata. Occasionally there may be attacks of vertigo, and apoplectiform and epileptiform seizures. There may be mental slowness, perhaps slight melancholia, or impulsive laughing or crying. Epigastric reflex often absent. The course of the disease is irregular, usually lasting about 5 years, occasionally 3 times as long.

Diagnosis.—The cardinal points are: intention tremor, nystagmus, scanning speech, exaggerated knee-jerks, ankle clonus, ataxia, and rigidity in the legs, attacks of vertigo, and apoplectiform and epileptiform seizures. Must be differentiated from spastic spinal paralysis, meningo-myelitis, Friedrich's ataxia, bulbar paralysis, paralysis agitans, dementia paralytica, hysteria, and chronic meningitis.

Prognosis.—Good as to life. The symptoms may disappear and the patient apparently recover, but after some years may reappear; or the disease may reach a certain state and remain stationary.

VIII. SYPHILIS OF THE NERVOUS SYSTEM

Occurs most often between the ages of 20 and 40, but may be met with at any age, more often in men. Predisposing causes are a neuropathic constitution, excessive physical exercise, alcohol, injuries, mental strain, and overwork. Usually appears in the 3d year, though it may develop at any time within 30 years, after infection.

Symptoms.—The following divisions are made (Dana):

(1) Syphilis of the Brain.—The chief symptoms are intense headaches, cranial-nerve palsies, optic neuritis, attacks of somnolence, coma, and hemiplegia. Other symptoms are nausea, vomiting, vertigo, men-

tal irritability and dulness, epileptic convulsions, polyuria, and polydipsia. The headache (persistent and intense) comes on gradually. Hemiplegia occurs a variable time after the headache, and with it there are some cranial-nerve (especially ocular) palsies.

(2) Cerebro-spinal Syphilis.—Nearly the same as are present in (1), but there are in addition symptoms due to the cord lesions—i. e., spastic paraplegia, spinal pains and pupillary involvement.

(3) Spinal Syphilis.—Usually those of a transverse myelitis, coming on slowly and developing into spastic paraplegia with much pain. There may be Brown-Séquard paralysis, with some ataxia.

(4) Syphilis of the Nerves.—The peripheral nerves are rarely affected, but deposits of syphilitic exudate sometimes cause symptoms of irritation. There are symptoms due to cranial-nerve palsies.

(5) Post-syphilitic Degenerative Processes.—These are general paresis and locomotor ataxia, to which syphilis may predispose.

(6) Hereditary Syphilis.—May present all or any of the symptoms of acquired syphilis. Usually develops under 5 years of age, but may occur up to 18 years, or rarely later.

Diagnosis of Syphilis of the Nervous System.—The cardinal points are: History of infection, pupillary changes, severe headaches, irregular and fleeting character of the symptoms, presence of optic neuritis, and therapeutic results. The headache of syphilis is worse at night, very intense, irregular in regard to the part of the head affected, and may be periodical. Hemiplegia, or paralyses of one or more cranial nerves, occurring after a headache of the above character, suggest syphilis. *Prognosis.*—Marked improvement, perhaps recovery, may be brought about, but if nerve tissue is destroyed the effect is permanent.

SECTION VIII

DISEASES OF THE MIND

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IN order to appreciate at their true value the nature of the symptoms found in the various mental diseases, it is necessary to have in mind an understanding of the nature and characteristics of the various mental processes.

THE MIND

Broadly speaking the function of the lower nerve-centres is so to order the functions of the several organs of the body that they are rightly timed and harmoniously adjusted in relation to each other.

The higher nerve-centres that underlie the manifestations of mind (or consciousness) have for their function so to regulate and control the actions of the individual as to bring him into harmonious relations with his environment. In order that the mind may perform this function three things are necessary: 1. A knowledge of the environment must be gained. 2. This knowledge must be associated and brought into relation with previous experiences. 3. It must be transformed into the appropriate actions. The *sensorium* subserves the first of these functions, the *intellect* the second, and the *motorium* the third. The function of the sensorium is *perception*, of the intellect *thinking*, and of the motorium *volition*.

To be more explicit: All our information of the environment must come primarily through the sense organs. The stimulation of these organs produces sensations which are unanalyzable elements of consciousness. Sensations alone cannot compose a mental state. They must be associated with previously experienced mental states. When this occurs perception takes place which may be defined as the forming in the mind of an object presented to the senses.

This association of sensations and perceptions with previous mental states, is an association with ideas which are images present in mind but not presented to the senses at the time.

The association of percepts and ideas, or in other words the assimilation and rearrangement of the materials of knowledge furnished by the senses and that already present in consciousness, is the process of *thinking*. When from the association of ideas a new idea comes forth the process is reasoning, and the new idea is a *judgment*.

Having received certain information from the environment, having reasoned about it, and having reached certain judgments, the motorium is called upon to initiate appropriate *actions*. There are usually several judgments found simultaneously which constitute so many *motives* to action. The conscious realization in action of the strongest motive is the process of *volition*, and is accompanied by a feeling of freedom to choose which motive shall predominate. The sum total of the actions of the individual is *conduct*.

A certain mental process having once occurred tends to leave its impress on the physical substratum of mind. This is the physical basis of memory, which, psychologically, may be said to be the recurrence to consciousness of a previous experience and the recognition of it as having occurred before.

All mental processes are accompanied by conditions of agreeableness or disagreeableness, pleasure or pain. When these states relate to relatively simple experiences, e. g., a musical sound or a shrill whistle, they are described as *feelings* or *affects*: when the experiences are relatively more complex, e. g., love, hate, they are known as *emotions*: when still more complex, e. g., honor, patriotism, as *sentiments*. The word affect is, however, generally used in psychiatry to cover all these conditions.

SUBSECTION I

GENERAL SYMPTOMATOLOGY OF THE PSYCHOSES

In describing the general symptoms of insanity the scheme of the several fundamental psychic processes already outlined will be followed, and the disorders of each taken up in turn and described, so that in the subsequent pages which deal with the question of diagnosis it will only be necessary to refer to them by name.

I. DISORDERS OF PERCEPTION

I. **Illusions.**—An illusion is an inexact, incorrect, or false perception. The information conveyed to the mind by the sense organ is misinterpreted so that the source of the sensory impressions in the environment is not appreciated at its true value. A strap lying on the floor may be perceived as a snake; the sighing of the wind may be perceived as the whispering of a human voice; a bad taste in the mouth may be perceived as poison, and so on throughout the different sensory realms. The distinguishing thing about an illusion is that an actual something in the environment is perceived, but the perception is not a correct one and conveys false information to the mind.

II. **Hallucination.**—An hallucination, on the other hand, is generally conceived to be a perception without sensory foundation in the environment. A snake is seen on the floor where there is nothing which could be mistaken for a snake; human voices are heard when there are actually no sounds in the environment which can have been interpreted as such; poison is tasted where there has been nothing in the food or in the mouth which could have given origin to the taste. The distinguishing feature of an hallucination then is a perception without there having been anything to perceive.

Recent studies, however, have made it highly probable that a large number of what have been supposed to be hallucinations are in reality dependent upon pathological, or even at times physiological, processes occurring in the sensory end organs, so that, with reference to the eye and ear, for example, they might be said to be of entoptic, or entotic origin respectively.

So far, however, as any given erroneous perception is concerned, it really matters little from the point of view as to its significance as a symptom of mental disease whether it be classed as an illusion or an hallucination. The mental process in both instances is identical. In practical use the two are not often distinguished, but false perceptions are generally spoken of as hallucinations, and as the mental process is the same in both hallucinations and illusions the necessity for their distinction does not arise, and the use of the term hallucination serves the purpose very well.

III. **Clouding of Consciousness.**—The process of perception, as we have seen, is dependent upon sensations coming from without, which, however, must be of sufficient strength to force their way into consciousness and wake up, as it were, the remains of former sensations with which they become associated. If sensory stimuli have not this strength they may be said to be inadequate. We are constantly beset on all sides by such inadequate stimuli. The presence of clothing on the different parts of the body ordinarily causes no appreciable sensations, and thus gives rise to no perceptions. The many trifling noises going on while one is absorbed in writing are not heard. The strength of the several sensory stimuli is not sufficient to cross the *threshold of consciousness*; their *threshold value*, as it is called, is too low to result in perception. In various diseases and conditions the threshold value of sensations is greatly altered. This is very marked in certain of the deliria, for example delirium tremens. In this disease we find the patient wholly occupied with his terrifying visions and quite oblivious to the outside world of realities. Loud sounds fail to attract his attention, the nurse comes and goes without interrupting the course of his delirium. Sensory stimuli of ordinary, or even of more than ordinary, strength fail to cross the threshold of his consciousness and cause perception. If, however, the patient be taken firmly by the shoulders and held, or mildly shaken, while a question is actually shouted at him, we may find that he will give a perfectly lucid and correct answer. The threshold value of the sensations has been sufficiently increased, the resistance has been broken down, and perception takes place.

This condition of clouding of consciousness may exist in any degree from a scarcely noticeable departure from clear consciousness to actual coma, and as we can readily see must be the cause of very imperfect perceptions of the environment. Ordinary stimuli are not appreciated at all, while those that have sufficient force usually only give rise to perceptions for the moment and are never adequately assimilated. Thus we find this condition prominently in evidence in the various deliria, where it is usually associated with disturbed effects, disorders of the train of thought, and hallucinations.

IV. **Disorientation.**—Orientation implies the correct apprehending of the environment, and one is said to be fully oriented when he understands his own position and relation with reference to the different aspects of his environment. These aspects are three, viz.: temporal, spatial, and personal. Temporal orientation then would imply correct answers to such questions as, In what year were you born? What year is this? What day is this? Spatial orientation would imply correct answers to, What city do you live in? On what street? What city are you in now? What institution is this? Personal orientation would imply a correct knowledge of who the individuals were with whom the patient came in daily contact, their official positions, and, in many cases, their names. Disorientation is the reverse of this condition and implies a lack of apprehension of these three aspects of the environment either singly or together.

II. DISORDERS OF THINKING

Disorders of the Content of Thought.—I. **Delusions.**—A delusion is a false belief, but as such it is not necessarily evidence of insanity. Many false beliefs have no pathological significance whatever. A man may believe that to-day is Thursday when in fact it is Friday. That is a false belief while it lasts, but has only the significance of a mistake. The belief of certain savages that dreams represent the wanderings of their disembodied spirits we know to be false, but not an evidence of mental disease. False beliefs or delusions then may be either sane or insane, and it is for us to endeavor to distinguish what constitutes a belief first as false and then, as such, what characterizes it as insane. There are three main characteristics of insane delusions in general. First: they are, as a rule, very evidently not true to facts, highly improbable, even manifestly impossible, often to the extent of being bizarre. Such, for instance, are the delusions of great wealth or of royal lineage, and those of a certain class of patients who believe that they have no stomach, no brains, even that they have no head. Second: they can not be corrected by an appeal to experience. Not originating in experience they can not be corrected by experience. It is impossible to argue the patient out of his insane beliefs. Third: they are out of harmony with the education and surroundings of the individual. The sick Fijian lying upon his back and crying for his soul to come back to him is but exemplifying the belief of the race that sickness is due to the soul or a part of it leaving the body. Should we find a modern American, with the usual public-school advantages, acting thus, we would be justified in supposing him unbalanced.

It sometimes happens, however, that a false belief does not show any of these characteristics and yet may be an insane delusion. A woman who says that her husband is untrue to her has not voiced a belief which has on its face any evidence of impossibility, and with no knowledge of the facts it can not even be said to be improbable. While not susceptible to the test by argument it is certainly not out of harmony with the individual's education and surroundings. In such cases it becomes important to study the origin of the belief, to find out upon what sort of foundation it is reared. If we find that it resulted from the patient having awakened on several occasions during the night and found her husband's legs cold, and having reasoned from this that he had been out of bed to keep an appointment with his paramour in an adjoining room, we will at once have no difficulty in stamping the delusion as insane.

Delusions may be classified for our purpose into fixed and changeable, systematized and unsystematized.

A *fixed delusion* is one which seems to be firmly embedded in the mind and is continuously adhered to by a patient, while *changeable delusions* are constantly changing and giving place to one another.

An *unsystematized delusion* does not enter into organic combination with the other facts of consciousness, but stands apart and seems not to have been assimilated. While it may be fixed, it exercises no special control over the patient's conduct; he seems to rest with its statement alone, and is unable to substantiate his position by cogent argument or example. A patient who believes that all the bones in his body are broken, but nevertheless goes about his affairs as usual, has an unsystematized delusion. A *systematized delusion*, on the other hand, is not only assimilated and associated with the other facts of conscious experience, but forms a motive power for conduct. It is supported by reasons, by argument, and by appeals to experience; it is acted upon as if it were an actual fact, and finally it may so reach out its influence by association with all the conscious experience of the individual, that the whole life of the patient is centred about and becomes secondary to it. The patient with a systematized delusion of persecution regulates his whole life in order to avoid his persecutors; the food is carefully tasted for poison and perhaps discarded; the bed he sleeps in must be insulated to prevent electric currents being applied to him while he sleeps; the keyhole and all cracks stopped up so that noxious vapors can not be injected through them. If the patient is asked for an explanation of this conduct he is ready with interminable reasons and appeals to experience, while his arguments are woven together with much ingenuity and no little logic. His delusion is systematized.

II. **Obsessions.**—By obsessions we mean ideas which occupy consciousness persistently and irrespective of the desires of the subject, often obtruding themselves at inopportune times and occupying the field of consciousness to the exclusion of other ideas. They are sometimes spoken of as besetments, as they come unbidden and refuse to go at the will of the subject. They exist with clear consciousness and are fully comprehended by the patient, who appreciates them at their true value.

The commonest and best known of the obsessions are the so-called *phobias* or fears. These fears are usually very specific, referring to some special class of objects or set of conditions, and receive names accordingly. Thus we have a mysophobia (fear of dirt); aichmophobia (fear of sharp points); metallophobia (fear of metal, such as door-knobs, money, etc.); agoraphobia (fear of wide or open spaces); claustrophobia (fear of narrow or closed spaces); pyrophobia (fear of fire); and so on indefinitely. Patients suffering from these obsessions are often completely dominated by them under conditions that call them into existence. The patient with agoraphobia crosses the street in fear and trembling, or, perhaps, can not summon sufficient courage to cross it at all unless someone is with him; while on the contrary the claustrophobiac can not endure a small or closed room, but must have the doors open, or, if in a crowded hall, is suddenly seized with fear and forced to make a hasty exit.

Of not infrequent occurrence also are the obsessions of doubt. These doubts may arise about anything, even the simplest acts of everyday life. A patient upon retiring may be seized with a doubt as to whether he turned the gas off or locked the door in the main hall, and is forced to get up and go and see, only to be seized again by the same doubts when he has returned to bed; another having written several letters is forced to open all of them to make sure that the right ones are in each envelope. Still others have doubts about religious or metaphysical matters. So we have doubters who question the problems of a future life or the existence of things as they appear to the senses, etc.

If the actions which the obsessions tend to initiate are resisted, the tendency becomes more and more imperative until yielding is forced, and finally these patients, although fully understanding their condition and the abnormality of their ideas, may pass their lives in a continual round of actions made necessary by their obsessions.

The term *fixed idea* has been applied to many of these conditions and as a term designates them very well. There is usually a distinction made, however, between obsessions and fixed ideas. While the obsessed patient recognises that his ideas are foolish, without foundation, pathological, the patient with a fixed idea does not. His idea, he thinks, is the natural result of certain conditions. It is a reasonable idea under the circumstances. Thus the rather timid young man who feels his pulse and discovers that it is too rapid and perhaps somewhat irregular, acquires as a result a fixed idea that he has organic disease of the heart. The woman whose husband has recently died, and who is harassed by the belief that he might have lived had she given him a certain medicine, has a fixed idea.

III. **Dream States.**—This term is applied to certain conditions because of the resemblance they have to conditions of dream consciousness. The mind is occupied by numerous dreamy ideas, and usually also by multiform hallucinations which may take the form of visions producing the state of ecstasy. The threshold value of sensations is raised so that no impressions of ordinary strength reach consciousness, the result being that the patient is quite oblivious of his surroundings. Unlike the normal dream state, however, psychomotor reactions occur corresponding to the content of consciousness.

Disorders of the Train of Thought.—I. **Flight of Ideas.**—In the normal process of thinking our thoughts are directed consistently to a well-defined end—the *goal idea*—and all other ideas fall into a subordinate position until this is attained. In flight of ideas the patient either has no goal idea, or else at once loses it, so that there is no consistent effort directed toward attaining it, and the thought therefore wanders here and there under the influence of chance associations. As a result the train of thought, instead of progressing, changes direction frequently, returns upon itself, and never reaches any logical end. The various ideas are not, however, incoherent—they do not fail to be connected one with another, although it may be quite impossible at times to see just what their connection is. If the associations are external, that is, originate in the surroundings, it is usually quite possible to place them. When, however, they are internal, that is, originating in the patient's mind, it may be quite impossible to conceive what they may be. An example will illustrate these various conditions: "Do you know I was kidnapped to be sent here twice. I saw a mock funeral of me before I left home. This was done because I am a great inventor. The Pope of Rome is the greatest human being in the universe. He is the head of the Catholic Church. My head (association of the word head in two different meanings) is good and sound, and I am certainly not insane. Do you hear that ticking of the clock? (External association.) It says, 'Call the little heifer, the heifer is sick.' Did you ever see the gloves veterinary surgeons use when they doctor sick cows? (Internal association.) How would you like to be a veterinary surgeon? Say, what are you keeping me here for anyhow? I want to go home." Here he was asked how he slept at night. "I have slept excellently; that is because I am of such a strong constitution. The Constitution of the United States (association as above with the word head; probably the association is in large part, at least, a sound, or—as it is called—a *clang association*) was signed by Thomas Jefferson. He was a just man, but he was not the inventor I am." While there are many places in the example where the connecting link is missing—probably because it was an association formed entirely within the patient's mind—still the connection can be made out in a sufficient number of instances to establish the characteristics of the train of thought. One of the principal characteristics of this type of the train of thought is its great liability to change of direction by external association, as, for example, the ticking of the clock in the above stenogram. This quality is known as *distractibility*. Any sensory impression is liable to be the starting-point of idea-association so that these patients' train of thought may be turned, almost at will, by such devices as shaking a bunch of keys before them, saying some word loudly, showing them a newspaper, or, in other words, momentarily distracting their attention.

II. **Circumstantiality.**—Circumstantiality, although superficially resembling flight of ideas, is quite different from it in its completely developed form. Although there is a frequent change of direction of the train of thought the goal idea is ultimately reached, and, in spite of numerous digressions, as each circumstance in the narrative is elaborated and explained the original pathway is regained and the general direction maintained. Thus a patient telling about a cane in her possession upon which a ribbon is tied for ornament, must tell how she came to have the idea of decorating the cane, who else in the neighborhood had such canes decorated, what they had said to her and she to them about it, how she had taken the ribbon off and now had put it in two boxes in the house, how the ribbons had become faded, where she got the boxes, who gave them to her, and what their color was. All these details must be entered into before she can proceed with the thread of her narrative.

This condition is often found normally, in a moderately developed form, in women, and has no special significance other than showing a lack of appreciation of the relative values of ideas. In the senile, where there is some mental impairment, the goal idea may be quite lost sight of in the mass of detail, and the resemblance to flight is then much more marked; still there is a more marked tendency to maintain the general direction of the train of thought.

III. **Retardation.**—This symptom, *difficulty of thinking*, as it is often called to distinguish it from psychomotor retardation shortly to be described, is a decided slowness in the elaboration of ideas. The patient's stock of ideas does not seem to be available or accessible; ideas come slowly to the mind; there is great difficulty in forming judgments, in coming to conclusions, in reaching decisions, which is felt by the patient as an *inadequacy* in dealing with mental problems. This condition is manifested by the patient in great slowness of speech, a long interval elapsing before an answer is given to a question or something done as requested (*initial retardation*), and when the question is answered or the act done, it is done very slowly and deliberately (*executive retardation*).

If such a patient be asked to count from 1 to 20, beginning at a signal and counting as fast as possible, it may be several seconds before he starts, and, while a normal person should do this in 2 or 3 seconds, he may take often 20 or 30, or even never finish at all.

IV. **Paralysis of Thought.**—A complete absence of all internally initiated conscious processes. Impressions from without are not assimilated, form no associations, leave no traces. Mental life is in abeyance or abolished.

III. DISORDERS OF VOLITION (Willing)

I. **Decreased Psychomotor Activity.**—This symptom corresponds in the motor sphere to difficulty of thinking in the psychic sphere. Whereas in difficulty of thinking we might say that there was a slowness in the liberation of ideas, in psychomotor retardation we can say there is a slowness in the liberation of voluntary motor impulses. The patient's movements are slow and deliberate, and we find here the same distinction of initial and executive retardation. This is a prominent symptom of depressive melancholia.

II. **Increased Psychomotor Activity.**—This is just the opposite of the above condition, and is due to an abnormally facile release of voluntary motor impulses. It manifests itself in great restlessness and constant activity, even to the point of violence and destructiveness. This is a prominent symptom of mania.

III. **Impulsions.**—Impulsions or impulses are tendencies to act which are more or less uncontrollable, often absolutely so. The act may be of any kind. In this class belong the so-called manias, such as kleptomania (a morbid impulse to steal), pyromania (a morbid impulse to set things on fire), dipsomania (an impulse to drink), etc.

These impulses appear without cause, and the patient is restless until they are carried out, but their accomplishment is often accompanied by a feeling of remorse.

Closely allied to these impulsions are the so-called *compulsions*. These compulsions, like the obsessions already described, are felt by the patient to be pathological, to be forced upon him as it were. The impulse of the dipsomaniac, like the fixed idea, is conceived by the patient as originating within and being a part of him, perhaps a natural development of his character; while the compulsion is often directed to the doing of some act distinctly abhorrent to the patient, such, for instance, as murder, and he may take elaborate precautions to protect others, or even have himself locked up to insure against its possibility.

If these compulsions are resisted or interfered with, they give rise to certain symptoms, which in marked cases constitute a veritable crisis. The patient feels weak, trembles, becomes dizzy, perspires, and finally yields, to find that at once all these symptoms disappear.

IV. **Stereotypy.**—In stereotypy the voluntary impulse once set in motion tends to continue or repeat itself in the same way indefinitely. Thus we have three forms of stereotypy, viz., stereotypy of attitude, of movement, and of speech.

In stereotypy of *attitude* the patient tends to maintain a particular, usually peculiar, position, such as standing in the corner, one arm raised, lying on the bed with the head hanging over the side. The muscles are usually tense and the patient resists an attempt to alter his position.

Stereotypy of *movements* manifests itself in a peculiar manner of doing things such as walking, eating, etc. When the word stereotypy is used without qualification this variety is referred to. When these peculiarities are constant and characteristic of the patient they are usually spoken of as *mannerisms*.

Stereotypy of *speech* shows itself in the constant repetition of the same phrases, and is more commonly known as *verbigeration*.

V. **Negativism.**—Negativism is a peculiar condition which is manifested by the patient doing exactly the opposite of what he is requested to do. Every attempt to get the patient to do anything results in the release of a motor impulse the exact opposite of that required for the performance of the act.

VI. **Suggestibility.**—Suggestibility may be said to be the exact opposite of negativism. The patient's reaction is determined by impressions or suggestions derived from others. It is manifested in various ways. In extreme cases the patient resembles a lay figure;

the limbs can be placed in any position and are there retained indefinitely. This condition is designated as *catalepsy*, or *flexibilitas cerea* (waxy flexibility). Often suggestibility is manifested by the patient repeating words or phrases said in his presence—*echolalia*—or actions done before him, such as taking out the watch, putting the hand to the face—*echopraxia*.

VII. **Stupor.**—Stupor is a condition in which there is usually a profound disturbance of consciousness, but the feature which gives it its distinctive outward character is psychomotor inhibition—voluntary motion to a greater or less extent in abeyance.

IV. DISORDERS OF THE EMOTIONS

I. **Exaltation.**—Exaltation is a condition of morbid emotional elation, a feeling of happiness and well-being not warranted by the condition of the patient or his surroundings. It is one of the most prominent symptoms of mania.

II. **Depression.**—Depression is the exact opposite of exaltation. It is a morbid feeling of unhappiness not warranted by the condition of the patient or his surroundings. It is one of the most prominent symptoms of melancholia.

III. **Emotional Deterioration.**—A condition of poverty of the emotions, manifesting itself by indifference and occurring in conditions of mental deterioration.

V. DISORDERS OF MEMORY

I. **Amnesia.**—Amnesia is loss of memory. The loss may be circumscribed—only for certain things or only extending over a very definite space of time—or it may be more general. Loss of memory extending over definite periods of time is usually the result of illness or injury. In such cases the amnesia may extend farther back than the beginning of the illness or the date of the injury. In which case it is known as *retrograde amnesia*.

II. **Paramnesia.**—This is a disorder of memory in which events are remembered which never happened. When these memories are projected into the past and associated with delusions often of an explanatory nature, as occurs in paranoia, the symptom is known as *retrospective falsification of memory*.

III. **Hyperamnesia.**—An exaggerated degree of retentiveness often seen in the remarkable memory for details in some cases of chronic delusional insanity, who seem to remember every detail in their lives as bearing on their delusional system.

SUBSECTION II

THE PSYCHOSES (DISEASES OF THE MIND)

In the diagnostic discussion of the psychoses which follows it is inevitable that those forms which resemble each other should be grouped under the same heading. It is, however, to be understood, that the resulting classification is not offered as in any sense final, but only as tentative and affording certain clinical and descriptive advantages. Many of the types could as well be placed under some other heading as under that in which they are found, while transition forms occur that do not strictly belong under any.

I. INFECTION-EXHAUSTION PSYCHOSES

(TYPE = ACUTE CONFUSION)

General Characterization.—An acute primary insanity characterized by clouding of consciousness, confusion, multiform and usually fleeting hallucinations in the various sensory areas, changeable delusions, the emotional attitude being variable and in general corresponding to the content of the delusions.

I. Febrile Delirium.—A condition of acute confusion of variable intensity following in its degrees the febrile movement. The milder cases usually exhibit symptoms only as night approaches, at which time they begin to mistake objects in the room, become disoriented, mildly confused, and restless. More severe cases present marked clouding of consciousness, disorientation, multiform and often terrifying hallucinations, and dreamy delusions. Objects in the room are mistaken, a spot on the floor is blood, the bed is on fire, visions are seen on the walls and ceiling. In this state there is considerable noisy excitement. This condition may become more aggravated, the excitement more marked, leading to great agitation, restlessness, and finally purposeless movements. The expressions become very incoherent, and a low muttering delirium develops, with subsultus tendinum and carphologia.

The onset and severity of the delirium is, to an extent, a measure of the mental stability of the patient. Delirium develops much more readily in the unstable, and in those predisposed to the development of psychotic symptoms.

II. Infection Delirium.—Under this head are included the mental disturbances which develop early in the infectious diseases, either before the fever appears at all or else when it is still so low that the mental disturbance can not be attributed to it and therefore must be due solely to the infectious agent (*initial delirium*). This condition is found associated with typhoid fever, typhus fever, smallpox, malaria, and hydrophobia. It usually takes the form of an acute confusion, but there may be delusions of a consistently disagreeable character, generally persecutory. The condition in hydrophobia is rather one of change of character, irritability, restlessness, usually depression, verging into a delirium with confusion, hallucinations, and excitement as the disease progresses.

III. Post-Febrile Psychoses.—These conditions either develop as a result of the delirium of the febrile state continuing after the fever has subsided, or may begin during the post-febrile period. In the latter case the disease is essentially an exhaustion psychosis.

The mental state is one of confusion with multiform hallucinations—the patient sees strange faces peering at him from the pictures on the wall, he can see through the walls into the next house, the pictures turn and change places without hands—there is marked disorientation, and delusions usually of a persecutory nature—poison is administered in the medicine. This condition may become more severe, the delirium more active, the utterances very incoherent, and finally a stuporous state develops, with a tendency to catalepsy.

The exhaustion in most of these cases may be profound and terminate fatally; a certain few go on to the development of a chronic delusional state. Improvement in the general physical state is accompanied by mental improvement.

IV. Exhaustion Psychoses.—These conditions develop after severe exhaustion from any cause—loss of blood, parturition, prolonged anxiety and worry, severe mental shock. There may be a prodromal period of restless irritability and insomnia, after which a condition of confusion develops, which may be very mild, constituting only a slight degree of perplexity, or more usually manifesting hallucinations, clouding of consciousness, disorientation, and dreamy delusions. Psychomotor excitement is common, the patient being very active and inclined to acts of violence and destructiveness (collapse delirium).

Stupor with catalepsy may constitute an episode, or be sufficiently in evidence to give its character to the attack. Certain cases may run a milder course with prominent confusion, hallucinations, and delusions, and a milder grade of excitement. There may be episodic stuporous states, and short remissions with lucidity are quite characteristic (*amentia*).

The confusion in some cases may proceed to a high grade of incoherence and agitation, with fever usually ranging as high as 103° to 104°—a severe grade of exhaustion with typhoid symptoms—followed in a large proportion of cases by coma and death (*acute delirium*).

Diagnosis of the Infection-Exhaustion Psychoses.—The diagnosis is to be made in general from the association of acute confusion, multiform hallucinations, changeable delusions, disorientation, clouding of consciousness, and variable emotional reactions, with specific infection or severe exhaustion. It must not be forgotten that certain other insanities, particularly dementia præcox and manic-depressive insanity, may originate under the same conditions which lead to the development of the infection-exhaustion psychoses; and further, that aside from the conditions of confusion described (*primary confusion*), states of infection and exhaustion may complicate any psychosis, producing a confusion engrafted on the original mental disorder (*secondary confusion*).

II. AUTO-TOXIC PSYCHOSES

I. **Uræmia.**—The auto-intoxication which develops as a result of renal disease produces mental symptoms of an acute confusion, with changeable delusions, hallucinations, clouding of consciousness, and restlessness. The character of the delusions may be more or less consistently grandiose, giving rise to an *expansive form*; or depressive, giving rise to the *depressive form*. In subacute cases a condition of suspicion and anxiety, with systematized delusions of persecution, sometimes develops.

Diagnosis.—The diagnosis is to be made from the association of an acute confusion with the uræmic states. The subacute cases may mislead, as they have the outward semblance of chronic insanity. The history will, however, show an acute onset, and the physical examination will disclose evidences of renal disease.

II. **Diabetes.**—The mental disorder associated with diabetes is usually of a mild chronic type. It is invariably a depression with melancholic ideas of sin, ruin, and, usually also, hypochondriacal ideas, especially with reference to the excretion of sugar. There is liable to be marked somnolence with some confusion and disorientation in the semi-somnolent state. Persecutory delusions are quite frequently developed, ideas of poisoning and the like.

Diagnosis.—The persecutory type must be differentiated from the chronic psychoses. Otherwise the diagnosis is made by the association of the mental symptoms with glycosuria.

III. **Gastro-Intestinal.**—Certain cases of acute confusion develop, associated with a profuse offensive diarrhœa, a high grade of indicanuria, vomiting, low fever, and perhaps mild albuminuria. Some of these cases go on to acute delirium with high fever, typhoid state, profound exhaustion, coma, and death.

III. THYROIGENOUS PSYCHOSES

I. **Myxœdema.**—Associated with the physical symptoms of myxœdema is a mental state of stupidity, indifference, and apathy, deepening into dementia. There is gradual failure of memory, lack of power of voluntary attention, slow association of ideas, and difficulty of apprehension. Sometimes a moderate degree of confusion with excitement develops.

II. **Cretinism.**—Associated with the physical signs of cretinism is a mental state, due to lack of development, which may range all the way from the profound degradation of idiocy to mild grades of imbecility.

IV. TOXIC PSYCHOSES

I. **Alcoholism.**—**Drunkenness.**—Alcohol, like fever, may be said to be a measure of cerebral resistance. The unstable, predisposed individual becomes intoxicated much more readily than the normal person.

The phenomena of drunkenness are, from the first, those of paralysis. In the early stages it is only the higher psychic functions, which are largely inhibitive, that are affected, so we see apparent stimulation in the excitement produced, with flight of ideas, pressure of activity, loss of the sense of propriety, degradation of the moral tone, and loss of power of voluntary attention. The lower centres next become paralyzed, and then appears muscular inco-ordination, manifesting itself first in the hands and facial muscles and the muscles controlling articulation; the speech becomes thick, and the gait unsteady. Sensory disturbances are manifested, such as diplopia and tinnitus aurium, and the senses of touch and pain are blunted. If the paralyzing action of the alcohol continues, coma results, which may prove fatal. The mood during intoxication may be a pleasant one, and is frequently one of boisterous exaltation, constituting the *exalted type*; on the other hand, a sad, depressive, lachrymose mood may prevail, constituting the *depressed type*.

Pathological Drunkenness.—Among certain predisposed individuals alcohol produces unusual and much more severe symptoms. In this condition we may find hallucinations and delusions dominating the field of consciousness, the delusions being usually of a persecutory character. In other cases the excitement may issue in a wild maniacal frenzy, or the depression may be so profound as to result in attempts at suicide. In some persons the paralyzing effects of alcohol are unusually pronounced, and coma appears early on the scene. Those who have latent hysterical tendencies may have hysterical attacks during intoxication, while alcohol frequently produces convulsions in epileptics. Aside from this latter action, however, the convulsive properties of alcohol alone are capable of producing convulsions in persons who have long indulged and are profoundly degenerated.

Delirium Tremens.—This disorder usually occurs as the result of a prolonged drunken debauch, during which the patient has had insufficient food and rest. According to some authors it may result directly from the withdrawal of alcohol. It may, however, appear as the result of a single excess, or in the moderate drinker following a traumatism or the initial symptoms of an acute illness. The disease may appear suddenly, but there is generally a prodromal period during which the patient is nervous, with coated tongue, suffering from anorexia, restlessness, tremulousness, disturbed sleep, and insomnia. This condition rapidly advances with the onset of the attack, the characteristic symptoms of which are rapidly developed. They are tremor, delirium, and albuminuria.

The *tremor* involves more particularly the small muscles of the hand, face, and tongue, but may also affect the entire musculature. It is increased by muscular tension, such as forcibly spreading the fingers apart.

The *delirium* is an acute hallucinatory confusion. The predominating hallucinations are visual, and characteristically take on the form of animals. The patient sees all sorts of horrible creatures, snakes, rats, mice, alligators, etc., which are uniformly in motion. Surrounded by the loathsome creatures, and by horribly grimacing faces, terrified by screams and shrieks (auditory hallucinations), he presents a picture of abject terror. In addition to these symptoms the patient may complain that insects or worms are crawling under his skin (paræsthesia), and mistake spots upon the bed or walls for bugs, mice, etc. (illusions). At the height of his excitement the patient is in constant motion, picking insects from his nightshirt, repelling the approach of terrible animals, shrinking from fearful visions, startled by terrifying shrieks, and, in the extreme frenzy of his fright, he may make murderous assaults on those about him, believing them to be his enemies, or more commonly attempt his own life to escape from his horrible surroundings. During all this time the patient is constantly talking, shrieking in fear at times, at others carrying on an incoherent discourse with imaginary persons, fragments of which often relate to his former occupation and friends. Often dreamy hallucinations and delusions relate altogether to his occupation, and the patient busies himself with his usual pursuits—*occupation delirium*. Physically he is in a condition of acute exhaustion. The pulse is rapid and of low tension, the temperature normal or only slightly elevated (occasionally high, the *febrile delirium tremens* of Magnan), the body bathed in a profuse perspiration, and constantly agitated by muscular shocks and tremore.

Albuminuria is found in a considerable portion of cases, probably considerably over 50 per cent, during the early stages. At the height of the delirium *leucocytosis* has been found. Occasionally one sees cases ushered in by all the typical prodromal symptoms, sweating, atonic dyspepsia, restlessness, tremor, præcordial distress, anxiety, and disturbed sleep, which do not proceed to the typical condition of mental confusion with multiform hallucinations. This is the so-called *abortive type*, the *delirium sine delirio* of Döllken.

Diagnosis.—Similar symptoms are found practically in senile dementia and general paresis. They can be differentiated by the history, such cases affording characteristic symptoms before the outbreak of delirium.

Chronic Alcoholism.—The effects of chronic alcohol poisoning are exhibited in every organ of the body, more particularly the central nervous organs, stomach, pancreas, liver, kidneys, and blood vessels, and thus give rise to characteristic symptoms, the most prominent of which are tremor, gastric catarrh, arteriosclerosis, albuminuria, and progressive mental enfeeblement.

The effects on the nervous system are shown in disturbances of sensation, motion, and the intellect. The *sensory* disturbances are paræsthesia (prickling, tingling, formication), hyperæsthesia, and hyperalgesia, occurring usually in patches, and anæsthesia, also of patchy distribution but sometimes affecting only one side (the *hemianæsthetic form* of Magnan). The disorders of the special senses involve principally the eye and ear, producing illusions and hallucinations, muscæ volitantes, photopsia, amblyopia, and amaurosis; and diminution of the acuteness of hearing, with subjective noises (hissing, ringing, roaring) due to middle or internal ear disease.

The *motor* disturbances are tremor, spasms and cramps, epileptiform attacks, and general motor enfeeblement with paresis.

The *mental* changes are gradual and progressive, the intellect is obtunded, the judgment overthrown, the moral sense blunted, and mendacity appears in its most bizarre forms; delusions may develop, the most characteristic of which is that of marital infidelity and jealousy, and the patient gradually sinks into a condition of permanent mental enfeeblement.

Diagnosis.—Alcoholic dementia is to be differentiated from other dementias largely by the history. Alcoholic dementia will have a history of progressive mental enfeeblement closely associated with alcoholic indulgence.

Alcoholic Pseudo-Paresis.—On a groundwork of mental enfeeblement the alcoholic may develop a true expansive delirium which, combined with the signs of alcoholism (ataxia, speech defects, tremor, pupillary anomalies, and muscular weakness), may make the distinction from paresis difficult—alcoholic pseudo-paresis. This similarity to paresis is noticeable, even when the expansive delirium is absent, in cases in which the mental reduction is marked, but becomes greatest when the symptom complex above outlined is ushered in by epileptiform attacks.

Diagnosis.—The distinction from true paresis can usually be made. Pupillary inequality is more common and the permanent results of apoplectic insults (hemiplegia, aphasia) are more often found in the alcoholic than in the true form. The results of polyneuritis should be looked for and if found suggest alcoholism. The most reliable differential sign is found in the course of the two maladies. True paresis is progressive, tending toward ever-increasing degradation, while in the alcoholic form removal of the poison results very shortly in a remission of all the symptoms, even, in some cases, amounting to a recovery. The symptoms, however, reappear subsequently if drinking habits are returned to.

Alcoholic Epilepsy.—As a result of chronic alcoholic toxæmia, the symptoms of which are marked throughout by their explosive character, it is not strange that actual convulsions, alcoholic epilepsy, should complicate the morbid picture. These convulsions, so far as their individual characteristics are concerned, are indistinguishable from true epilepsy. Occurring, however, in a person beyond the period of adolescence, who is addicted to the immoderate use of alcohol, their origin should be suspected. The diagnosis is made clear if they cease upon the withdrawal of alcohol. As this sometimes does not occur the diagnosis can be made only by excluding the causes both of true and symptomatic epilepsy other than from alcohol.

Alcoholic Hallucinosis.—This psychosis may come on suddenly in a chronic alcoholic as the result of an unusual excess, or it may be of gradual evolution. It is sometimes preceded by one or more attacks of delirium tremens. It is characterized by hallucinations, auditory and visual predominating, with delusions of a persecutory nature in which the sexual element is frequently prominent.

Whether of sudden or gradual onset the first symptoms are hallucinations, with which persecutory delusions are intimately bound up. The patient hears voices making all sorts of inimical remarks, telling him that his children are not his own, calling him an onanist, reviling or threatening him. A voice is sometimes referred to the epigastrium (epigastric voice), and in every way his persecutors annoy him by their malign comments. Visual hallucinations, if they occur, are equally unpleasant. Hallucinations of smell and taste are not infrequent.

The delusions of this state harmonize well with the hallucinations. The patient is persecuted by invisible enemies who inject noxious vapors in his room at night, poison his food, draw off his semen, and produce nocturnal pollutions.

In this state of persecutory insanity, the patient may be alternately fearful of impending danger, have anxious and angry states, and often react by attacking his supposed persecutors. Throughout this condition the patient is well oriented and there is no clouding of consciousness.

Diagnosis.—The diagnosis from delirium tremens is made by the absence of disorientation, and by the marked prevalence of auditory hallucinations in the form of threatening voices. From paranoia the distinction is made by the very rapid systematization of the delusional system as opposed to the slow evolution of that disease.

Alcoholic Pseudo-Paranoia.—In some cases of chronic alcoholism a paranoid state is developed in which psycho-sensory disturbances (hallucinations) are of secondary importance or not present at all. The characteristic delirium in these cases is that of marked infidelity.

This delusion of marital infidelity and jealousy may not be accompanied by any noticeable degree of impairment of judgment or mental enfeeblement, and in these cases it may be extremely difficult to make a differential diagnosis between this form of alcoholic insanity and true paranoia. Particularly is it difficult to recognize paranoia with subsequent or coincident alcoholic indulgence.

Dream States.—Less common and more unusual effects of alcohol are the conditions of so-called *trance*, *automatism*, *double consciousness*, *spontaneous somnambulism*, which are followed by *amnesia*. In these conditions the subject of alcoholism may do almost anything imaginable, make contracts, transfer property, commit criminal acts, take long journeys, enter into complicated business or professional transactions, and later have absolutely no knowledge of what he has done. During a protracted debauch the subject may suddenly start off on a journey and travel under an assumed name, meanwhile conducting himself in such a manner as not to lead to any comment on the part of those whom he meets. Suddenly, without warning or after a night's sleep, he "wakes up" to a realization of his true situation with absolutely no memory of how he got where he is, or what he has been doing since he started away from home.

II. **Opiumism.**—The symptoms of a single dose are at first those of mild stimulation of the mental faculties, followed by a period of quiet, half-waking, half-sleeping, interrupted by multiform pleasant hallucinations (predominantly visual), which show no tendency to delusive elaboration in the waking state. This condition is followed by malaise, headache, dry mouth, constipation, and nausea.

Psychic disturbances in the chronic *habitué* develop more often as the result of abstinence than of continued use. They may be characterized by predominating depressive or exalted effects, or a paranoid state may develop. As in other toxic psychoses there are apt to be present more or less confusion and a tendency to multiform hallucinations. Dementia is a rare sequel.

Diagnosis.—The diagnosis can often be made without the anamnestic data. The patients frequently deny their habit—mendacity is a prominent symptom—and they are often shrewd enough to find means of indulgence, even though carefully watched. The moral degradation is pronounced and they will go any length to obtain their drug. Symptoms which should excite suspicion are periods of torpor and languor, in marked contrast to the activity of alcoholism, amounting at times to an inability to even sit up, occasional signs of stimulation, small pin-point pupils, yellowish-brown cachectic complexion, and, above all, the numerous scars of hypodermic injections.

III. **Cocainism.**—The symptoms resulting from the use of cocaine are those of marked stimulation. The pulse is increased and the pupils are dilated. The patients are active and extremely talkative, often repeating their remarks a number of times; they are constantly busy—some of them writing endless letters—and their whole appearance indicates an acute intoxication. The effects are, however, very fleeting and the dose has to be frequently renewed. Chronic addiction results in marked emaciation, cachectic anæmia, insomnia, sometimes epileptiform attacks, and various paræsthesias, the most marked of which is a sensation of crawling under the skin ("cocaine bug"). In the psychic sphere occur incapacity for mental application, lessened moral sense, mendacity, irritability, impaired judgment, and sometimes the delusion of marital infidelity. These symptoms may be followed by mental confusion with hallucinations, but more characteristically by a paranoid state. From true paranoia this is differentiated by the greater variety of delusions, those of paranoia being less variable and rather noticeable for their monotony. In the paranoid state of alcoholism (hallucinosis) on the other hand, the hallucinations are more stereotyped.

The abstinence symptoms are not so severe as with morphine and may not appear for several days. Erlenmeyer has called attention to a profoundly depressed, lachrymose, demoralized condition, with moaning and sighing, which may supervene. The persecutory delirium may persist for a long time and constitute the patient a very dangerous individual.

IV. **Miscellaneous Intoxicants.**—Various other substances may produce marked mental disturbances as a result of acute or chronic poisoning or habituation. The limits of this article permit only of their mention. They are chloral, cannabis indica, somnal, sulphonal, paraldehyde, ether, chloroform, antipyrin, phenacetin, trional, chloralamid, iodoform, belladonna, hyoscyamus, salicylic acid, quinine, carbon monoxid (illuminating gas), the preparations of lead, arsenic, mercury, and the bromides.

The mental effects of poisoning from all of these is in the main an acute hallucinatory confusion. Occasionally a paranoid state may be present.

V. PSYCHOSES OF OBSCURE OR UNKNOWN PATHOLOGY

I. **Paranoia.**—**General Characterization.**—Paranoia is a chronic, progressive psychosis characterized by systematized delusions of persecution, hallucinations of hearing, and showing little or no intellectual impairment.

Symptoms.—The disease is typically regularly progressive through three stages: 1. The stage of subjective analysis. In this stage the patient worries about himself and develops *hypochondriacal ideas*. He also begins to interpret everything that goes on about him as having some reference to himself—*delusions of relativity*. 2. The stage of persecution. This stage is characterized by *hallucinations of hearing* and *systematized delusions of persecution* re-enforced by *retrospective falsification of memory*, the patient going back through the experiences of his early days and finding various events there which are explicable in the light of his delusional system. 3. The stage of transformation of the personality. In this stage the patient develops ideas of self-importance. These three stages are intimately connected, and the system of delusions is elaborate and supported by detailed logical argument.

In some cases there is strictly no persecutory stage and the grandiose ideas are developed primarily, constituting the *expansive* form of the disease. These cases may be free from hallucinations. They are the inventors, reformers, and religious fanatics who often become very well known.

Diagnosis.—The disease is diagnosed by its long course, extending over several years as a rule, the presence of elaborately systematized delusions which are supported by logical argumentation, the presentation of the form of thought, absence of intellectual impairment, and regular progressive course. It is to be differentiated from the paranoid forms of other psychoses.

II. **Manic-Depressive Insanity.**—This disease manifests itself by recurrent attacks, which are usually either of an exalted or of a depressed type, and these attacks, until recently, were supposed to be distinct diseases. The separate attacks are recovered from, but tend to recur, often with marked periodicity. There is no apparent intellectual enfeeblement, even after a considerable number of attacks.

The disease occurs in two distinct stages, the manic and depressive, but attacks may occur partaking of the symptoms of both.

Manic Stage.—The characteristic symptoms of this stage are: (*a*) flight of ideas; (*b*) psychomotor excitement; (*c*) emotional exaltation.

Depressive Stage.—The characteristics of this stage are: a. difficulty of thinking; b. psychomotor retardation; c. emotional depression.

Mixed States.—There are three principal mixed states, as follows:

Maniacal Stupor.—Emotional exaltation, decreased psychomotor activity, difficulty of thinking.

Agitated Depression.—Emotional depression, increased psychomotor activity, flight of ideas.

Unproductive Mania.—Emotional exaltation, increased psychomotor activity, difficulty of thinking.

The Periodical Insanities.—The manic and the depressive stages, in any degree of severity, may occur combined or not with a lucid interval in any possible series of relation to each other. The more common of these are as follows:

Recurrent Mania.—Recurrent manic attacks, separated by lucid intervals.

Recurrent Melancholia.—Recurrent depressive attacks, separated by lucid intervals.

Circular Insanity.—Regular alternation of manic and depressive attacks, without lucid intervals.

Alternating Insanity.—Regular alternation of manic and depressive attacks, each attack followed by a lucid interval.

Insanity of Double Form.—Regular attacks, each attack consisting of both a manic and a depressive stage, and each followed by a lucid interval.

III. **Dementia Præcox.**—*General Characteristics.*—Dementia præcox is a psychosis essentially of the period of puberty and adolescence, characterized by a dementia tending to progress, though frequently interrupted by remissions. Upon the foundation of dementia are erected various psychotic symptoms, many of which show a marked tendency to episodic manifestations.

General Symptomatology.—The disease usually begins with a period of depression, and also often of temporary confusion. After this initial stage the symptoms are those of progressive dementia. These symptoms are: *failure of voluntary attention; lack of interest* shown by such patients both with reference to themselves and their surroundings; *failure of memory*, particularly for recent events; *emotional deterioration* manifested by an absolute lack of emotional reaction upon such occasions, for example, as the death of a parent or near relative. All these evidences of deterioration occur without loss of orientation or clouding of consciousness.

In connection with these mental symptoms certain psychomotor symptoms occur, particularly in katatonia—more especially muscular tension and negativism—which also show the fundamental unity of the different types.

This disease occurs in four fairly distinct forms, although many transition cases occur.

Simple Dementia (*Heboidophrenia*).—This form manifests more the simple dementia uncomplicated by formal psychotic symptoms. It usually begins with a period of depression, and during its course various delusions and hallucinations may occur. There may be slight transitory excitement, and some evidence of psychomotor disturbance—slight muscular tension, mannerisms, peculiar habits.

Diagnosis.—The principal thing in diagnosis is to differentiate the early stages from melancholia and neurasthenia, which they resemble.

Hebephrenia.—In this form there is also usually a prodromal period of depression. Delusions and hallucinations are more prominent and partake of absurd characteristics, indicating an underlying defect. There is *poverty of ideas*, and also a certain *looseness* of train of thought, which simulates quite often a flight of ideas. A high degree of incoherence is not infreqnent.

Diagnosis.—As in the simple demented type, the principal differentiation is to be made, in the first stage, from melancholia and neurasthenia.

Katatonia.—This form may come on suddenly, but is usually of slow onset with symptoms of depression. During this prodromal period hysterical attacks and epileptiform convulsions may occur. In its developed form the disease occurs in two stages:

Katatonic Stupor.—This stage is characterized by *stupor* of varying degrees, with marked evidences of *muscular tension* and *negativism.* The negativism may result in mutism, retention of urine, fæces, and saliva. We find a different picture with *flexibilitas cerea, command automatism, echolalia, and echopraxia.* The patient may be well oriented, without any material clouding of consciousness.

Katatonic Excitement.—In this condition there is much *psychomotor excitement.* The patient is in constant activity, often throwing himself about in the bed, shouting and talking. A high grade of incoherence is common, and verbigeration is quite frequent.

Diagnosis.—The early stages, as in the other forms, must be differentiated from melancholia and neurasthenia. The stupor is to be distinguished from the stupor of the depressive stage of manic-depressive insanity by the presence of muscular tension, negativism, or catalepsy. The excitement is to be distinguished from the manic stage of manic-depressive insanity by the evidences of dementia, the presence of verbigeration, stereotypy of movement, grimacing, and the fact that the incoherence is out of all proportion to the degree of excitement.

Paranoid Forms.—These conditions exhibit a more or less well-organized system of delusions and hallucinations, but with evidences of dementia or early deterioration.

Diagnosis—The diagnosis is to be made from paranoia by the evidences of dementia, particularly shown in the absurd characters of the delusions and their loose systematization, and by the presence of some of the signs of psychomotor disturbance characteristic of this disease.

Mixed Forms.—Transition cases are frequently seen. The characteristic association of a degree of dementia with the typical psychomotor disturbance determines the diagnosis.

VI. PSYCHOSES OF KNOWN PATHOLOGY

Paresis.—**General Characterization.**—General paresis is an organic disease of the brain, of an inflammatory and degenerative nature, including in the main the leptomeninges and the cortex, and manifesting itself by certain physical symptoms and a progressive mental deterioration upon which may be engrafted various other symptoms of mental disturbance.

The disease is arbitrarily divided into three periods:

First Period.—The physical symptoms that may be found during this period are Argyll-Robertson pupil, loss of consensual light reflex, loss of sympathetic light reflex, and some abnormality of the patellar tendon reflexes. The mental symptoms are gradual change of character, failure of memory, inability to work continuously, lack of judgment, failure of moral sense, with perhaps addiction to alcohol. The speech may begin to show the stumbling, slurring defects of the later stages, as may also the writing. Tremor, especially of the facial muscles, may also begin to show.

Second Period.—This stage is ushered in by the so-called paretic seizures—apoplectiform or epileptiform attacks. The physical symptoms of the first stage are aggravated. The mental are those of deepening deterioration. If the symptoms are only those of dementia, the case belongs to the *demented type*. Grandiose delusions may occupy the field, and such cases constitute the *expansive type* of the disease; while if the excitement is very great there develops the *agitated type* or, with depressive delusions, the *depressed type*.

Third Period.—This period begins when the patient becomes filthy in his habits. The physical symptoms are all aggravated, the patient is soon bedridden, and the mental life is reduced to the point of practical extinction.

Diagnosis.—If we find a patient in middle life with Argyll-Robertson pupils and abolished knee-jerks, and tabes can be eliminated, we may strongly suspect paresis. If in addition we learn that there has been a change of character in the individual, if he has become irritable and restless, fails to remember business engagements, is less careful of his personal appearance, and presents, in short, a host of minor symptoms which individually mean little but in their *ensemble* speak for mental deterioration, the diagnosis is rendered practically certain.

The principal diseases for which paresis may be mistaken are acquired neurasthenia, some one of the psychoses, alcoholism, brain tumour, cerebral syphilis, disseminated sclerosis, arteriosclerotic insanity, and Korsakoff's psychosis.

In differentiating paresis from acquired neurasthenia the general mental attitude of the patient is of great significance. Whereas the neurasthenic is given to exaggerating his ills, constantly complains of his aches and pains, and keeps close observation of every change of symptoms, the paretic is usually indifferent or may, on the contrary, consult a physician under protest and in the firm belief of the uselessness of so doing, as he feels so well. In addition to this there is in neurasthenia no dementia, no disturbance of speech or writing, no history of seizures, the tendon reflexes are equal and not abolished, the pupils equal, respond to light and accommodation, and are more apt to be dilated, while in paresis they are frequently unequal and often very much contracted.

From alcoholism the diagnosis is often not so easy. The deterioration of the chronic alcoholic has much in common with the dementia of paresis. We must turn to the physical signs and note carefully the historic facts. Following a long debauch, however, symptoms may arise which, in the absence of a history, would warrant a diagnosis of paresis—the so-called alcoholic pseudo-paresis. These symptoms disappear though, in a remarkable manner, when the alcohol is withdrawn.

Symptoms occasionally develop in the course of brain tumour which closely resemble paresis. The diagnosis must be made on the pre-eminently focal character of the physical signs in the former disease.

From disseminated sclerosis the differentiation is sometimes difficult. The combination of intention tremor, nystagmus, scanning speech, and spasticity will, however, usually leave little room for doubt, although some of these cases do ultimately develop the typical signs of paresis.

From cerebral or cerebro-spinal syphilis the diagnosis is again quite difficult. If the lesion is a gummatous meningitis the signs are rather of multiple lesions than a diffuse process. If, on the other hand, the disease affects principally the vessels, with resulting endarteritis obliterans, thrombosis, and softening, the symptoms are focal, and convulsions developing afterward constitute a true post-apoplectic epilepsy. Disturbances of speech and writing either are not present or, if they are, do not partake of the nature of a paretic disorder, but are true aphasias due to focal lesions. Palsies, if present, are permanent and nocturnal headaches common. Antisyphilitic remedies produce immediate results in many cases.

From arteriosclerotic insanity the diagnosis is often quite difficult. The extreme age of these patients and the presence of a high grade of arteriosclerosis will usually indicate the correct interpretation of the symptoms.

Korsakoff's psychosis is to be differentiated by its history of polyneuritis, and, usually, of alcoholic excess or other causes of the neuritic symptoms. If the patients live they do not proceed to the profound dementia of paresis. The Korsakoff syndrome is occasionally found associated with paresis.

VII. PSYCHOSES ASSOCIATED WITH OTHER DISEASES

The Neuroses.—The so-called neuroses, especially hysteria and neurasthenia, might properly be classed with the psychoses and designated as psycho-neuroses, as mental symptoms are almost invariably present and, in hysteria and neurasthenia especially, properly form part of the symptom-complex.

I. **Hysteria.**—The mental symptoms of hysteria may be divided into these constant phenomena, which are present throughout the course of the malady: the symptoms of the *interparoxysmal period*, the so-called *mental stigmata*; and those more or less closely connected with the paroxysms—the *episodic phenomena*.

Mental Stigmata.—The principal symptoms of the interparoxysmal period are anæsthesia (disseminated, segmental, hemianæsthesia), *hyperæsthesia*, usually disseminated; *motor disturbances*—contractions, catalepsy, paralysis; *amnesias*—partial or general; and *debility of the emotions—loss of will power, suggestibility.* It must be fully understood that all these symptoms, even the sensory and motor disturbances, are purely mental.

Episodic Phenomena.—These phenomena may precede or follow an hysterical crisis, or as in epilepsy, may be substituted for one. They are principally states of *exaltation*, *depression*, *delusions*, *lethargy*, *ecstasy*, *somnambulism*, *fixed ideas*, *delirium*, and *choreiform movements.* Conditions of delirium with great confusion, clouding of consciousness, and hallucinations are common. Dream states are also quite characteristic of this disease, as they are of epilepsy and alcoholism. It will often be found that the crises of hysteria are associated with certain subconscious ideas, usually connected with some previous experience having a large content of painful emotion, which has been forgotten.

A characteristic of these episodic manifestations is their very frequent association with amnesia.

Diagnosis.—Epilepsy is the most difficult disease from which to differentiate hysteria. This is particularly so because of the convulsive attacks in each. The diagnosis must often rest upon the presence of the hysterical stigmata in the interparoxysmal period, as the attacks are often not seen and can not be distinguished by the description given. The presence of these stigmata will usually suffice, as hysteric convulsions and true epileptic convulsions seldom occur in the same patient. Hystero-epilepsy is not a combination of the two diseases, but hysteria with associated epileptiform attacks.

The differentiation from the other psychoses is to be made from the history and the presence of the hysterical stigmata.

II. **Neurasthenia.**—*Symptoms.*—Like those of hysteria, the manifestations of neurasthenia are protean and numerous. The disease may be hereditary, constituting so-called *constitutional* neurasthenia; or it may be *acquired* by exhausting and debilitating conditions, usually acting over a considerable period. Symptoms of hysteria are not infrequently combined with those of neurasthenia, constituting hystero-neurasthenia. Neurasthenia is usually classified in accordance with the organs most prominently affected, into *cerebral*, *spinal*, *genital*, *gastric*, *angiopathic*, or in accordance with the cause, as *lithæmic* and *traumatic*.

The symptoms are those of *fatigability* and *depression*, with special symptoms associated with the viscera, such as dyspepsia, diarrhœa, headache, various paræsthesias, particularly band-like and pressure sensations of the head. The depression is often associated with hypochondriacal ideas, phobias, obsessions, fixed ideas, and impulsions.

Diagnosis.—The diagnosis must be made from the early stages of paresis and dementia præcox, and from the depressive stage of manic-depressive insanity.

III. **Epilepsy.**—The mental disturbances associated with and due to epilepsy may be considered as divided into the paroxysmal and the interparoxysmal.

The *paroxysmal* disturbances are either associated with the attack—unconsciousness, psychic epilepsy (dream states, automatism, furious maniacal attacks followed by complete amnesia); or else with pre- and post-epileptic attacks, often of a maniacal order, but frequently, especially following an attack, being delirious. Amnesia follows all these states.

The *interparoxysmal* disturbances go to make up the epileptic character. They are in the main transitory attacks of ill-humour, sometimes violent temper, and impulsiveness, associated with a certain degree of dementia manifested by dulness, apathy, and unreasonableness, frequently with religious fervour. Delusional states not uncommonly occur. If the epilepsy has begun in early life a degree of imbecility or idiocy results.

Diagnosis.—These various mental conditions are diagnosed by a careful study of the history, and the presence of undoubted epileptic convulsive seizures. Differential diagnosis must be made from hysteria, katatonia, the delirium of alcohol and other intoxicants and infections, and from the delirious states of paresis.

Other Nervous Diseases.—I. **Polyneuritis** (*Korsakoff's Psychosis*).—The mental state of this psychosis accompanies polyneuritis and is usually of alcoholic origin, but may be caused by other poisons, as those of typhus fever, tuberculosis, or influenza; and the Korsakoff syndrome is seen not infrequently in general paresis. The signs of polyneuritis may be very slight.

Symptoms.—The patient is amnesic both for events in the immediate past and for the whole period of time during which he has been ill. This defect of memory is associated with a composed bearing and apparent lucidity on casual questioning. A more careful examination, however, will show not only this memory defect, but probably also that the patient is disoriented as to time and place.

The characteristic symptom is associated with the amnesia, and consists of a peculiar *falsification of memory*. The gaps in memory are filled by all sorts of *fabrications*, which are narrated in great detail and with a perfect appearance of lucidity. A patient who has been confined to his bed for days with foot and wrist drop tells, when asked where he was the day before, about having gone to the races and details his conversation with different persons, describes the events, tells what horses won, and the like.

Diagnosis.—The association of the peculiar falsification of memory with fabrication and, usually, disorientation, with foot and wrist drop is characteristic. Paresis is to be distinguished by the absence of evidence of polyneuritis.

II. **Chorea** (*Sydenham's*).—Patients with chorea are usually impatient, irritable, and emotionally unstable. Some of the cases develop terrifying dreams and hallucinations, especially at night. Marked psychotic symptoms become manifest in the variety of the disease known as *chorea insaniens*; an acute confusion, sometimes of violent type, is seen, with hallucinations and often a paranoid condition with delusions of persecution. Sometimes a condition of stupor is observed.

Diagnosis.—The diagnosis is made by the association of the mental symptoms with the characteristic choreic movements.

III. **Huntington's Chorea.**—This disease is associated on the psychical side with gradual mental impairment.

IV. **Exophthalmic Goitre.**—This disease is not infrequently associated with hallucinations of hearing, voices being heard saying disagreeable things to the patient. With these hallucinations occur anxious and agitated states. The prognosis in these cases is bad. Many of them die.

V. **Paralysis Agitans.**—This disease is often associated with a mild degree of mental enfeeblement.

VI. **Multiple Sclerosis.**—The mental condition is usually one of slight impairment, especially with emotional instability. The patient laughs and cries very easily.

Diagnosis.—The disease must be differentiated from paresis, which it often closely resembles.

Organic Diseases and Injury of the Brain.—I. **Tumour.**—Mental symptoms are more apt to occur when the tumour is located in the prefrontal region. They are change of character, irritability, childishness, emotional instability, with a tendency to hebetude, and some clouding of consciousness. Hallucinations may develop as the result of the invasion of sensory areas by the growth.

Diagnosis.—The diagnosis is made from the presence of the classical symptoms of tumour and a study of the localizing symptoms.

II. **Syphilis.**—An acute delirium may develop during the early secondary manifestations of the disease, later marked manifestations may be connected with local or general disease of the brain. Gummata are rare and present the symptoms of tumour. The most common condition is a progressive disease of the cerebral vessels, often with thrombosis.

The mental symptoms are those of dementia, to which are added local symptoms, if softening has occurred, depending on the location of the affected area. These cases may go on for some time and finally develop the typical symptoms of paresis.

III. **Apoplexy.**—The mental condition following apoplexy is usually one of impairment which, if the lesion is considerable, progresses to marked dementia. If the softening involves the speech area, especially if it produce sensory aphasia, the dementia is much more rapid in progress, as it must be remembered that these patients are usually also senile.

IV. **Arteriosclerosis.**—This condition is associated with a progressive failure of the mental faculties, and with local symptoms due to areas of softening. The picture often closely resembles paresis, but the patient is much further advanced in years than is usual for the paretic.

V. **Traumatism.**—The most frequent symptoms following trauma are those of hysteria and neurasthenia. Dementia præcox, manic-depressive insanity, and paresis may follow an injury.

After the injury a delirium may develop. Aside from this, mental symptoms may not occur for a considerable time and when they do they usually consist of an apathetic dementia, often with irritability. Frequently there are memory defects, especially amnesia for the time of the injury, and these defects may be filled in with fabrication. There is almost always intolerance of alcohol.

VI. **Meningitis** and **Insolation.**—These also may be followed by a degree of mental defect.

Diseases Other than Nervous.—Various other diseases have from time to time mental symptoms associated with them. The great majority of such diseases, if not all of them, have elements of infection or toxæmia, and exhaustion combined with or a part of them. We therefore see the mental symptom complex of confusion arise most typically. In some cases, especially, the less acute paranoid conditions appear, and hallucinosis is of occasional occurrence.

Head has shown that certain visceral diseases, especially of cardiovascular and pulmonary origin, often have associated mental symptoms, although they may not appear except on the most careful examination. The symptoms found are: (*a*) hallucinations of vision, hearing, and smell; (*b*) moods, either of depression or exaltation, and (*c*) suspicions, usually occurring when a depression has persisted for some time.

These conditions take their origin as a result of reflected visceral pains. Each spinal segment has both a visceral and a cutaneous representation. Disease occurring in the visceral area is referred to the cutaneous surface supplied by the same segment. The cutaneous distribution of the fifth nerve corresponds to the visceral distribution of the vagus, so pain occurring in the vagus territory will be referred to the scalp and thus occur points of tenderness in this region with which the hallucinations are associated. The mood of exaltation is essentially transitory, arising as a contrast phenomenon of the depression, and is a result of the disappearance or lessening of the reflected somatic pain.

VIII. PSYCHOSES OF THE INVOLUTION PERIOD

I. **Melancholia.**—This disease occurs usually between 40 and 50 years of age in women, rarely before 50 in men. It is characterized by emotional depression, anxiety, and apprehension coming on after a prodromal period which may be of several months' duration. The symptoms of this period are indefinite and consist largely of peculiar sensations referred to the head, pressure, vertigo, tinnitus, insomnia, irritability, and depression. This depression deepens, there are typical melancholy ideas, such as ideas of sin, and finally a marked degree of apprehension and anxiety may occur. In these latter cases the patient is constantly active, wringing the hands, moaning, and crying—*agitated melancholia.* Ordinarily the patient is fully oriented, but in extreme cases confusion may develop with disorientation and clouding.

The advent of senescence may introduce an element of dementia which modifies the picture.

Diagnosis.—The principal disease to be distinguished is manic-depressive insanity. This is usually possible by the absence of retardation. In some cases, however, this is present and the distinction must be made by the age, and absence of previous attacks.

II. **Pre-Senile Insanity** (*Senium præcox*).—Arbitrarily the senium is said to begin at 60 years of age, and cases which show senile changes before this time are said to be pre-senile.

Pre-senile insanity is rare and usually takes a delusional form, of a persecutory and hypochondriacal character. There is usually some dementia present, which may give the delusions an absurd type.

III. **Senile Insanity.**—Aside from the ordinary decay of the mental faculties incident to old age, active psychotic symptoms may develop which constitute a veritable psychosis.

Delusions are the rule, although as they are erected upon a demented foundation they are apt to be absurd and to show little tendency to systematization. Emotional deterioration is also present and the mood is marked by silly and childish characteristics. The memory defect is well defined and consists typically of amnesia for recent events. These patients are given to wandering, especially at night, as they often suffer from insomnia, and at these times show much confusion. The Korsakoff syndrome may also be present.

Cases which follow an uncomplicated course are spoken of as *simple senile deterioration*; if confusion dominates the picture it constitutes the *confusional type*, and a paranoid type may also occur. *Senile delirium* often terminates the case. It may be the result of a terminal infection. Epileptic attacks are episodic manifestations.

Diagnosis.—The diagnosis is made by the presence of a psychosis showing evidences of dementia in a person having the signs of senile decay. The occurrence of the Korsakoff syndrome is a possibility.

IX. BORDERLAND AND EPISODIC STATES

Phrenasthenia.—A psycho-neurosis tending to chronicity, but often marked by remissions, and exhibiting the following symptoms:

1. *Obsessions.*—These are of two classes—the obsessions of doubt and the various phobias (mysophobia, claustrophobia, panophobia, etc.).

2. *Morbid Impulsions and Compulsions.*—Tendencies to acts which are more or less out of the voluntary control of the patient. These include the various "manias" (kleptomania, pyromania, etc.).

3. *Tics.*—Essentially psychic in origin and occurring in persons with a mental condition which has been termed "infantilism."

Pathological Traits of Character.—Various forms of pathological character are found, such as the different types of so-called "cranks"; those who suffer from *constitutional inferiority*; those who are naturally, and more or less constantly, sad and depressed—*psychopathic depression*; the various types of suspicious individuals who may have tendencies to litigation; and a host of other ill-balanced defectives.

Sexual Perversions.—The principal manifestations are:

1. *Homosexuality.*—Sexual attraction for persons of the same sex.

2. *Active Algolagnia* (Sadism).—The gratification of the sexual feeling by the infliction or sight of pain—real or simulated. In the latter case the sadism is *symbolic*, and is more common in men.

3. *Passive Algolagnia* (Masochism).—The gratification of the sexual feeling by suffering pain, real or simulated. In the latter case it is *symbolic*. More frequent in women.

4. *Fetichism.*—Sexual excitement and gratification by the sight or contact of some article of wearing apparel or a part of the body.

5. *Exhibitionism.*—Sexual gratification by exposing the genital organs.

6. *Necrophilia.*—The desire to have sexual congress with a dead body.

X. IDIOCY AND IMBECILITY

Idiocy.—A condition of profound mental defectiveness either congenital, or acquired during the developmental period.

Idiots may be broadly separated into the apathetic and excitable. The following is a classification based upon pathology (Ireland):

1. Genetous (cases whose pathology can not be determined antemortem). 2. Microcephalic. 3. Hydrocephalic. 4. Eclampsia. 5. Epileptic. 6. Paralytic. 7. Traumatic. 8. Inflammatory (the result of encephalitis). 9. Sclerotic. 10. Syphilitic. 11. Cretinism (including the endemic and sporadic or myxœdematous forms), and 12. Idiocy by Deprivation.

Idio-Imbecility.—A condition midway between idiocy and imbecility.

Imbecility.—A condition of mental deficiency which can, however, be materially improved by training, but not sufficiently to take a place in the world. *Moral Imbecility* is a condition of mental defectiveness which is shown in the absence of the highest functions, particularly the moral, capable of training to a considerable degree, but always a menace to society.

Feeble-Mindedness.—A condition of slight mental defectiveness capable of much improvement by educational methods. The afflicted individual may ultimately take a place in the world, and be self-supporting under favourable circumstances.

SECTION IX

DISEASES OF THE MUSCLES

PREPARED BY HENRY GOODWIN WEBSTER, M. D.

I. **Myositis.**—Dujardin-Beaumetz recognises three varieties:

Symptoms.—(1) *Simple Acute Form.*—Characterized by lassitude and mild constitutional disturbance, with pain and tenderness in one or several muscles, coming on after unusual exertion and exposure. After a few days it resolves or passes into (2) or (3).

(2) *Acute Primary Infectious Form.*—Ushered in like (1), or succeeds it. There are lassitude, prostration, and muscular pain. The

affected muscle is prominent, sensitive to pressure, and either of a wooden hardness or soft and doughy. The overlying skin may be reddened or edematous. When deep muscles only are involved, the local signs fail, and the diagnosis rests upon the constitutional disturbance. When suppuration occurs fluctuation often appears. There are fever (even to 104° F.), dry, coated tongue, free sweating, dyspnœa, loss of appetite, and perhaps a vesicular eruption over the affected part. Fatal cases develop diarrhœa and the typhoid state.

(3) *Primary Acute Infectious Polymyositis* differs from (2) in its long prodromal period—3 to 5 weeks of lassitude and wandering pains—its extensive distribution, and the absence of suppuration. There are distinct chills, œdema of face and extremities, pain (general), and redness, the latter a fine macular eruption on the extremities, face, and abdomen. Gastric disturbances supervene, the œdema becomes hard, the patient rigid, and the muscular reactions and reflexes disappear. There are profuse sweats, intense thirst, coated tongue, constipation, scanty and albuminous urine. The entire muscular system becomes involved and death ensues.

Course and Prognosis.—Form (2) lasts from 5 to 20 days. A number recover. The course of form (3) is longer—3 or 4 months—and few recover. Recovery from form (1) is the rule, but it may run into the infectious variety.

Diagnosis.—Acute articular rheumatism, typhoid fever, suppurative arthritis, pyæmia, osteomyelitis, and other acute suppurative conditions may simulate the acute infectious variety, but the peculiar condition of the muscle should make the diagnosis clear. A localized cellulitis may be distinguished by being more superficial, and by the involvement of the neighbouring lymphatics. The distinction between polymyositis and trichinosis is difficult, and microscopic examination of a section of the muscle may be required.

II. **Chronic Localized Myositis.**—Some authors emphasize the appearance in the substance of the skeletal muscles, particularly the deep extensors of the trunk, shoulders, and neck, of hard nodules varying in size from .5 to 5 or 6 cm., and in consistency from elastic resistance to cartilaginous hardness, and claim a causative relation with certain forms of occipital headache, lumbago, and sciatica.

III. **Myositis Ossificans Progressiva.**—This rare disease appears as a tumefaction of the muscles at the back of the neck. The overlying skin is somewhat reddened, and there is slight fever. After the swelling subsides the muscle is found to be permanently hardened, and a progressive substitution of bony for muscular elements takes place. Muscle after muscle is invaded until the entire system is involved. After a year or more death ensues.

IV. **Myotonia** (*Thomsen's Disease*).—(*a*) *Symptoms.*—This affection is met with in Norway, Sweden, and Germany, but is rare in America. It is hereditary; appears in childhood; often attacks several members of a family; and is characterized by tonic contraction of the voluntary muscles, especially those of locomotion, prehension, and speech. It is noticed that the child is clumsy, that it can not perform the nicer movements, but that after a time the spasm relaxes and these motions may be perfect. This is also true for relaxation, the patient being unable to relinquish his grasp until some seconds after he has willed the action. The arms and legs suffer most, the eyes, face, and vocal organs least, while sensation and the reflexes are undisturbed. There is occasional mental hebetude. Cold and excitement increase the disorder. Although the muscles are unusually large, they lack power, and present "Erb's myotonic reaction." Either current causes a normal contraction, which, however, develops and relaxes very slowly, while during the interval slow, wavelike contractions occur, passing from cathode to anode. The disease pursues a chronic course, with remissions, and is incurable.

Diagnosis.—May be made from pseudo-hypertrophic paralysis by the gait, the absence of paralysis, and the myotonic reaction.

V. **Paramyoclonus Multiplex** (*Myoclonia*).—Friedreich's disease must not be confused with the hereditary ataxia of the same authority. It occurs most commonly in young men; affects the muscles, especially of the hands and feet, and is marked by clonic contractions, continuous or paroxysmal. It may follow severe mental or physical strain. The muscles are subject to rapid symmetrical, usually rhythmical, clonic spasms. Sensation is undisturbed, and during sleep the movements cease. The affection may be general, and the rapid contraction cause the body to be thrown to and fro with great violence. Voluntary efforts at control often serve to intensify the spasms. Féré reports cases of improvement under treatment as well as a few of spontaneous cure.

SECTION X

CONSTITUTIONAL DISEASES

PREPARED BY HENRY GOODWIN WEBSTER, M. D.

I. **Chronic Rheumatism.**—Cold, dampness, and exposure commonly predispose, although it may follow an acute attack. Usually there is little but pain and stiffness of the joints (page 101), although there may be tenderness, redness, and swelling. All symptoms are aggravated in the morning, gradually wearing off during the day. The disease attacks many joints, passing from one to another, with alternating periods of quiescence and exacerbation, which are often dependent upon changes in the weather. One joint only may be attacked. Ultimately marked changes in and about the joint with muscular atrophy and great deformity may occur. In severe cases there may be constitutional disturbances, especially gastric disorders and anæmia. As a rule the disease grows steadily worse in spite of treatment.

II. **Muscular Rheumatism.**—Pain in the muscles and their attachments coming on suddenly after exposure and cold, and known, when occurring in definite locations, as lumbago, torticollis, pleurodynia, etc. Fever is usually absent. Generally the pain is aching, sometimes severe, cutting, or cramping, and may be constant or occur only upon use of the affected muscles. As *lumbago* it attacks the muscles of the lumbar regions and lower back; as *torticollis* it affects the neck, usually the trapezius and sterno-mastoid with their immediate neighbours, generally on one side only; as *pleurodynia* the intercostals suffer. The latter is particularly distressing, as respiratory movements and coughing aggravate it. The back, shoulder, arm, or abdominal muscles may be involved. Pleurodynia is differentiated from pleurisy by the lack of physical signs; from intercostal neuralgia by the more general and constant pain and the presence of tender nerve points.

III. **Diabetes Insipidus.**—The diagnosis depends upon the persistent polyuria and the *urinalysis* ((15), page 711). Nervous or physical shock, violent exertion, or excess, may predispose. The onset may be acute, more often gradual. The patient usually enjoys good health, but notices a large output of urine and corresponding thirst, usually without bulimia. The condition may persist for years, the patient passing from 6 to 12 times the normal daily quantity of urine. While polyuria may be a symptom in a number of definite conditions, such as cerebral or medullary disease, meningitis, and disorders of the abdominal viscera, including tuberculous peritonitis and tumours, true diabetes insipidus occurs as a well-marked disease, probably due to vasomotor disturbance of the renal vessels.

Differential Diagnosis.—From diabetes mellitus by the low specific gravity and absence of glucose; and from chronic interstitial nephritis by the presence of albumin and casts, arteriosclerosis, and cardiac hypertrophy, with high-tension pulse. Hysterical subjects occasionally pass large quantities of pale clear urine (*urina spastica*), and nervous excitement not infrequently produces similar phenomena. The brief duration should make the diagnosis clear.

Prognosis.—Patients may live to the normal age limit without particular discomfort, or may fall victims to intercurrent disease. Occasional cases of spontaneous cure occur. The prognosis of symptomatic polyuria is dependent on the nature of the causative lesion.

IV. **Diabetes Mellitus.**—*Types and Symptoms.*—This disease may manifest itself in a variety of ways. The *urinary characters* are given in (16), page 711. See also 8 (*a*), page 710.

(1) A small number of cases are acute in onset and course. Many of these patients have suffered severe nervous shock, and nearly all are young adults with a family history of diabetes, rheumatism, or syphilis. This is the type seen in children. The symptoms are typical —increased urine, glycosuria, ravenous hunger and thirst, rapid failure of flesh and strength, general itching, irritability of temper, and sleeplessness. Such cases, as a rule, prove rapidly fatal.

Hirschfeld describes an acute type analogous to experimental pancreatic diabetes in which polyuria is less common or absent, and the assimilation of fats and albuminoids is markedly defective.

(2) More often the severe type of diabetes begins insidiously, and even after a year or more the nervous symptoms, debility, and loss of flesh may be attributed to neurasthenia. Loss of sexual power, impaired vision, pulmonary troubles, eczema, and other complications gradually develop, while the urine, although containing immense amounts of sugar, is not sufficiently increased in quantity to attract attention. Finally, the characteristic symptoms appear, and the patient either enters upon a chronic remitting course, lasting for a few years, with mental, pulmonary, or digestive disorders, and is carried off by intercurrent disease; or progresses rapidly to a fatal termination. Toward the end carbuncle, gangrene, myocarditis, or tuberculosis are common, and diabetic coma ends the scene.

(3) Possibly the most frequent form is that appearing in fat or lithæmic patients. The train of symptoms is much the same as in (2), but the patients respond more readily to treatment, and live in comparative comfort for many years, the sugar in some cases disappearing entirely. Chronic interstitial nephritis often succeeds.

All degrees between these types may be met with.

Complications.—Many and important. In the kidneys there may be a diffuse or chronic interstitial nephritis. Albuminuria is frequent; uræmia less common. In the liver there is often hypertrophy, rarely pigmented cirrhosis; sometimes gallstones. In the lungs tuberculous disease is common; acute lobar pneumonia, infarction, and gangrene occur. Dilatation of the stomach is met with, as well as bulimia and polyphagia. Stomatitis, gingivitis, caries, and loss of teeth are frequent, the tongue is often dry and red, and the saliva scanty. The pancreas is not uncommonly diseased. In the nervous system, melancholia and insanity are referable to the higher functions; neuritis and the various forms of paralysis are frequent; trophic changes, such as onychia and glazing of the skin, bronzing of the skin (*diabète bronzé*), late in the disease, and due to hæmochromatosis, occur; and most important of all, diabetic coma (see 8, page 79), caused by β-oxybutyric acid circulating in the blood, often heralded for a day or two by the sudden appearance of a large number of tube casts in the urine. Death usually follows after 36 hours. Of the special senses, ocular symptoms are most frequent, including retinitis, optic neuritis, cataract, amaurosis, iritis, and opacities of the vitreous humour.

Intense itching, especially about the external genitals, is the most annoying skin manifestation; next furunculosis, carbuncle, and gangrene. Purpura and xanthoma are rare. Arteriosclerosis is apparently responsible for certain complications, especially gangrene of the extremities. Perforating ulcer may occur. Profuse sweating occasionally appears, with or without diminished urine. The endocardium and myocardium are frequently the seat of extensive changes leading to valvular disease and fatty degeneration. Sexual disorders, especially impotence, usually appear early.

Prognosis.—It is essential that the family and personal history should be thoroughly sifted to determine the presence of syphilis, gout, or other constitutional disease, the outlook being more hopeful for those in whom such diseases can be detected. Favourable signs are late onset, obesity, and long-continued disease with but slight loss of flesh and strength. If the sugar diminishes rapidly under treatment the outlook is hopeful. In children an early fatal termination is the rule. Coma is of the gravest import.

V. **Gout.**—Three forms: *acute*, *chronic*, and *irregular*.

Symptoms.—(1) *Acute Gout.*—The attack usually occurs between the hours of 12 to 2 A. M., often without warning, in a person whose family history shows the lithæmic diathesis, or who has acquired it by constant overindulgence in nitrogenous food, wines (especially champagne), or malt liquors; by sedentary habits; by saturnism; or by privation (poor man's gout). The attack is usually precipitated by unusual dietetic indulgence, exposure, or injury.

The onset is sudden, with excruciating, vicelike pain in the proximal great-toe joint, chill, fever (rarely over 102°), marked restlessness, and insomnia. By morning the pain lessens, the joint is swollen, and the skin red, tense, and shining. During the day the pain may disappear, returning at night. An attack may last from 5 to 8 days, or longer, with gradual amelioration of all symptoms. During the attack the urine is scanty, high-coloured, and acid, although the total uric-acid excretion is diminished. As the symptoms subside the uric acid increases. The phosphoric-acid excretion varies directly with that of uric acid.

While the ankle, midtarsal, and knee joints, and outer side of the foot are frequently involved, in the order named, the metatarsophalangeal joint of the great toe is by far the most often affected. In some cases polyarthritis, usually unilateral, may be present. The affected joint may be left somewhat stiffened. The severity of the constitutional disturbance varies with the intensity of the arthritis.

Premonitory symptoms, especially in patients who have suffered repeated attacks, include constipation, palpitation, irritability of temper, dyspepsia, bronchitis, and the urinary changes already noted. In a certain number of cases the joint symptoms are slight, or, beginning severely, will rapidly subside, with corresponding alarming internal symptoms, so-called "retrocedent" or "suppressed" gout. The symptoms may be gastro-intestinal, pulmonary, cardiac, or cerebral. If the first, there is nausea and vomiting, much severe pain, usually diarrhœa and great, even fatal, prostration. The pulmonary variety appears as asthma. If the heart is affected there may be dyspnœa, pain, and arrhythmia, even syncope. Rapidly developing fatal pericarditis has been reported. Headache and delirium, probably uræmic, are the most common cerebral symptoms.

(2) *Chronic Gout.*—Acute attacks, previously described, recur more or less regularly, the patient in the interim being in poor health. The gradual deposit of sodium urate in the articular surface of the joints, and later in the surrounding tissues, with the continued inflammation, produces first disability, later deformity. This condition spreads from the feet to the hands, the knees and elbows being involved in severe cases, the deposits even extending up and down the tendon sheaths and into the neighboring bursæ. Lastly, deposits occur in the cartilages of the ear and throughout the skin. These deposits ("tophi," "chalk stones") in long-standing cases may be exposed by ulceration, especially about the finger joints.

With these local symptoms gastric and bronchial irritation are associated, and arteriosclerosis and heightened tension appear, the vascular disturbances inducing cardiac hypertrophy and chronic interstitial nephritis. Intercurrent attacks of acute polyarthritis may occur. In the later stages the gouty symptoms are associated with those of eczema, endarteritis, diabetes, bronchitis, endocarditis, or nephritis, the last usually being the direct cause of death.

(3) *Irregular Gout.*—Lithæmia (gouty diathesis) comprises an ill-defined group of symptoms occurring in members of gouty families,

or in the gastronomically overindulgent. The symptoms are varied. The manifestations in the gastro-intestinal tract are catarrhal gastritis, or simply functional disturbance with constipation, foul breath, coated tongue, deficient biliary secretion, a "bilious attack." In the vascular system there is arteriosclerosis, with increased tension, leading to cardiac and renal changes, either of which may predominate; or the sclerotic process in the aorta or the cerebral vessels may favour the growth of aneurisms, and death from apoplexy may result; or myocarditis or pericarditis may determine a fatal issue. As mentioned, chronic interstitial nephritis supervenes. In the lungs bronchitis is usual—sometimes emphysema or pleurisy. The skin lesions are pronounced, eczema in particular; next acne, psoriasis, and urticaria. In the eye, iritis is most common, though retinitis is not infrequent. Glaucoma and suppurative panophthalmitis have been described. Of nervous symptoms headache is common; next neuralgias and sciatica; lastly peripheral disturbances, itching eyeballs, burning and itching feet, and cramps in the legs. Basilar meningitis may develop. Sugar and oxalates are found in gouty urine, so also small quantities of albumin. Lithic acid crystals may deposit on standing. Spontaneous urethritis has been reported.

Differential Diagnosis.—Acute polyarticular attacks may be distinguished from rheumatic fever by the history, frequently obtainable, of previous typical monarticular attacks. Rheumatism selects the larger joints, is less painful, causes less superficial inflammation, and never shows the marked venous engorgement peculiar to gout. The temperature runs higher, there may be delirium, even convulsions, which seldom accompany gout. The latter is often afebrile even with several joints involved. Arthritis deformans, however, is often differentiated with great difficulty from chronic gout. In these cases careful inquiry may demonstrate a gouty family history, which, coupled with the patient's previous manner of life and the finding of tophi, may serve to separate the conditions. So, too, the gouty origin of the various disorders referable to the special organs may be distinguished from malarial or syphilitic taints. It must be borne in mind that either of the latter may be present in a gouty person.

Prognosis.—In vigorous patients, with single attacks, the outlook is favourable, providing the prescribed regimen be strictly followed. The sudden subsidence of articular and the appearance of internal symptoms is of serious import. The pronounced tendency to endarteritis, endocarditis, and nephritis should always be considered.

VI. **Arthritis Deformans.**—**Varieties and Symptoms.**—Charcot recognises the following forms: *general progressive* form, *monarticular* form, and *Heberden's nodosities*. Of the first named *acute* and *chronic* varieties exist, the latter being much the more frequent and differing in intensity and rapidity of onset.

(1) *Acute Form.*—The onset of the acute form closely simulates acute articular rheumatism. Many joints are involved at once. There are pain and swelling of the joints, the former out of all proportion to the latter; but the temperature does not run high and hyperæmia is seldom marked. Such cases are most often noted in young women, especially after childbed. A subacute form appears in children, girls particularly, subsequent to exposure, privation, and injury, occasionally with a rheumatic family history (GARROD). They have reasonable hope of recovery.

(2) *Chronic Form.*—In this there is a symmetrical progressive involvement of the peripheral joints. Swelling in or about the joint, with moderate pain, signalizes the invasion of the disease. In extreme cases it tends to involve all the articulations. There are frequent remissions, and the pain is variable. Many severe cases suffer little or no pain. The ultimate result is locking of the joints, due to the bony outgrowths (osteophytes), with deformity due to muscular atrophy and contracture. Garrod enumerates the following symptoms: symmetrical distribution (not invariable); enlargement, due to osteophytes, or gradual infiltration of entire joints, or serous effusion, alone or combined; muscular atrophy, frequently with exaggerated reflexes, leading to deformities; pigmentation and glossiness of the skin about the joints; subcutaneous nodules; pain, exceedingly variable in degree, numbness, and tingling; undue rapidity of the pulse, and frequent palpitation. The deformity in the hand and wrists is striking. Osteophytes, infiltration, and effusion cover up the natural tapering at the wrist, the carpus is pushed toward the radial and the fingers toward the ulnar side; they are flexed on the hand, and all the small joints are enlarged and deformed (Fig. 74, page 293).

(3) *Monarticular Form.*—Identical with the other types of the disease in its anatomical and histological features, it is peculiar in that it rarely attacks any but the aged, and selects the hip, knee, shoulder, or vertebral articulations. A history of traumatism is more frequently obtainable, and men seem more often affected. The conditions known as "morbus coxæ senilis" and "spondylitis" are the most frequent varieties of the monarticular form.

(4) *Heberden's Nodosities.*—Women are the chief sufferers. The condition is limited to the fingers, and appears in middle life. A history of heredity, gout, and rheumatism may be elicited. The characteristic deformity is a symmetrical bony outgrowth on the dorsal aspect of the distal phalanges (Fig. 75, page 294). With these

are often associated little translucent cysts, possibly pouches of the synovial membrane. Pain may accompany the growth, and there may be redness and swelling. Tenderness is often, but by no means always, present. The thumb frequently escapes.

(5) In *children*, in addition to the general progressive forms, Still has described a set of cases in which the general enlargement of the joints is associated with fever and swelling of the spleen and lymph glands. The disease usually begins before the second dentition.

Diagnosis.—The acute form is hardly distinguishable from acute articular rheumatism. Pain out of proportion to the signs of local inflammation and the presence of deforming changes should exclude the latter. Arthropathies, while anatomically the same, are associated with the other symptoms of locomotor ataxia. Gonorrhœal and scarlatinal arthritis, cerebro-spinal meningitis, pyæmia, etc., can scarcely be confused with arthritis deformans because of their acute character and previous history. From chronic gout the distinction has been made (page 1137). Osler describes a localized arthritis of the shoulder joint which closely simulates arthritis deformans by the pain, thickening of the ligaments, muscular atrophy, and occasional neuritis. The bones are unaffected. (See also pages 101 to 104.)

VII. **Rickets.**—*Symptoms.*—Beginning between the sixth and twenty-fourth months, the disease usually manifests itself by irritability, restlessness, and slight fever, most marked toward night. The child resents handling, wakes frequently, cries out, and is subject to drenching sweats, especially about the face and head. In a previously quiet sleeper this restlessness and intolerance of the bedclothing is always suggestive. Increasing hypersensitiveness of the entire surface, with anæmia and disturbed digestion, as evidenced by diarrhœa, with flatus and foul passages, follow, and enlargement of the liver and spleen is recognisable. Dentition is delayed, the fontanels do not close, and skeletal changes become manifest.

The course of the disease up to this point is estimated at from eight to ten months. There is progressive pallor, and the flesh has become soft, flabby, and doughy. Various nervous phenomena are present. The loss of muscular tone may simulate paralysis.

Of skeletal changes, possibly the first noticed are those in the thorax (see page 318). The abdominal walls are relaxed and protrude unduly. These changes may appear as early as the third month of the disease. The skull is subject to marked deformities. Nutritional changes allow softening and absorption of the bony tables, which become in spots thin as parchment, are compressible, crackle under the touch, and permit the internal pressure to materially alter the shape of the cranium. This condition, "craniotabes," is most frequent in the parieto-occipital region. Coincidently flat, bony plates form over the frontal and parietal prominences, producing the deformity known as "caput quadratum." A distinct furrow may mark the sagittal and coronal sutures. This cranial enlargement, with flattening of the malar prominences, makes the face seem small and out of proportion (Fig. 36, page 171). A systolic murmur over the anterior fontanel is by no means peculiar to rickets (OSLER).

All the long bones show changes; in the shaft from muscular tension, in the ends from epiphyseal proliferation. Knock-knee, bowleg, and, in bad cases, talipes result, while even the clavicles and scapulæ may be deformed. Pelvic deformities in girls are important from their influence on parturition. Of nervous symptoms there may be, aside from those already noted, laryngismus stridulus, tetany, and convulsions, and occasionally hydrocephalus and ocular symptoms. Particularly susceptible children may develop maniacal symptoms. Taylor and Wells describe a form of "pressure palsy" due to inflammatory change in the vertebræ.

Prognosis.—The disease is not in itself fatal, but its resulting deformities are to be remembered and guarded against.

VIII. **Obesity.**—Two forms are recognised: the *plethoric* and the *anæmic*, which later merge into the *hydræmic* (OERTEL).

The *plethoric form* appears in adolescents and young adults, beginning as a rounding out of the entire frame, at first symmetrical, later becoming extreme, and producing the characteristic appearance of obesity. The mucous membranes and skin become congested, there is gradual loss of muscular tone and increasing laziness, all this in hearty subjects, large eaters, and drinkers. The pulse, at first strong, gradually slows and weakens, the area of cardiac dulness increases, and the apex beat becomes diffuse. The belly grows pendulous, the abdominal and thoracic organs enlarge, their pressure induces dyspnœa at night, and the overerect posture in walking causes backache. The condition merges into the hydræmic form.

The *anæmic type* begins in patients who are already anæmic or chlorotic. Fat is present in doughy, flabby, shapeless masses, the muscular tone is gone, the skin cool, the body temperature subnormal, the heart weak, the patients complain of dyspnœa and palpitation, and sleep much. They are seldom great eaters, but take large quantities of sweets and water. A majority of these patients are women, commonly with menstrual irregularities. The condition may appear *post partum*, or after prolonged lactation. In men it frequently follows typhoid, secondary syphilis, and chronic alcoholism. This type is, at times, seen in anæmic or rhachitic children. The urine is scanty and irregular, and œdema is common.

The *hydræmic form* is merely an aggravated condition, in which the anæmia is profound and all the symptoms intensified. As the disease progresses there is loss of muscular power, lowered tone, and inability to withstand disease, while various organic changes develop, especially myocarditis and valvular changes; small, weak, dicrotic, and irregular pulse; and arteriosclerosis. A tendency to nephritis, lithæmia, and diabetes is often noted. The *course* of the disease is slow, covering years, and the *prognosis*, unless suitable treatment be faithfully followed out, is far from good.

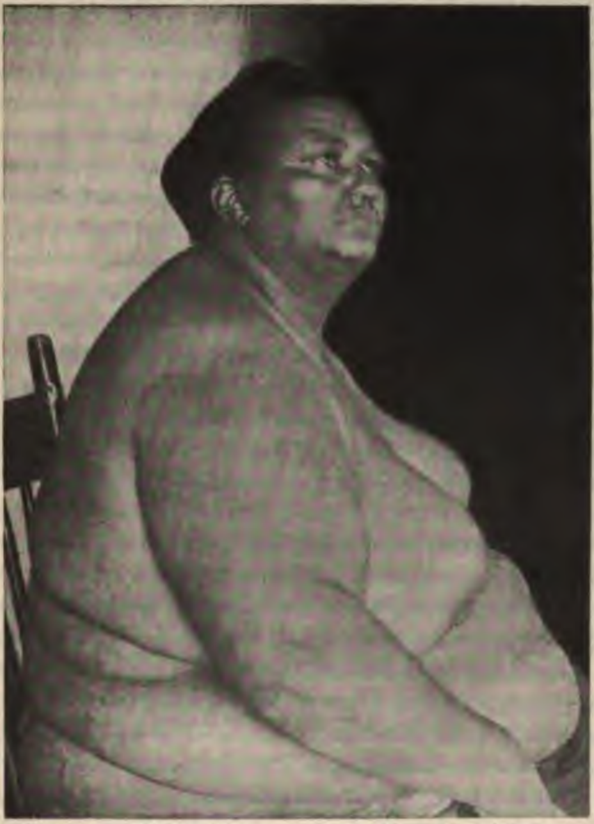

FIG. 272.—Adiposis dolorosa in a colored woman. (Pearce.)

IX. **Adiposis Dolorosa** (*Dercum's Disease*).—The diagnosis depends upon the finding of "irregular symmetrical deposits of fatty masses in various portions of the body, preceded by or attended with pain." The disease occurs in women at the middle time of life. Associated with neuralgic pains, or paræsthesias, bunches of fat develop, which may become huge, pulpy, and pendulous. These do not occur on the feet, hands, or face. In some cases the thyroid is atrophied, but the nature of the disease is unknown.

SECTION XI

THE INTOXICATIONS: SUNSTROKE

PREPARED BY FRANK WHITFIELD SHAW, M. D.

I. **Alcoholism.**—VARIETIES AND SYMPTOMS.—A periodical excessive consumption of alcoholic liquor is known as *dipsomania*, hereditary or acquired. During the intervals there is an absence of desire. It is a form of acute alcoholism, but when long continued the resulting structural changes are those of chronic alcoholism.

(I) **Acute Alcoholism.**—*Symptoms.*—These are disturbances of the mental functions, muscular inco-ordination, and finally unconsciousness, with deep though rarely stertorous breathing. The face may be flushed or pale, and the pulse is usually slow and full. The pupils are equally dilated. The temperature is rarely elevated, not infrequently subnormal. In uncomplicated cases convulsions are rare, although slight muscular twitchings may be present. The odour of the breath is characteristic. When the condition is well-marked there is a nearly complete loss of voluntary muscular power. By persistent effort the patient may be aroused to incoherent speech, followed almost immediately by a return to the narcotized condition.

(II) **Chronic Alcoholism.**—*Symptoms.*—There is unsteadiness of the muscles, particularly those of the legs, hands, and tongue, manifested by tremor, which for a time can be temporarily overcome by taking alcohol. There gradually develop mental sluggishness, impaired judgment and memory, with irritability of temper and a progressive change in moral character. Indigestion and catarrhal gastritis are early and constant findings. The breath has a peculiar odour, the bowels are constipated, the tongue is furred, and there is anorexia. Gastrectasia is not uncommon in habitual beer drinkers. The most constant change occurring in the liver is cirrhosis, preceded, sometimes for years, by enlargement and tenderness. The eyes are watery and red, and *acne rosacea* is common.

(III) **Delirium Tremens** (*Mania a potu*).—A result of long-continued alcoholic poisoning of the brain centres; is a common attendant of pneumonia in alcoholics, and may be induced by sudden fright, shock, or accidents.

Symptoms.—Insomnia, restlessness, and depression are early premonitions. The delirium manifests itself by constant incoherent talking, unnatural activity of mind and body, and hallucinations of sight and hearing. There is a disordered imagination, the patient fancying that he sees and hears animals (mice or snakes), and that he is being pursued by enemies. The terror induced by this condition may be so marked that the patient in his efforts to escape may do himself bodily harm. Insomnia continues during the active period of the delirium, which may last for several days, a return to natural sleep heralding improvement. The temperature usually ranges from 101° to 103°, although it may be much higher in fatal cases. The pulse is rapid and of fairly good quality at first, becoming very weak if the delirium is prolonged. Muscular tremor and a tremulous tongue (usually heavily coated) are constant. Fatal cases end by heart failure.

LATER SYMPTOMS ATTENDING ALCOHOLISM.—Insanities, dementia paralytica, and epilepsy have been attributed to alcoholism. Hereditary epilepsy is claimed as a result of long-continued dipsomania in one or both parents, particularly in the father. Chronic encephalomeningitis may occur, and acute alcoholic neuritis is a painful condition attending advanced alcoholic poisoning. There is a chronic form of alcoholic neuritis known as *polyneuritis potatorum* (*pseudotabes*, *ataxia of drunkards*). It presents two varieties, the paralytic and the ataxic. There are pains in the lower extremities, ataxia, areas of anæsthesia, occasional loss of the superficial reflexes, and paralysis and atrophy, chiefly of the extensors of the fingers and toes. Arteriosclerosis, with sequent cardiac dilatation and granular kidney, is not infrequent.

DIAGNOSIS.—(1) **Acute Alcoholism.**—Usually easy, provided the unconsciousness is not complete. It is based mainly upon a history of alcoholic excess, the odour of the breath, and acute gastric disturbances (frequent and prolonged vomiting), with muscular incoordination and confused mentality. There is a pronounced inclination to sleep, an indifference as to location, and a decided, although good-natured, resentment of interference. A sound sleep assures the disappearance of most of these symptoms and a gradual return to the normal. Only in cases associated with unconsciousness and other evidences of cerebral or constitutional disturbances is there any difficulty in the differential diagnosis. It must be borne in mind that even moderate indulgence in alcohol may be coincident with apoplexy, fracture of the skull, acute uræmia, or sunstroke. In cases of reasonable doubt the patient should receive the benefit and be treated as if suffering from the graver condition.

For the separation of acute alcoholism from other forms of *coma*, especially *apoplexy*, *uræmia*, and *sunstroke*, see pages 76 to 79.

Fracture of the Skull.—In this the coma may be profound, presenting many of the symptoms of cerebral hemorrhage, with few if any evidences of external injury. If there is no improvement, but rather a gradual increase in the symptoms after a reasonable length of time, a suspicion of fracture is justifiable. A careful examination of the entire scalp will usually reveal a point of circumscribed œdema indicative of injury to the bone. Otherwise the differential diagnosis is similar to that from hemorrhage (page 77).

(2) **Diagnosis of Delirium Tremens.**—Occurs only in more or less chronic alcoholism, not in persons of ordinarily temperate habits. In addition to the history there are hallucinations of sight and hearing, incoherency of speech, great irritability and restlessness, some fever, insomnia, prostration, and a frequent, feeble pulse. The lungs should be examined for pneumonia and the body for traumatisms.

II. **Morphine Habit** (*Morphinomania, Morphinism*).—If the use of morphine is discontinued after the indications for its employment have passed no injurious effects will follow; otherwise its continued taking develops a craving for the drug, with the ultimate formation of the morphine habit.

Early Symptoms.—At first slight, and the general health is little, if at all, impaired. The drug produces feelings of satisfaction and personal comfort, a condition which lasts as long as it is given in sufficient quantities. When discontinued, feelings of lassitude and mental depression, nausea, and sometimes vomiting, accompanied by epigastric distress, ensue. More or less general itching of the skin, particularly about the nose, is a quite constant symptom.

Later Symptoms.—As the necessity for larger doses becomes more urgent disturbances of the general health are manifest. The victim is restless and irritable, sleep is disturbed, appetite and digestion deranged, and there is constant mental depression. Chills followed by profuse sweating are not uncommon. The pupils are dilated, sometimes unequally, except while under the direct influence of the drug, when they are minutely contracted. There is a gradual weakening of the moral sense, and the patient becomes an inveterate liar. In women, hysteria and neurasthenia are not uncommon. Ultimately a condition of asthenia, with anorexia, is induced. The victim gradually becomes emaciated, sallow, gray, and prematurely aged, finally dying from extreme weakness.

III. **Acute Opium Poisoning** (*Opium Narcosis*).—Due to an overdose, and may occur in *habitués* as well as in non-users.

Symptoms.—The period between the taking of a poisonous dose by the stomach and the first indication of symptoms varies from a very few to 20 or 30 minutes. When taken with suicidal intent by subjects of alcoholic mania, there is often a marked resistance to its narcotic effects, followed by sudden and complete coma. The onset is usually abrupt. The patient may be talking naturally one moment and the next be profoundly unconscious. The jaws may at first be fixed and resist efforts to open them. Later relaxation occurs. The pupils become minutely contracted, do not react to light, and sensation is lost in the cornea. Coincidently the respirations drop to 10 or 12, sometimes 4, to the minute. The heart action is weak, and the pulse feeble, well-nigh imperceptible.

The face is pale, sometimes cyanotic, and the skin dry or bathed in perspiration. The coma may resist everything but severe bodily punishment. When partially aroused the speech is incoherent, and the patient relapses quickly into narcosis. There is retention of urine, later associated with vesical tenesmus, lasting for several hours. The tongue may drop back into the pharynx, seriously diminishing the already impaired oxygenation. The respiration may be stertorous and the cheeks flap. Under successful treatment the coma gradually lessens, the respirations become more frequent, the colour of the skin and the heart action improve. Relapses are frequent, and hours or days may elapse before the patient is out of danger. Opium narcosis following alcoholic excess may be complicated by acute mania or peripheral neuritis of one or more of the extremities.

Diagnosis.—It may be necessary to differentiate between the coma of acute opium poisoning and that of uræmia, alcohol, sunstroke, and cerebral hemorrhage, for which see pages 76 to 79.

IV. **Lead-poisoning** (*Plumbism; Saturnism*).—This metal may enter through the lungs, skin, or digestive system. It is found in the air in regions where lead ore is being smelted, in white-lead factories, and is contained in water, wines, and cider which have been confined for some time in lead pipes or lead-lined containers. It may enter in milk, cosmetics, hair dyes, thread, false teeth, and adulterated food or food products—e. g., baking powder containing chromate of lead. Plumbism is not common in children, occurring usually between 30 and 40 years of age, in women rather than men.

VARIETIES AND SYMPTOMS.—(I) **Acute Lead-poisoning.**—Symptoms may appear in a few weeks after exposure, or be absent for months or years. Anæmia, sometimes rapid, may follow a short exposure. Constipation, followed by severe diarrhœa, vomiting, and occasionally abdominal tenderness and distention, are present. Usually the abdomen is contracted, hard, and perhaps scaphoid. There is colic (*colica Pictonum*) of a most severe type felt over the entire abdomen. It intermits, with dull abdominal pain during the intervals. It is a true, non-inflammatory colic due to the action of the metal upon the terminal nerves. The paroxysms are of gradual onset, but steadily increase in severity and frequency. The pain is twisting or grinding, and may be accompanied by pain or cramps in the extremities. The point of maximum intensity of the pain is usually in the region of the umbilicus, whence it radiates in all directions, but may be in other parts of the abdomen. It is relieved by deep pressure, and sometimes excited by food. The temperature is not elevated, and may be subnormal.

Arteriosclerosis, contracted kidneys, hypertrophy of the heart, and distinct gouty deposits are not infrequent results of continuous exposure. Gouty subjects are especially liable to lead-poisoning. Albumin is frequently found in the urine, due either to the irritation produced by the elimination of the lead or to a coexisting nephritis. Other symptoms described are convulsions, epilepsy, and delirium. Abdominal distention and severe entero-colitis are somewhat rare, and usually occur in persons recently exposed.

Acute Lead-poisoning in Children.—If slight, this may easily be overlooked. A not uncommon source of infection in the very young is found in toys composed wholly or in part of lead. The symptoms often resemble those of a slight indigestion. There may be indefinite malaise, and either constipation or strong-smelling diarrhœal movements. If the child is old enough he may complain of a constant pain in the region of the umbilicus, not sufficiently severe to cause crying, but which does not yield to simple remedies. The child will go to the nurse many times during the day seeking comfort for a distress which is persistently referred to the abdomen. There is constant slight fever (99° to 101°), which at the beginning of the attack may reach 104°, but this high temperature soon responds to simple laxative treatment. It may be confused with slight malarial infection, but does not respond to quinine. With such symptoms, if careful inquiry is made as to the toys used by the child, a source of lead infection may be found.

(II) **Chronic Lead-poisoning.**—The symptoms are due to the prolonged absorption of small quantities of lead, and usually one or more of these predominate. The specific effects are *disturbances of nutrition, colic, arthralgia, paralyses,* and *lead encephalopathy.*

(1) Disturbances of Nutrition.—Anæmia may be profound, the red cells sinking to 50 per cent, with wasting, especially of the muscles, and a peculiar yellowish complexion. The latter (*icterus saturninus*) is not due to the deposit of bile pigments. Along the border of the gums may be found a dark or bluish-black line, often lacking in persons having clean teeth. In the absence of teeth it is not

present. It may be confounded with a line on, not in, the gums, which is readily removed by cleaning the teeth (OSLER); and a black line found in miners, due to deposition of carbon. For others see page 239. Certain general symptoms are: Habitual dryness of the mouth, with an astringent, sweetish, or faintly metallic taste, coated tongue, fetid breath, obstinate constipation, and distressing dyspepsia. As the disease progresses there is loss of muscular power and occasional tremors. The patient becomes apathetic, irritable, and morose. Coinciding with these symptoms there may have been several attacks of lead colic, followed by pains in the muscles or joints, perhaps by evidences of beginning paralysis.

(2) Lead Colic (*Colica saturnina*).—This affection (*painter's colic, Devonshire colic, colica Pictonum*) follows, as a rule, a period of malnutrition, dyspepsia, and constipation, but may develop suddenly, and be ushered in by nausea and vomiting. The paroxysms of pain are more severe in the afternoon and at night. Relapses are frequent, and continued exposure may lead to the colic becoming chronic The termination of an attack may be as sudden as its onset.

(3) Arthralgia (*Arthralgia saturnina*).—Paroxysmal pains, with exacerbations and remissions, are not infrequently present in the joints and contiguous muscles, without swelling, redness, or fever. The pain may be increased by exercise and cold. The knees are most commonly affected, next the elbow and shoulder joints; the flexor muscles more frequently than the extensors. The large lumbar muscles, the intercostal, and those of the neck are often involved. The pain is severe, tearing, and burning.

(4) Paralysis (*Paralysis saturnina; Lead Palsy*).—Usually a later manifestation, and, with few exceptions, limited to adults. One attack predisposes to others. As a rule there is no fever. The paralysis may be *local* or *general*. R. D. is marked, muscular atrophy begins early, and the wasting may be so decided that there is stretching of the ligaments, with ultimate partial or complete dislocation of the bones. Early symptoms are coldness, numbness, and hyperæsthesia, seldom anæsthesia; feebleness, stiffness, impairment of motor power, and tremor of the affected muscles. The symptoms are more marked at night. If persisting, they soon lead to the development of the characteristic paralysis. In the great majority of cases the extensor muscles are affected, one or more of the muscles of a group being the seat of the paralysis. The following localized forms have been recognised (Dejerine-Klumpke, summarized by Osler):

Anti-brachial.—In this the musculo-spiral nerve is involved, with paralysis of the extensors of the fingers and wrist (wrist-drop). The supinator longus usually escapes. Distention of the synovial sheaths, causing a prominent swelling over the wrist (Gruebler's tumour), occasionally follows the long-continued flexion of the carpus.

Brachial.—The muscles involved are the deltoid, biceps, brachialis anticus, and supinator longus. The atrophy is of the scapulohumeral form and is bilateral.

Aran-Duchenne.—There is marked atrophy of the small muscles of the hand and of the thenar and hypothenar eminences, resembling the early stage of poliomyelitis anterior chronica.

Peroneal.—In this the lateral peroneal muscles, the extensor communis of the toes, and the extensor proprius of the big toe are involved, with the production of the steppage gait ((3), page 37).

Respiratory.—Paralysis of the intercostal and laryngeal muscles and adductor paralysis of the larynx nave been noted.

Generalized Palsies.—There is a rare type of palsy pursuing a course like that of ascending paralysis, with rapid wasting of all four limbs. A febrile form of general paralysis has been recognised.

(5) Encephalopathy (*Encephalopathia Saturnina*).—Extremely rare, and is due to the action of lead upon the nerve centres. It embraces lead insanity, delirium, convulsions, epilepsy, and coma. A large number of symptoms may precede the more active cerebral manifestations, such as headache, vertigo, troublesome insomnia, distressing dreams, dimness of vision, alterations of the pupils, tinnitus aurium, dysphagia, constriction of the pharynx, and stupor or excitement. Colic, arthralgia, and palsy are also prodromal.

Lead encephalopathy may be divided into three forms, the *delirious*, *comatose*, and *convulsive*. The *delirium* is at first tranquil, later becoming furious and paroxysmal, with intervals of somnolence. Finally, true sleep supervenes, followed by complete restoration. The *coma* may be gradual or instantaneous; may be the only active indication of the disease; or be preceded or attended by convulsions or delirium. *Convulsions* are the most common; are occasionally followed by true epilepsy; and may be partial or general. If partial, the face, a single limb, or one side of the body alone may be involved. They may be associated with dulness, and followed by more or less complete coma. Amblyopia, usually followed by recovery, has been noted, but occasionally optic-nerve atrophy results.

DIAGNOSIS.—Excepting colic, which may occur with few if any prodromata, little difficulty is experienced in the diagnosis of lead-poisoning. The history and the subsequent course and symptoms are usually sufficiently distinctive. The abdominal pain, being purely a neurosis, may at first be mistaken for flatulent (page 882), hepatic (page 889), renal (page 1025), or appendicular (page 872) colic or ectopic gestation (page 871).

V. **Arsenic-poisoning.**—Arsenic in poisonous quantities may enter the body by way of the intestinal or respiratory tracts. It is the active principle of Paris green and Rough on Rats, is employed in the manufacture of coloured papers and artificial flowers, and is found in many fabrics. The action of certain moulds upon the arsenical organic matter in wall paper produces a volatile oil which renders poisoning through the lungs possible.

VARIETIES AND SYMPTOMS.—(I) **Therapeutic Overaction.**—Medicinal doses may cause symptoms of considerable severity, i. e., a metallic taste, salivation, nausea, vomiting of glairy mucus, epigastric pain and soreness, diarrhœa, tenesmus, and at times dysenteric stools. The heart is irritable and feeble, with palpitation, cough, oppressed breathing, œdema of the eyelids, and occasionally general œdema. There may be itching of the eyelids, urticaria, eczema, pityriasis, psoriasis, falling out of hair and nails, trembling, stiffness, contraction of joints, disorders of sensibility, and herpes zoster (BARTHOLOW).

(II) **Acute Arsenic-poisoning.**—The gastro-intestinal symptoms are intense epigastric and abdominal pain, uncontrollable vomiting, colic, diarrhœa, tenesmus, dryness of the mouth and fauces, intense thirst, intestinal irritation, bloody and offensive stools, retracted abdomen, strangury, priapism, suppression of urine or bloody urine, menorrhagia in women, and involuntary evacuations. Without gastro-intestinal symptoms the condition may become suddenly one of profound coma very similar to that of extreme opium narcosis. Later symptoms may be paralysis, neuralgic pains, and numbness.

(III) **Chronic Arsenic-poisoning.**—Pronounced and common symptoms are debility and anæmia, with some gastro-intestinal irritation and pain, disturbed mucous secretion, redness or bleeding of the gums, sensory disorders, such as tingling or numbness, and œdema or puffiness of the eyelids. Later symptoms are multiple neuritis, pigmentation, stiff joints, neuralgic pains, and arsenical paralysis. The latter resembles that of chronic lead-poisoning, except that the legs, rather than the arms or wrists, are usually affected. It attacks the extensors and the peroneal muscles, causing the "steppage gait" (page 37). R. D. is usually present. A tolerance is at times acquired, which will render harmless an ordinarily poisonous dose.

DIAGNOSIS.—Based mainly upon the history and the form in which the poison is taken. Attention should be given to wall paper, artificial flowers, and wearing apparel. Paris green, in common use by suicides, gives an intensely green colour to the vomitus.

VI. **Food-poisoning** (*Bromatotoxismus*, Vaughan).—Food, including milk, may contain the active organisms of tuberculosis, trichinosis, typhoid fever, scarlet fever, and diphtheria; may be infected with the bacteria of putrefaction or by certain fungi, and, finally, actively poisonous ptomaines may exist in shellfish and fish.

(I) **Meat-poisoning** (*Kreotoxismus*).—Arises commonly from decomposed sausages (*botulism*, *allantiasis*), ham, pork pie, and headcheese; occasionally beef, veal, and mutton.

Symptoms.—The primary symptoms (BALLARD, VAUGHAN, OSLER) in a mild type are vomiting, diarrhœa, abdominal pains, muscular weakness, thirst, and headache, preceded by a period of incubation varying from 12 to 48 hours, during which there are no prodromes. In other cases there are languor, anorexia, nausea, chilliness, dyspnœa, vertigo, faintness, cold sweat, headache, pain in the trunk or abdomen, dysphagia, and intense thirst. A single case seldom, if ever, presents all of these symptoms. At the close of the incubation period more active symptoms develop, beginning with abdominal pain, constant diarrhœa, and vomiting. The pain (crampy, tearing, burning) in the chest or between the shoulders causes extreme prostration and faintness. The diarrhœa is frequently uncontrollable, with dark and very offensive stools. Headache, intense thirst, and restlessness are frequent. There is fever, and the pulse reaches 100 to 128.

Less frequent symptoms are "excessive sweating, cramps in the legs, or in both legs and arms, convulsive flexion of the hands or fingers, muscular twitching of the face, shoulders, or hands, aching pains in the shoulders, joints, or extremities, a sense of stiffness of the joints, pricking or tingling or numbness of the hands lasting far into convalescence, a sense of general compression of the skin, drowsiness, hallucinations, imperfection of vision, and intolerance of light. In other cases yellowness of the skin, either general or confined to the face and eyes, appeared. In the fatal cases death was preceded by collapse like that of cholera, pinched features, and blueness of the fingers and toes and around the sunken eyes" (OSLER).

Symptoms similar to those just described have been observed in cases of poisoning by canned meats. In other cases poisonous symptoms may arise from eating certain game birds.

(II) **Poisoning by Milk Products.**—The symptoms of poisoning by milk (*galactotoxismus*), cheese (*tyrotoxismus*), custard, and ice cream do not differ from those occurring in meat-poisoning.

(III) **Poisoning by Shellfish and Fish.**—(1) Oysters.—These, if they have undergone decomposition, may become poisonous. The symptoms are usually gastro-intestinal, and differ only in degree from those of meat-poisoning.

(2) Mussels (*Mytilus edulis*).—The edible mussel becomes poisonous in filthy water. The symptoms, unlike those of meat-poisoning, are seldom gastro-intestinal. The onset is acute, and death may

occur within a few hours with symptoms of collapse (see page 167). Even in cases that recover these symptoms are usually present.

(3) Fish (*Ichthyotoxismus*).—Putrefaction occurring in fish, especially haddock, mackerel, and cod, results in the production of toxic ptomaines. The *symptoms* observed are usually referable to the nervous system, the gastro-intestinal tract being seldom involved.

(IV) **Grain-poisoning** (*Sitotoxismus*).—(I) ERGOTISM.—The ergot fungus (*claviceps purpurea*), found in certain grains, may cause severe symptoms if food containing it has been used for a long time. Two varieties are recognised, *gangrenous* and *convulsive*.

Gangrenous.—Symptoms are spasmodic muscular contractions, pain, tingling, occasionally anæsthesia; finally blood stasis and gangrene, usually in fingers and toes, sometimes in nose and ears.

Convulsive.—During the early stages the symptoms may be those of the gangrenous form, followed by pronounced nervous disturbances. The prodromal period (1 to 2 weeks) usually presents headache, slight fever, and occasional tingling or pain, soon succeeded by muscular cramps and spasm, the latter continuing either for a few hours or for several days. In severe cases there may be early delirium or epilepsy (sometimes fatal); but dementia or melancholia are somewhat more frequent occurrences. Death is not uncommon in chronic ergotism. Degeneration of the posterior columns may ensue, resulting in a condition not unlike that of *tabes dorsalis*.

(II) LATHYRISM (*Lupinosis*).—A condition due to the presence in the food of the seeds of the *Lathyrus*, and occurs in India, Italy, and Algiers. Its most constant symptom is spastic paraplegia, resulting from what is probably a slow, toxic, spinal sclerosis.

(III) PELLAGRA (*Maidismus; Italian leprosy; Alpine scurvy*).—Due to the use, as food, of diseased maize. It is unknown in America, but common in France, Spain, and Italy. The body becomes almost coal-black, and the victims suffer from prostration and melancholia. The skin is thickened and rough, and finally exfoliates. Suppuration, with the formation of black crusts, is not uncommon. There are also diarrhœa, indigestion, and salivation. Mild cases may persist for months, and the more severe are attended by spasms, pain in the back and head, paralysis (chiefly a paraplegia), and melancholia or suicidal mania.

VII. **Sunstroke** (*Heat Exhaustion; Thermic Fever; Insolation; Coup de Soleil*).—Contributing causes are excessive bodily fatigue, insufficient diet, and overuse of beer and whisky. Two forms are recognised: (1) *Heat exhaustion* and (2) *Sunstroke*.

SYMPTOMS.—(I) **Heat Exhaustion.**—The attack may occur in bed, or at work, or while walking. Nausea and vomiting, usually preceded by dizziness and fulness or pain in the head, are early symptoms. There is a feeling of oppression and great weakness, the patient falling or sitting down. The skin is either flushed or pale, and may be hot and dry or bathed in cold perspiration. The pulse is usually rapid and feeble, the respirations shallow and sighing. The temperature ranges from subnormal to 102°. In many cases the condition is one of collapse (page 167). The unconsciousness is usually not profound and yields readily to treatment. In mild cases the pupils react to light and are moderately dilated.

(II) **Sunstroke.**—*Symptoms.*—The patient is usually found unconscious. The symptoms of heat exhaustion may have preceded the attack, but, as a rule, the insensibility develops very rapidly. The face is hot and flushed, the pulse rapid and bounding, and the respirations either loud, slow, and stertorous, or feeble, gasping, and laboured. In extreme cases the symptoms of collapse (page 167) may be present. Even in the cases which recover there frequently occur, during the period of coma and high temperature, tonic or clonic contractions of the muscles, either localized or general. With a drop in the temperature the muscular spasm becomes less frequent and the insensibility less profound. Relapses of high temperature, often with a return of coma and collapse, are not uncommon even after restoration to complete consciousness.

Complications.—These are: repeated relapses, persistent vomiting, retention of urine, general convulsions, cyanosis, active delirium, failure to respond to stimulation, pneumonia, pulmonary œdema, and fatal intestinal hemorrhage.

Sequelæ.—Sequelæ do not always occur, as the recovery is often complete. The most constant sequel is an intolerance of high, or moderately high, temperatures. Less frequent are peripheral neuritis, meningitis, muscular atrophy or tremor, wrist- or foot-drop, difficulty of speech, long-continued acceleration of pulse and respiration, headache, vertigo, epilepsy, spinal irritation, cutaneous anæsthesia or hyperæsthesia, enfeebled memory, deafness, cardiac lesions, indigestion, impaired nutrition, and anæmia.

DIAGNOSIS.—This is based upon the existing temperature and atmospheric conditions and the mode of onset. Heat exhaustion with moderately low body temperature may be mistaken at first for acute alcoholism, uræmia, or apoplexy. The history and the course of the attack are sufficiently distinctive for the discrimination. Sunstroke, with its sudden onset, extremely high temperature, and accompanying symptoms, should rarely be misdiagnosed.

SECTION XII

DISEASES DUE TO THE ANIMAL PARASITES

PREPARED BY FRANK WHITFIELD SHAW, M. D.

I. **Distomiasis.**—(1) *Liver Flukes.*—Some of these are the *Fasciola hepatica* (in ruminants), *Distomum lanceolatum* (sheep and cattle), and *Distoma felineum* (in cats). Among the *symptoms* assigned are emaciation, diarrhœa, ascites, and jaundice. The liver becomes enormously enlarged, and chronic cholangitis may coexist.

(2) *Blood Flukes* (*Schistosoma hæmatobium*).—Found in the blood of the portal vein and the veins of the spleen, bladder, kidneys, and mesentery. Hæmaturia, with dysuria, resulting from irritation due to the ova of this parasite in the blood, is a serious and constant symptom, second only to the progressive anæmia attending it.

(3) *Bronchial Fluke Distomum Westermanni, Parasitic Hæmoptysis*).—Found in the bronchial tubes, and produces attacks of hæmoptysis, which are endemic in many Eastern countries.

II. **Nematodes.**—(1) **Ascaris Lumbricoides** (*Roundworm*).—This worm (see (*i*), page 147) is most frequently found between the 3d and 10th years of childhood, but is rare during infancy. The *symptoms* may be very indefinite, the condition not being suspected until a worm is vomited or is found in the stools. There may be a wide range of symptoms: chills, hysterical attacks, epileptiform convulsions, strabismus, and perhaps temporary paralyses. More commonly the usual indications are grinding of the teeth, picking of the nose, irritability, and, in extremely nervous children, mild convulsive attacks. The *diagnosis* is simple when the worm or its ova (B, Fig. 261, page 679) are found. If not, the administration of full doses of santonin, followed by a purgative, will establish the diagnosis.

(II) **Oxyuris Vermicularis** (*Pinworm*). — The worms (see page 149) are found principally in the rectum and colon. The more pronounced *symptoms* are irritability and restlessness, intense irritation and itching about the anus and external genitals, incontinence of urine, and, in the female, vaginitis. The latter is probably due to the *Bacillus coli commune* with which the oxyuris is freely covered, rather than to the worm itself. The *diagnosis* presents no difficulties when the stools are properly examined, the worms with their ova (A, Fig. 261, page 679) being easily detected.

(III) **Trichiniasis.**—*Symptoms.*—These depend entirely upon the number of trichinæ which reach maturity in the intestinal canal.

If only a small number enter the stomach, and but few of these reach the muscles, there may be no symptoms.

The more marked early symptoms are abdominal pain, cardialgia, nausea, vomiting, and marked muscular exhaustion. The period of incubation varies from 3 to 14 days. An exhausting diarrhœa may appear early, and, if not fatal, may be followed by obstinate constipation. The muscles become swollen, tense, and painful to the touch. The muscles of mastication and respiration may be involved, with serious impairment of their functions. Fever may be slight or reach 104°. Profuse sweating is an early and persistent symptom. The urine is usually decreased. Early œdema of the eyelids and face, appearing later in the lower limbs, is said to be almost pathognomonic. In grave cases the symptoms may resemble those of typhoid fever. The group of symptoms which render the *diagnosis* fairly clear are œdema of eyelids and face, great prostration, violent muscular pain from motion or pressure, catarrhal symptoms of the bronchi, marked dyspnœa, continued sweating, and extreme restlessness. The most significant change in the blood is a leucocytosis with a marked eosinophilia. (See also p. 744). The *complications* are: long-continued diarrhœa, extreme dyspnœa, difficult deglutition, marked typhoid symptoms, bronchitis, pleurisy, pneumonia, sleeplessness, and general exhaustion.

(IV) **Anchylostomiasis** (*Uncinariasis, Egyptian Chlorosis; Brick-makers', Miners', Dirt-eaters' Anæmia*).—*Cause.*—*Uncinaria duodenalis* (Old World), *Uncinaria Americana* (America). Widely distributed. Occurs chiefly in sandy soil and deep mines. Mode of transmission through the skin, the parasite producing a local dermatitis, the so-called "Ground Itch," at the point of entrance. To reach its final abode in the small intestine the larvæ take the following route: through the hair follicles of the skin into the lymph vessels, the thoracic duct, the veins, the right heart, the lung capillaries, the air cells, the bronchi and trachea, the pharynx, and from there along the alimentary tract to the small intestine. In the Southern States and West Indies it is, perhaps, the most common disease of the white population.

Symptoms.—Of these the most important is anæmia, becoming extreme; the skin varying in colour from waxy or tallow to tan. It is accompanied by progressive emaciation, causing rapid ageing in adults, while the growth of children is stunted to a marked degree. Cardiac symptoms are prominent, cervical pulsation extreme, pulse 80 to 130, temperature generally normal, at times subnormal; at others a little raised. Difficulty in breathing may be prominent. Œdema of face and ankles is common, enlargement of abdomen is generally marked. The tongue may show peculiar blue patches; the appetite is variable, often taking abnormal directions, eating of plaster, soil, etc. Miscarriage is frequent. A peculiar cadaveric or fishy stare is noticed in many cases. The examination of the blood reveals an anæmia varying from a slight diminution of hæmoglobin and cells to the findings of a pernicious anæmia, though the hæmoglobin is usually relatively lower than in the latter disease. The red cells may be as low as 500,000. They are altered in size, polychromasia is present, nucleated cells, and even megaloblasts are found. There is frequently absolute leucocytosis, though this is believed to be due to complications. Eosinophilia is an important finding in this disease, varying from a slight increase to 30 or 40 per cent. The parasite extracts the blood by suction, and also forms a hæmolytic toxin.

The symptoms differ in severity with the general health of the infected person, and with the number of parasites in the intestine. If present in scanty numbers the evidences of their action are few; but if in multitudes, their destructive action on the blood is excessive.

Diagnosis.—The only absolutely diagnostic point is the finding of worms or eggs in the stools (Fig. 262). In a majority of the cases a diagnosis may be made by placing a small portion of the fæces, which are of a reddish colour, on blotting-paper, whereupon a distinct blood-red stain is left. Ordinary white blotting-paper is employed, and the stool should lie upon it for one hour.

Stiles suggests that if a microscopic examination is impracticable, the worms may be found by giving a moderate dose of thymol, followed by a saline. The resulting stools are to be washed a number of times in a deep vessel, decanting the washwater. The remaining sediment should be inspected for the worms. To the naked eye they appear about half an inch long, and about the diameter of a large pin, with one end sharply recurved.

Prognosis.—The mortality is high, but chiefly due to intercurrent diseases. The American disease seems not to be of such a severe type as that due to *Duodenalis.*

(V) **Filariasis** (*Filaria bancrofti*, *Filaria diurna*, and *Filaria perstans*).—There are three conditions which may be classed as symptoms, due to this parasite, the first two of which are due to the *Filaria bancrofti* or *nocturna; hæmatochyluria*, the occasional passage in the urine of blood clots, the urine being of an opaque white or milky appearance, and showing a slightly reddish deposit on settling; *lymph scrotum*, showing enormous thickening of tissues and distended lymph vessels; and *elephantiasis*. See also page 635.

(VI) **Tricocephalus Dispar** (*Whipworm*).—The symptoms are few and rare. By some it is thought to be the cause of beri-beri. Profound anæmia with diarrhœa have been associated with this worm. The diagnosis depends upon the presence in the stools of the eggs (See C, Fig. 261, page 679).

(VII) **Dioctophyme Gigas.**—The worm, male, about 1 foot long, female nearly 3 feet, is in man usually found in the region of the kidney, and has been known to entirely destroy that organ.

(VIII) **Strongyloides Intestinalis.**—This worm is found abundantly in the diarrhœa of hot countries, and is sometimes associated with miner's anæmia. When found in large numbers it is usually responsible for severe diarrhœa and anæmia.

III. **Cestodes** (*Tapeworms*).—The cestodes, or tapeworm group of intestinal parasites, possess a twofold clinical interest based upon their regional distribution and condition of maturity. The *mature worm* occupies the small intestine, the symptoms to which it gives rise depending largely upon the size and number present, and even when abundant they seldom prove directly fatal; but, *per contra*, the visceral distribution of the *larvæ* or immature parasite frequently causes grave and important symptoms. The following group includes the more important varieties of the mature tapeworm, the symptoms arising from each being much the same.

(I) **Intestinal Cestodes** (Tapeworms).—*Tænia saginata:* In its larval condition it is known as *Cysticercus saginata* or beef measle worm. It is the most common form of tapeworm in man, being derived from beef used as food (see also page 149). *Tænia solium:* The pork or armed tapeworm (see also page 150). In its larval condition it is known as *Cysticercus cellulosæ*. *Tænia cucumerina* (dog) and *Tænia elliptica* (cat) are by some considered as the same species. They are found in both adults and children. *Tænia flavopunctata:* A small variety of tænia; when found it is usually in children, giving rise to few symptoms. *Bothriocephalus latus:* Is also known as the *Tænia lata* or broad tapeworm. It is a very common worm in Sweden and Switzerland.

Symptoms.—The symptoms arising from this group of intestinal parasites are all of the same general character, are both local and general, and persist until the entire parasite has been removed.

(1) Local Symptoms.—The diagnostic local symptoms are the finding of segments in the stools, or at times in the clothing, the segments extruding themselves from the anus, particularly in the case of the *Tænia saginata;* and the occasional vomiting of segments, especially in women. In the latter case, if it is the *Tænia solium*, portions are likely to remain in the stomach, rendering the patient liable to measles or cysticerci. There may be distressing itching

about the anus, with abdominal uneasiness, fulness, or pain, nausea, vomiting, and diarrhœa. At times there may be a sense of movement in the intestine due to the worm, this condition being increased by fasting and often relieved by a full meal.

(2) General Symptoms.—The appetite is variable, the breath fetid, and the tongue usually furred. The patient may be pale and emaciated, showing discolorations about the eyes. Anæmia is not uncommon in long-continued cases, with dizziness, fulness in the head, buzzing in the ears, twitching of the face, and dull headache. A fatal form of anæmia may be due to the bothriocephalus (SCHAUMANN). There may be great mental depression, even hypochondriasis, in chlorotic and hysterical persons, and not infrequently some uterine disorder. Fainting, chorea, and epileptic fits are rare.

(II) **Visceral Cestodes.**—The larvæ of two of the cestodes may infest the solid organs, producing affections that may be serious, the *Tænia solium* and the *Tænia echinococcus*. The larva of the former is known as the *Cysticercus cellulosæ* or pork measle worm; that of the latter as the *echinococcus*.

CYSTICERCUS CELLULOSÆ.—Infection occurs by swallowing the eggs or mature segments of the tapeworm. They have been found in the brain, muscles, eyes, liver, kidneys, lungs, heart, and subcutaneous tissues. When present in large numbers in the muscles there are stiffness, pain, general weakness, numbness and tingling, and painful nodules containing cysticerci are found in the subcutaneous tissues. When in the brain and cord they may give rise to obscure symptoms—e. g., evidences of diabetes and anomalous nervous manifestations. The cysticercus has been found pressing upon the floor of the 4th ventricle. It may be present in the vitreous humour of the eye. The *diagnosis* of the condition when affecting the internal viscera is practically impossible; but they can be detected in the eye, so also in a subcutaneous nodule after excision.

ECHINOCOCCUS.—Derived from the *Tænia echinococcus* or hydatid tapeworm, and is the cause of hydatid tumours. The mature worm is found in the dog, infection occurring by swallowing the eggs. Hydatid tumours are most common in the liver, but may occur in the lungs, kidneys, spleen, omentum, subperitoneal tissue, heart, brain, spinal canal, pelvic viscera, and bones.

Symptoms and Diagnosis.—Small and few cysts in the *liver* cause little or no disturbance. If very large, there may be a feeling of weight or pressure in the region of the liver. If near the surface, there is a distinct tumour which may have a firm, tense, sometimes fluctuating, feeling. If situated to the left of the suspensory ligament, they may press upward on the heart and increase the area of cardiac dulness. If suppuration of the cyst occurs, pyæmic symptoms rapidly follow, i. e., jaundice, rigours, sweating, rapid, feeble pulse, and loss of weight. The cysts may perforate any of the surrounding hollow organs—stomach, bile passages, colon, pleura, bronchi, pericardium, or peritoneum. Perforation into the inferior vena cava or pericardium is rapidly fatal. They may open externally. External rupture and aspiration of the cysts are frequently followed by urticaria. The general health may be good. In simple echinococcus the liver is irregularly enlarged; in the multilocular variety the enlargement is regular and smooth; jaundice is a common symptom, there are progressive emaciation and, later in the disease, frequent hemorrhages. It is confined almost entirely to the liver, the symptoms being not unlike those of tumour or cirrhosis. The hydatid thrill or fremitus (page 474) is a diagnostic sign when it can be elicited. The sudden development of septic symptoms where there has been an enlargement of the liver associated with previously good health is suggestive of suppurating hydatids. In the same connection a sudden intense jaundice may indicate perforation into the bile passages.

When the larvæ develop primarily in the *pleura* the early symptoms may be those of compression of the lungs and displacement of the heart. The physical signs are those of effusion, and the condition readily may be mistaken for simple hydrothorax. Hydatid disease of the right lobe of the liver may encroach upon the right pleural cavity and pass for primary involvement of the pleura. Examination of aspirated fluid, which reveals a non-albuminous liquid (see also (8), page 717), is sufficient for differentiation. The hydatid may rupture through the chest wall, in which case echinococcus cysts are frequently found in the discharge.

Development of the cysts in the *lungs* is attended by symptoms of compression, later by the formation of cavities, and occasionally gangrene. Pulmonary hemorrhage may follow extensive destruction of lung tissue. Most of the cases of primary hydatid formation in the lung are during life mistaken for gangrene or tuberculosis. Occasional rupture occurs, either into the pleura with resulting empyema, or into a bronchus. In the latter case there is a discharge of fluid containing fragments of cyst membrane, and sometimes partial or complete hooklets. The majority of these cases are fatal, rupture into the pericardium inevitably so.

In the order of frequency, hydatid development in the *kidneys* is second only to that occurring in the liver. The kidney may reach an enormous size and resemble a hydronephrosis. An exploratory puncture is necessary to a positive diagnosis. When rupture occurs into the pelvis of the kidney, cysts or membrane may be passed in the urine, attended by colicky pains resembling renal colic. The general health is not usually much impaired.

In the *brain* the symptoms are, as a rule, those of tumour, i. e., convulsions, distressing headache, and gradually developing blindness. There is nothing to distinguish this condition from other forms of brain tumours.

IV. **Parasitic Arachnida.**—(I) **Sarcoptes (Acarus) Scabiei** (*Itch Insect*).—This is the common parasite of itch, and is found ordinarily in the folds of the skin, or where the skin is delicate. The insect, usually the female, is about 0.45 mm. in length, pearly white in colour, easily detected by the naked eye, and occupies a small burrow in the epidermis. The principal *symptoms* are distressing itching and an eruption which may be papular, vesicular, or pustular, and general irritation of the skin resulting from the scratching. There is seldom any doubt as to the *diagnosis*, the appearance of the parasite under the skin, accompanied with the evidences of irritation and scratching, being usually sufficient.

(II) **Demodex Folliculorum** (*Steatozoön*).—Principal habitat is in the sebaceous glands of the face, neck, and chest, which present minute elevations, containing in their centres exposed blackish points (*comedones* or *blackheads*).

V. **Parasitic Insects.**—(I) **Pediculi** (*Body Lice*).—These are:

Pediculus Capitis.—Found in regions containing long hair, principally in the head. They multiply rapidly by the deposit of eggs which cling to the body of the long hairs, appearing as white specks, sometimes called nits. The *symptoms* are itching and irritation of the scalp. If abundant, an eczema or pustular dermatitis, with crusts and scabs, may appear on the head, resulting in a dense matting and tangling of the crusts and hair, known as the *plica polonica*. There is no difficulty in *diagnosis*, the eggs or nits clinging to the shafts of the hair being distinctly visible to the naked eye.

Pediculus Corporis.—Lives in the clothing and sucks blood from the body. The resulting hemorrhagic specks are very common on the back, abdomen, and neck, and create a distressing desire for constant scratching. Urticaria may follow as a result of the irritation. The so-called vagabond's disease—*morbus errorum*—occurs in cases of long standing, and is characterized by a rough, thickened, often pigmented skin. The *diagnosis* depends upon the finding of the parasite in the clothing, and the irritation and peculiar hemorrhagic spots on the skin. The only condition with which it may be confounded is the bronzing of Addison's disease (page 1008).

Pediculus Pubis.—The *symptoms* produced by this louse resemble those just mentioned. They occupy the sites of the shorter hairs, especially the pubes, occasionally the axilla and eyebrows.

(II) **Cimex Lectularius** (*Common Bedbug*).—The *symptoms* due to the sting of this bug vary according to personal susceptibility. In some individuals there is little or no reaction, in others there may be intense urticaria and hyperæmia of the skin. The bite resembles somewhat a papular urticaria, but has in its centre a dark pin-point discoloration made by the proboscis of the animal. The *diagnosis* depends upon the finding of the bug and inspection of the bite.

(III) **Pulex Irritans** (*Common Flea*).—Easily transferred from one person to another during temporary crowding in public places. The irritation following a bite is similar to that of (II), preceding.

(IV) **Pulex Penetrans** (*Sand Flea-jigger*).—Much smaller than (III) and usually attacks the feet. It penetrates the skin and burrows, producing inflammation, sometimes vesicular or pustular swelling.

SECTION XIII

DISEASES OF THE TROPICS

PREPARED BY HARTWIG KANDT, M.D.

SOME diseases of the tropics have been, in the past, and still are, described elsewhere in this volume. These are (see Index) ankylostomiasis, beriberi, cholera, dengue, dysentery, leprosy, malaria, Malta fever, bubonic plague, and yellow fever. Those hitherto considered but slightly, or not at all, follow:

I. **Acute Febrile Icterus** (*Weil's Disease, Infectious Jaundice*).—Common in Egypt and throughout the tropics; observed during hot weather in countries of moderate climate. Sporadically, or in endemics.

Cause.—Uncertain, attributed to drinking of water contaminated with Proteus fluorescens, which has been grown from urine, and isolated from liver and kidneys *post mortem*.

Symptoms.—Males between 25 and 40; butchers, farmers, laborers predisposed. Onset abrupt, with chill, temperature rise to 104° F.; fever remittent in character, declines by lysis after a week or two, and tends to return. Gastric symptoms, diarrhœa, muscular pains, and headache come early and stay. Herpes common, rarely an erythema. On the 3d or 4th day jaundice of varying intensity develops; stools may be clay-colour. Nervous symptoms and emaciation prominent, delirium and coma in grave forms. Epistaxis and hematuria frequent, albuminuria with casts the rule.

Diagnosis.— From *typhoid fever* by the sudden onset, intense jaundice, absence of rose spots, and negative Widal test. From *malaria* by the absence of plasmodia in the blood. From *yellow fever* by the temperature curve, and absence of pernicious vomiting. From *dengue* by the albuminuria and absence of skin eruptions.

Prognosis.—Good as to life, duration about a month, convalescence slow.

II. **Febrile Tropical Splenomegaly** (*Kala Azar, Dum-dum Fever*).—Widely distributed in tropics and subtropics. Common in China, India, Assam, Africa, also in Panama and the Philippines, in epidemics and sporadically.

Cause.—Unknown, considered a trypanosome infection, the so-called Leishman bodies (*q. v.*) having been found in the bone marrow, spleen, and liver of victims. The same bodies are obtained by splenic puncture during life.

Symptoms.—Onset very gradual. Splenic enlargement the most prominent symptom, dulness may extend to pubis. Liver only moderately enlarged. Fever intermittent, remittent, or irregular, with paroxysms resembling those of malaria. In the later stages intestinal symptoms, with diarrhœa and bloody stools. Intestinal ulcerations may cause perforation. Cancrum oris common. Finally anæmia develops with hæmorrhages, dyspnœa, and œdema. Complications and secondary infections are frequent. Malaria coexists in many cases.

Diagnosis.—From malaria and leucæmia by blood examination, and by the finding of Leishman bodies in the material obtained by splenic puncture.

Prognosis.—Lasts for months or years; invariably fatal.

III. **Tick Fever.**—Due to the bite of certain blood-sucking ticks (*q. v.*), or wood lice. These ticks are found all over the world, abounding in the tropics. Following the bite, and after a variable period of incubation, the victim is seized with abdominal pain, vomiting, diarrhœa, chills and fever of the relapsing type.

IV. **Epidemic Dropsy.**—An affection, endemic in India, resembling beriberi very closely in its symptomatology. The only distinguishing features are the presence of fever and of a multiform eruption on face, body, and limbs.

V. **Tropical Ulcer** (*Oriental Sore, Biscra Button; Biscra, Aleppo, Delhi, Bagdad Boil*).—Very common in the Oriental tropics. Europeans not immune. Transmission by inoculation from direct contact.

Cause.—Unknown. Leishman bodies are found constantly in the granulations of tropical ulcer. Splenomegaly is, however, a very rare concomitant.

Symptoms.—The ulcers, up to 20 in number, are found on the uncovered parts of the body. First evidence of infection is a small red macule, which turns into a papule and nodule. Slowly this nodule increase in size, discharges pus, a crust forms, drops off, and exposes a granulating ulcer. Crusts keep on forming and separating, leaving punched-out ulcers of increasing size. Their diameter may exceed 1 inch. After months or years the lesions heal by granulation, a depressed scar remaining. Secondary infections are not infrequent. Systemic symptoms in uncomplicated cases are slight. One attack confers immunity.

Diagnosis.—The appearance of such ulcers, during or after residence in the tropics, should arouse suspicion. The detection of Leishman bodies in the granulations is very suggestive.

VI. **Yaws** (*Frambesia, Polypapilloma Tropicum*).—Common in tropical Africa, Asia, and the West Indies. A contagious skin disease of unknown origin, transmitted by direct contact.

Symptoms.—Incubation period from 2 weeks to several months. Without prodromata, crops of painless nodular excrescences appear on the skin of face or limbs, ulcerate, and present finally the appearance of raspberries, owing to a number of yellow points of necrosis on their flattened surface. At first there is itching, articular pains, and fever; later the itching ceases. The disease lasts from 2 months to a year or more and leaves superficial scars.

VII. **Tropical Sloughing Phagedena.**—Common in tropics, but cases presenting a very similar appearance are met in moderate climates.

Symptoms.—It resembles hospital gangrene, but is less contagious and rarely fatal. The earliest symptom is the appearance of a blister on an extremity. It ruptures after a few hours and exposes a gray area of superficial gangrene, tending to spread rapidly at its margins. The floor of the ulcer is of a dirty yellow color, the odour very offensive. Systemic symptoms are usually slight. After a variable period, usually a week, the slough separates, and recovery, without much damage to the deeper structures, is the rule. The infecting organism is unknown, but is probably of the pyogenic variety.

VIII. **Madura Foot** (*Mycetoma, Fungus Foot of India, Pied de Cochin*).—Widely distributed through the tropics, endemic in India, lately in Panama.

Cause.—Probably the Streptothrix mycetoma, a vegetable fungus of the Actinomyces group, which enters the foot through wounds or abrasions.

Symptoms.—Dark races and barefooted people are especially susceptible. The disease affects the foot, rarely the hands or other parts. At first there is a small, round, painless, nodular swelling on the plantar surface or dorsum of the foot. Gradually softening takes place, leaving a craterlike opening, from which an oily, seropurulent material is discharged, containing the characteristic reddish granular bodies or black granules resembling particles of gunpowder. Other nodules form on the skin and break down, while the deep structures of the foot undergo similar degenerative changes. Finally the diseased part becomes badly deformed, the limb above wasting away, while the affected foot doubles in size and loses its natural contour. Systemic symptoms, except those due to long-continued suppuration, are lacking.

Prognosis.—The disease tends to stay localized, but is incurable without amputation.

IX. **Climatic Bubo.**—**Symptoms.**—A non-venereal swelling of the inguinal glands, unilateral or bilateral, associated with fever. Common in tropics, especially among sailors. Resolution or suppuration takes place. Some do not consider climatic bubo a pathological entity, and suggest that it may be a variety of bubonic plague or dengue.

X. **Ainhum** (*Dactylolysis Spontanea*).—**Symptoms.**—Endemic in India; consists of a very slow (1 to 10 years), painless, spontaneous amputation of one or more toes at the plantar fold. The little toe is the one most frequently affected; sometimes the condition is symmetrical. Constitutional symptoms are absent. The cause of ainhum is unknown. Some consider it a tropic disease, others take it for a manifestation of leprosy.

XI. **Goundou** (*Big Nose*).—A tropical affection of unknown origin, restricted to the negroes of Africa.

Symptoms.—Children and young adults are affected. Ushered in by headaches. Hard symmetrical tumours develop slowly on the upper portion of the nose. There is fever and a purulent nasal discharge. After a few months all constitutional symptoms subside, but the tumours are permanent. They seem to be exostoses of the nasal bones, do not involve the overlying skin, and may become as large as oranges. Nasal breathing is not, as a rule, interfered with.

XII. **Guinea Worm Disease** (*Dracontiasis, Dranunculosis, Medina Worm Disease*).—Very common in certain parts of Southwestern Asia and East Africa.

Cause.—The Guinea worm, a parasite about 30 inches long and $\frac{1}{16}$ inch thick. Only the female is known. The worm in its embryonic state is probably ingested in water, penetrates the intestine, and migrates into the subcutaneous tissues, where it completes its development. When mature it travels downward, head first, until it reaches the leg or foot, where it pierces the skin. Whenever water is near it protrudes consecutive portions of the uterus through the mouth; these burst and discharge a milky fluid, consisting of innumerable living embryos.

Symptoms.—The worm reaches the skin about 1 year after ingestion has taken place. The first evidence of its presence is the appearance of a small blister at the point where it tries to pierce the skin, usually above the ankle. This event is ushered in by fever, sometimes by an urticarial rash. Soon the blister will be seen to rupture, and a small ulcer is left through which the worm's head may protrude, while its coils can be felt above like a phlebitic vein. The phenomenon of discharging the young can be elicited by playing a stream of water on the foot. The patient is unable to walk, but no other symptoms develop except those due to local irritation or infection from outside. After emptying its uterus the worm may be extracted with ease. Two or more worms may appear at the same time in the same individual.

XIII. **Endemic Hæmoptysis** (*Distomum Pulmonale*, or *D. Westermanni, Fluke Worm of the Lung*).—Endemic in China, Japan, Corea, and Formosa, observed in other Oriental countries and the United States.

Cause.—An infection by the Distomum Westermanni, a fluke worm belonging to the genus Paragominus. In size and shape the adult parasite resembles a watermelon seed. The embryo is probably ingested in water and finds its way into the lungs, where it matures, deposits its ova, and forms many small cysts. These communicate with the bronchi, and in coughing the eggs are expelled with the bronchial secretions.

Symptoms.—Incubation period unknown. Chief symptoms are a chronic cough with bloody expectoration and occasional hæmorrhages. Secondary anæmia frequently results. In the blood-stream the eggs may be carried to other organs (liver, brain), producing grave symptoms.

Diagnosis.—A case of hæmoptysis of long standing, without evidence of tuberculosis, should prompt an examination of the sputum for the ova of this parasite.

Prognosis.—Uncertain, not usually fatal unless from complications.

XIV. **Bilharzia Disease** (*Endemic Hæmaturia*).—Endemic in Egypt and North Africa, occurs in Asia, sporadically in other tropical countries. Males seem predisposed; white races not immune.

Cause.—The Schistosomum hæmatobium (*q. v.*), a trematode about 1 cm. long and of a milk-white colour. The adult worms are invariably found in the portal vein. It is not known how they get there; perhaps through the skin, perhaps by ingestion of contaminated water. Also found in the liver, bladder wall, and the pelvic and intestinal veins, where they lay their eggs. These are armed with a thornlike spine with which they penetrate the vessel walls and reach, finally, the cavities of bladder and rectum, to be swept away in the urine and fæces.

Symptoms.—Are frequently slight or absent; their gravity depends on the number of parasites present. A drop of blood at the end of urination, due to the laceration of a blood-vessel by the sharp-spined ova, may be the first symptom. Backache and vesical tenesmus are followed by catarrhal or purulent cystitis, with hematuria. Urethritis, peri-urethral abscess, and renal disease are frequent complications. Vesical calculi, with a nucleus of dead ova, are common. In the rectum the local irritation sets up a proctitis of varying intensity, leading sometimes to prolapse, and simulating dysentery. The prolonged hæmorrhage causes anæmia.

Diagnosis.—By examining urine and fæces for the ova of Schistosomum hematobium. The last portion of urine voided is used for this purpose.

Prognosis.—Depends on gravity of infection, cure rare, kills by renal complications, or exhaustion.

XV. **Schistosomum Japonicum** (*S. Cattoi*).—Endemic in China and Japan; parasite resembles in form and habits that of Bilharzia, but is smaller. Its ova are devoid of spines, do not invade the bladder, and are found chiefly in the mucous and submucous coat of the intestines. The symptoms are those of chronic enteritis, anæmia, and cachexia. Splenic and hepatic enlargement is the rule.

XVI. **Intestinal Fluke Worms.**—Three varieties have been observed. Their life history and symptoms are unknown. Two of these, the Distomum Buski and Distomum Rathusi, inhabit the upper intestine; the Gastrodiscus Hominis, the lower intestine.

XVII. **Cestodes.**—Four species of tapeworm are distinctly tropical: (1) *Tænia Nana;* common in Africa, South America, and the Orient. About 1 inch long, scolex armed with a single circle of hooks. Intermediate host is the rat. Causes intestinal symptoms and anæmia. (2) *Tænia Madagascariensis.* (3) *Tænia Africana.* (4) *Bothriocephalus Mansoni;* observed in China, Japan, and Australia. Man is the intermediary host.

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